Curr Cardiol Rep (2017) 19:105
DOI 10.1007/s11886-017-0924-0

Ventricular Tachycardia with ICD Shocks: When to Medicate and When to Ablate

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Abstract
Purpose of Review Ventricular tachycardia occurrence in implantable cardioverter defibrillator (ICD) patients may result in shock delivery and is associated with increased morbidity and mortality. In addition, shocks may have deleterious mechanical and psychological effects. Prevention of ventricular tachycardia (VT) recurrence with the use of antiarrhythmic drugs or catheter ablation may be warranted. Antiarrhythmic drugs are limited by incomplete efficacy and an unfavorable adverse effect profile. Catheter ablation can be effective but acute complications and long-term VT recurrence risk necessitating repeat ablation should be recognized. A shared clinical decision process accounting for patients' cardiac status, comorbidities, and goals of care is often required.
Recent Findings There are four published randomized trials of catheter ablation for sustained monomorphic VT (SMVT) in the setting of ischemic heart disease; there are no randomized studies for non-ischemic ventricular substrates. The most recent trial is the VANISH trial which randomly allocated patients with ICD, prior infarction, and SMVT despite first-line antiarrhythmic drug therapy to catheter ablation or more aggressive antiarrhythmic drug therapy. During 28 months of follow-up, catheter ablation resulted in a 28% relative risk reduction in the composite endpoint of death, VT storm, and appropriate ICD shock (p = 0.04). In a subgroup analysis, patients having VT despite amiodarone had better outcomes with ablation as compared to increasing amiodarone dose or adding mexiletine.
Summary There is evidence for the effectiveness of both catheter ablation and antiarrhythmic drug therapy for patients with myocardial infarction, an implantable defibrillator, and VT. If sotalol is ineffective in suppressing VT, either catheter ablation or initiation of amiodarone is a reasonable option. If VT occurs despite amiodarone therapy, there is evidence that catheter ablation is superior to administration of more aggressive antiarrhythmic drug therapy. Early catheter ablation may be appropriate in some clinical situations such as patients presenting with relatively slow VT below ICD detection, electrical storms, hemodynamically stable VT, or in very selected patients with left ventricular assist devices. The optimal first-line suppressive therapy for VT, after ICD implantation and appropriate programming, remains to be determined. Thus far, there has not been a randomized controlled trial to compare catheter ablation to antiarrhythmic drug therapy as a first-line treatment; the VANISH-2 study has been initiated as a pilot to examine this question.

Keywords Ventricular tachycardia · Catheter ablation · Antiarrhythmic medications · Implantable cardioverter defibrillator · Sudden cardiac death

Introduction

Ventricular tachycardia (VT) is potentially life-threatening, particularly in patients with structural heart disease. The risk of sudden cardiac death (SCD) is the highest in patients with ischemic heart disease [1] but varies in different populations depending on the nature of the underlying heart condition,

This article is part of the Topical Collection on *Invasive Electrophysiology and Pacing*

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Published online: 13 September 2017

specific family history, and genetic variants [2]. On the other end of the spectrum, VT in the setting of normal heart (idiopathic VT) is generally considered to have a benign prognosis [3]. This variation in prognosis has important implications for selecting appropriate therapy modalities.

Implantable cardioverter defibrillator (ICD) implantation is generally recommended for patients with sustained VT and structural heart disease [4•, 5, 6]. Sustained monomorphic VT (SMVT) in this setting is most commonly a result of reentry through myocardial scar, with the most frequent etiology being prior infarction. This type of VT typically occurs in the absence of acute ischemia [7, 8]. Ventricular scars leading to re-entrant VT also occur in patients with non-ischemic cardiomyopathies, including idiopathic dilated cardiomyopathy, hypertrophic cardiomyopathy, infiltrative heart diseases (e.g., sarcoidosis), arrhythmogenic right ventricular cardiomyopathy, and repaired congenital heart disease or valvular heart disease.

