REVIEWS

Utility of cardiac computed tomography to identify arrhythmia substrates for ventricular tachycardia and sudden cardiac death

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ABSTRACT

Sudden cardiac death (SCD) is the leading cause of death in the U.S., and many of these events are attributable to malignant ventricular arrhythmias such as sustained ventricular tachycardia (VT). Most of the efforts to identify arrhythmia precipitants in these patients are based on imaging to look for myocardial or coronary artery disease. As advances in cardiac computed tomography (CCT) are made, it has demonstrated its usefulness in identifying structural intracardiac pathology and measuring parameters of cardiac anatomy and function. In this article we review the different etiologies of VT/SCD that are identifiable by CCT, and its potential usefulness in the workup for VT/SCD.

Key Words: Cardiac computed tomography, Ventricular tachycardia, Sudden cardiac death

1. INTRODUCTION

Sudden cardiac death (SCD) is a major cause of death in the U.S., estimated to cause 213,000 deaths annually in North America.[1] Many of these events are attributable to malignant ventricular arrhythmias, such as mono/polymorphic ventricular tachycardia (VT) or ventricular fibrillation. Most of the efforts to identify arrhythmia precipitants in these patients are based on imaging to look for myocardial disease, both acquired and inherited, and the exclusion of coronary artery disease (CAD).[2] Transthoracic echocardiography (TTE) is especially helpful in identifying patients with low left ventricular ejection fraction (LVEF), who may then undergo placement of an implantable cardioverter-defibrillator (ICD). Invasive coronary angiography provides assessment of obstructive CAD and allows for immediate revascularization and alleviation of ischemia. However, the risks and costs of cardiac catheterization reduce the utility of this as a screen for patients at risk for SCD, and it is usually reserved for survivors of an event.

Monomorphic VT is the most common mechanism of SCD, as compared to bradyarrhythmias or acute infarction.[3] Ventricular tachycardia eventually degenerates into ventricular fibrillation, causing hemodynamic collapse and sudden cardiac death. There are many conditions that predispose patients to ventricular arrhythmias, which may be categorized as structural abnormalities or conduction/electrical abnormalities (congenital, drug-induced, or idiopathic). CAD is

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the most common cause of structural abnormality underlying SCD in the Western world, responsible for 75% of SCD's.[4] Cardiomyopathies (including dilated, hypertrophic, arrhythmogenic right ventricular dysplasia, and those related to valvular disease) and primary electrical disorders (channelopathies) account for most of the remainder.

It has been recognized that low LVEF is a nonspecific predictor of potentially fatal arrhythmic events.[5] However, in a substantial proportion of patients who receive an ICD based on reduced LVEF, they never receive therapy over the lifetime of the device.[6] Furthermore, only about 1/3 of patients who die suddenly have an LVEF that meets criteria for prophylactic ICD placement under the current guidelines.[6] Therefore, it is important to explore other methods for identifying patients who might benefit from therapies which can reduce mortality from SCD. Cardiac imaging, beyond the standard TTE and coronary angiography used to workup patients who present with VT/SCD, is increasingly recognized as having the potential to help identify substrates for malignant ventricular arrhythmias.[7,8]

As further advances in CT imaging technology are made