In this review, we will focus on management options in patients with prior myocardial infarction and ICD who develop VT leading to therapy from the device.

ICD Therapy for VT

Three large, prospective randomized trials (AVID, CASH, and CIDS) evaluated ICD therapy compared to amiodarone or other antiarrhythmic drugs in survivors of cardiac arrest or life-threatening VT. They showed a significant improvement in overall mortality [9]. Although these trials did not address patients with hemodynamically stable SMVT, there is general agreement that ICD therapy may be beneficial in patients with structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable [4•, 5, 6]. The principal benefits in patients with hemodynamically stable VT are the ability to reduce the need for antiarrhythmic drugs in patients with infrequent tachycardia episodes and the ability to terminate SMVT with antitachycardia pacing (ATP) rather than with shocks.

While ICDs can be life-saving, ICD shocks have consistently been associated with increased mortality [10–12], an observation that was not seen with ATP alone [13]. In SCD-HeFT, a single appropriate shock increased the risk of death fivefold and additional shocks were associated with further threefold increased mortality risk [14]. There is mounting evidence from prospective trials that recurrent ICD shocks may impair myocardial function [12, 15, 16], which could offset some of the benefit of ICDs by harm from shocks. In addition, repeated ICD shocks are associated with significant risk of psychological morbidity. The occurrence of ≥ 1 ICD shock is associated with significant reductions in mental well-being and physical function and an increase in patient concerns and anxiety; in some series, depression has been reported [17–26].

Trials of programming ICDs to treat VT without shocks were successful and form the basis for modern programming [27, 28]. Further shock reduction can be achieved with prolongation of the ICD detection interval for patients with primary prevention ICDs [29] but has less apparent benefit for patients who already have VT [30, 31].

Given the limitations of ICDs, VT suppression therapy is still required for many patients with VT substrate. Revascularization is usually not effective as an antiarrhythmic strategy in patients with SMVT, although such patients should be investigated for myocardial ischemia which, if present, should be treated to improve prognosis [32, 33]. Effective treatment of congestive heart failure does reduce both heart failure mortality and sudden death [34–37] but has not been effective in reducing recurrences of VT. It had been hoped that cardiac resynchronization therapy would significantly reduce VT; unfortunately, this only appears to be the case in those without a history of VT [38], particularly if the EF improves [39]. Once the substrate for VT becomes manifest, reduction of VT requires specific therapy, either antiarrhythmic drug therapy or catheter ablation.

Clinicians and patients often choose to suppress VT when there appears to be an increased risk of arrhythmia-related morbidity or mortality. Common clinical indications include sustained VT requiring ICD shocks or external cardioversion/pharmacologic conversion and symptomatic VT treated by ATP or VT storm. Data to inform the most appropriate threshold for the initiation of suppressive therapy for VT remains very limited, although trials are currently under way to address this question [40].

Antiarrhythmic Drug Therapy

With the exception of beta-blockers, none of the antiarrhythmic medications for ventricular arrhythmias has been shown to improve survival. In fact, class I antiarrhythmic drugs increased mortality in patients with ischemic heart disease [41]. In the ICD era, only amiodarone and sotalol have been shown in randomized trials to reduce the frequency of recurrent VT. Amiodarone is the most effective available antiarrhythmic agent for the reduction of VT [42]. It substantially reduces 1-year recurrent VT probability for patients with ICDs who have experienced VT (HR 0.27, $p < 0.001$) [43]. When used for primary prevention of sudden cardiac death in heart failure patients, it reduced the probability of arrhythmic death without improving total mortality [44–47]. It is suggested that its benefit is offset by the long-term risk of side effects [48]. Potential amiodarone side effects are numerous and often relate to longer term exposure. In an 11-year long-term follow-up substudy of the CIDS trial, side effects were seen in 82% of patients and drug was discontinued in 50% over a mean follow-up 5.6 years and, within 8 years, 100% of patients

assigned to amiodarone had either died, had recurrent VT, or did not tolerate the drug [49]. In a meta-analysis which included studies with follow-up ≥ 1 year, even relatively low-dose amiodarone was significantly associated with frequent side effects [50]. Sotalol appears to be less effective than amiodarone but better than beta-blockers in reducing recurrences of VT [43, 51]. Sotalol, similar to other antiarrhythmic drugs, confers a dose-dependent risk of proarrhythmia [52] mostly related to torsades de pointes. In the presence of an ICD, it has a relatively good safety profile and is a frequent choice as a first-line therapy for patients without high risk of sotalol-induced proarrhythmia [53••]. However, sotalol should be avoided in patients with severe reactive airways, left ventricular ejection fraction (LVEF) < 20%, severe renal insufficiency, or severe QT prolongation [54–56]. Other antiarrhythmic drugs such as dofetilide, azimilide, and ranolazine are either ineffective [57–59], are unavailable [60], or have limited evidence to support use [61, 62].

Catheter Ablation for Scar-Related VT

Catheter ablation is a non-pharmacologic approach to reduce VT recurrences through interrupting critical conducting myocardial channels within the ventricular scar which typically has low electrogram voltage. Due to its relative efficacy and the disappointing effectiveness of antiarrhythmic drugs, its use for post-myocardial infarction (MI) VT has tripled in the last decade [63]. The ablation strategy, risks, and outcomes are related to the nature and location of the underlying myocardial substrate. Most SMVTs, particularly in the setting of ischemic heart disease, have critical isthmuses close to the subendocardium and can be approached endocardially. Subepicardial scar is increasingly recognized as an ablation target in patients with non-ischemic cardiomyopathy, e.g., Chagas disease, arrhythmogenic right ventricular cardiomyopathy (ARVC), and idiopathic dilated cardiomyopathy. Catheter ablation procedures for these conditions usually involve a percutaneous subxiphoid puncture [64–67].

While catheter ablation for VT is "minimally invasive," it does require instrumentation of the left ventricle in patients who have significant left ventricular dysfunction and, often, other comorbidities. In a contemporary cohort experience of unselected patients undergoing transcatheter VT substrate ablation, procedural risks included a mortality risk of 0.4%, as well as risks of cardiac perforation of 1.4%, a risk of systemic embolism including stroke/MI of 0.8%, and a risk of vascular complications of 2%. Overall, the risk of a major complication was approximately 5–6% [68]. This rate is in the same range as the observed complication rate in multicenter experiences. In the Multicenter Thermocool prospective cohort study of 231 patients, the 30-day mortality was 3%, mostly due to VT which was not successfully ablated rather than due to complications, while overall, there were significant complications in 7.3%. In phase 2 of this study, when investigators had experience with the catheter, the complication rate fell to 5.1%, with the major contributors including cardiac perforation in 0.4%, CHF in 1.3%, and respiratory complications in 0.8% [69]. These complication rates are concordant with those of other multicenter experiences [70].

Although acute success of catheter ablation is reported in the 70–90% range, long-term outcomes are more guarded. In patients with ischemic cardiomyopathy, reports in the past 5 years with follow-up longer than 2 years show a VT-free survival of 42–54% [71••, 72•, 73•]. Long-term VT-free survival in patients with non-ischemic cardiomyopathy is still less favorable (38% over 6 years of follow-up) [72•]. This is partly attributed to the midmyocardial or subepicardial location of VT re-entry circuits in this patient group, which necessitates epicardial ablation in more than a third of patients [67].

Bundle branch re-entrant VT (BBR-VT) is the second most common form of VT in patients with non-ischemic cardiomyopathy. Catheter ablation of the right bundle has shown very high acute success in large series for this arrhythmia, at the expense of a 10–30% risk of high-grade AV block [74, 75]. Long-term prognosis is usually more guarded, however, as these patients often have severe underlying heart disease and concomitant myocardial re-entrant VT [75].

Despite all these limitations, recent retrospective multicenter analyses examining the outcomes of catheter ablation in the setting of structural heart disease have suggested a possible survival benefit among those with a successful procedure. Tung et al. [76•] analyzed the outcomes of 2061 VT ablation patients from 12 international centers. At 1 year of follow-up, transplant-free survival was significantly higher in patients without VT recurrence than in those with recurrence (90 vs. 71%, $p < 0.001$). This observation was independent of ejection fraction or heart failure severity. Similarly, Yokokawa et al. [77•] analyzed data from 1064 patients who underwent VT ablation for post-infarction VT at 7 international centers. Non-inducibility of VT at the end of the procedure was found to be independently associated with 35% reduction in mortality risk over a 2-year follow-up period.

Randomized Studies of Catheter Ablation vs. Medical Treatment

There are four published randomized trials of catheter ablation for SMVT in the setting of ischemic heart disease; there are no randomized studies for non-ischemic ventricular substrates. The SMASH-VT trial compared ICD implantation along with VT substrate ablation to ICD implantation alone in patients with recent VT [78]. In this trial, ablation of the VT substrate reduced ICD shocks from 31 to 9% over a mean follow-up of 22.5 ± 5 months ($p = 0.003$) and reduced VT from 33 to 12%

($p = 0.007$) in comparison to a control group that did not use antiarrhythmic drug therapy. The VTACH trial [79] studied the effect of catheter ablation in patients with ischemic cardiomyopathy, who experienced hemodynamically tolerated SMVT without specific antiarrhythmic drug therapy in the control arm (approximately one third of patients in both arms were treated with amiodarone). Over a mean follow-up of 22.5 months, ablation prolonged the time to recurrent VT significantly (HR = 0.61), with a relative risk reduction of 25% and an absolute risk reduction of 18% at 2 years. These results were not seen in the more recent SMS study which enrolled patients with hemodynamically unstable VT, and did not observe a reduction in VT recurrence but did observe a reduction in the number of ICD interventions over 2.3 years of follow-up [80•]. The small sample size of this trial and loss of some patients to follow-up leaves a reasonable possibility that catheter ablation might show a benefit in this population in a larger trial [81•]. The VANISH trial randomly allocated patients with ICD, prior infarction, and SMVT despite first-line antiarrhythmic drug therapy to catheter ablation or more aggressive antiarrhythmic drug therapy [71••]. During 28 months of follow-up, catheter ablation resulted in a 28% relative risk reduction in the composite endpoint of death, VT storm, and appropriate ICD shock ($p = 0.04$). In a subgroup analysis, patients having VT despite amiodarone had better outcomes with ablation as compared to increasing amiodarone dose or adding mexiletine [82].

Clinical Management of VT Syndromes

First-Line Therapy for Suppression of VT

In the setting of structural heart disease, acute management of sustained monomorphic VT should follow clinical guidelines [53••]. A synchronized direct current cardioversion should be considered in the case of hemodynamically unstable VT. If VT is hemodynamically stable, intravenous amiodarone [83] or procainamide [84•] should be administered and, if not successful, intravenous sedation and cardioversion may be required. The presence of ICD can change the mode of presentation, as most of the hymodynamically unstable VTs will fall within the tachycardia detection zone and would lead to ATP and/or shock delivery. Long-term VT suppression using sotalol or amiodarone is generally not recommended for a single VT episode (class IIB) [53••]. For recurrent VT, amiodarone is more effective than sotalol but is plagued with the poor long-term adverse effect profile [43, 49–51]. Catheter ablation can be considered as a first-line therapy in selected cases, particularly in the setting of ischemic heart disease; however, randomized studies comparing antiarrhythmic drugs vs. catheter ablation as a first-line therapy for VT are still ongoing [85•].

Suppression of Drug-Refractory VT

Antiarrhythmic drug failure or intolerance is not an uncommon phenomenon in patients with structural heart disease. Catheter ablation is associated with better outcomes than escalation of antiarrhythmic treatment in the setting of ischemic heart disease [71••]. It is also the preferred treatment modality in patients with non-ischemic heart disease who receive ICD therapies despite antiarrhythmic treatment [53••, 86]. Antiarrhythmic combinations can be considered in patients with VT recurrences despite AADs and catheter ablation(s). Amiodarone and mexiletine can be combined with the intent of reducing SMVT recurrences [87], although recent data suggests that this has limited efficacy [82]. Limited experience is also present with combinations of sotalol and either quinidine or procainamide [88], or amiodarone plus mexiletine plus either quinidine or procainamide [89].

VT Storm

VT storm is typically defined by three or more episodes of sustained VT/VF or appropriate ICD shocks within 24 h. It is increasingly recognized in the ICD era and is associated with significant morbidity and mortality [90, 91]. Heart failure deterioration due to the ongoing arrhythmia and/or multiple ICD shocks can add further to the acuity of presentation [12]. ICD programming to avoid shocks is often needed [92•]. Catheter ablation with adequate hemodynamic support, whether medically or by using circulatory support devices, may be life-saving [93–95]. Superior VT-free survival has been reported when catheter ablation is successful [96–99]. Overall survival however is limited by the severity of the underlying heart disease. VT ablation can provide a period of stabilization to allow for more definitive treatment options like cardiac transplantation or assist device implantation.

In cases when antiarrhythmic drug therapy and catheter ablation are ineffective, not tolerated, or unavailable, other treatment strategies might be needed. Sedation and/or general anesthesia reduced arrhythmia burden in case reports of patients with refractory VT [100, 101]. Cardiac sympathetic denervation may also reduce arrhythmia burden in refractory patients [102–104]. This has been shown for left or bilateral cardiac sympathetic denervation with probable superiority of the latter [103].

Slow VT Below ICD Detection

Very slow conduction within the myocardial scar either due to antiarrhythmic drugs or the nature of the underlying disease can lead to relatively slow ventricular tachycardia—which may be slower than anticipated and thus below the VT

detection programming of the ICD. These VTs represent an interesting clinical challenge as they are generally tolerated [105], but are often incessant and can lead to significant deterioration of hemodynamics if left untreated [106]. Lowering ICD tachycardia detection limits may result in repeated delivery of ATP and/or shocks and can potentially lead to inappropriate therapy for sinus tachycardia or supraventricular arrhythmias. Escalation of antiarrhythmic agents often slows the VT further without terminating it. Catheter ablation represents an important treatment strategy for this clinical situation. Activation and entrainment mapping during VT can be performed to identify critical conducting channels within the scar and to target them with radiofrequency energy. Further substrate modification to target other faster VTs should be performed concomitantly [107].

Hemodynamically Stable Ischemic SMVT with Normal or Near-Normal LVEF

Catheter ablation may have a greater likelihood of effective suppression of ventricular arrhythmias when the VT is hemodynamically tolerated, permitting more specific targeting of culprit substrate within the myocardial scar. Although confirmatory clinical data are sparse, catheter ablation may be considered for first-line therapy for patients with a prior history of MI who present with stable SMVT [108]. This was the approach in the VTACH study, which identified a larger treatment effect for catheter ablation in patients who had LVEF > 30% [79].

VT in Patients with Left Ventricular Assist Devices

Left ventricular assist devices (LVADs) are increasingly used in heart failure patients as a bridge to transplant or as destination therapy. Due to the underlying myocardial scarring, 25–59% of those patients develop VT and are at increased risk of ICD shocks and overall mortality [53••, 109–113]. Although LVADs provide hemodynamic support during VT, reduction of RV function during VT can lead to diminished cardiac output and right-sided heart failure. VT in this situation tends to be refractory to antiarrhythmic drugs [109, 111, 114], and thus, catheter ablation might be required [113, 115, 116]. Since VT prior to LVAD implantation is the strongest predictor of VT afterwards [110, 114, 117], some authors advocate for a prophylactic VT ablation prior to implantation or concomitant surgical ablation of VT during LVAD implantation [118, 119], particularly in patients with non-ischemic cardiomyopathy who tend to have epicardial substrate that will no longer be accessible percutaneously after the procedure due to development of post-procedure pericardial scarring [53••].

Clinical Implications

There is evidence for the effectiveness of both catheter ablation and antiarrhythmic drug therapy for patients with myocardial infarction, an implantable defibrillator, and VT (Fig. 1). If sotalol is ineffective in suppressing VT, either catheter ablation or initiation of amiodarone is a reasonable option. If VT occurs despite amiodarone therapy, there is evidence that catheter ablation is superior to administration of more aggressive antiarrhythmic drug therapy. Early catheter ablation may be appropriate in some clinical situations such as patients presenting with relatively slow VT below ICD detection, electrical storms, hemodynamically stable VT, or in very selected patients with LVAD. Finally, patient preference should be considered during clinical decision-making after clear explanation of the potential risks and benefits of management options. The optimal first-line suppressive therapy for VT, after ICD implantation and appropriate programming, remains to be determined. Thus far, there has not been a randomized controlled trial to compare catheter ablation to antiarrhythmic drug therapy as a first-line treatment; the VANISH-2 study has been initiated as a pilot to examine this question [85•].

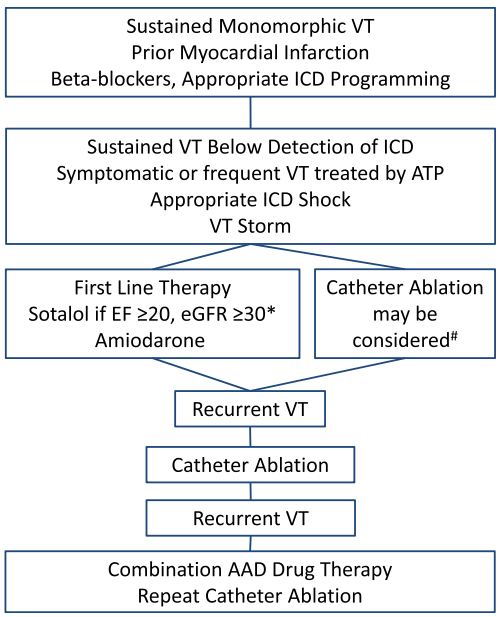

Fig. 1 Suggested flow chart for the management of SMVT after a prior myocardial infarction. *Sotalol should be avoided in patients with severe heart failure, prolonged QTc, advanced renal dysfunction, EF < 20%, or if there is a history of torsades de pointes. Amiodarone may be more appropriate in the setting of VT storm. #Early catheter ablation may be appropriate in patients presenting with relatively slow VT below ICD detection, electrical storms, hemodynamically stable VT, or in very selected patients with LVAD. Trials are currently underway to identify the most appropriate first-line therapy. See text for details

Conclusions

Ventricular tachycardia occurrence in ICD patients is associated with increased mortality particularly if shocks are delivered. In addition, shocks have several mechanical and psychological deleterious effects. Therefore, prevention of VT occurrence, by antiarrhythmic drugs or catheter ablation, is warranted. Antiarrhythmic drugs are plagued by limited efficacy and unfavorable adverse effect profile. Catheter ablation can be more effective particularly in some clinical circumstances, but acute complications and long-term VT recurrence risk necessitating repeat ablation should be recognized. A shared clinical decision process accounting for patient's comorbidities, hemodynamics, and goals of care is often required.

Compliance with Ethical Standards

Conflict of Interest Amir AbdelWahab has no conflict of interest.

John Sapp reports grants from Biosense Webster and St. Jude Medical and personal fees from St. Jude Medical. In addition, Dr. Sapp has a patent related to ECG mapping of VT pending and a patent related to a needle ablation catheter issued.

Human and Animal Rights and Informed Consent All procedures performed by the authors in VANISH study involving human participants were in accordance with the ethical standards of the institutional and national research committees and with the Helsinki Declaration.

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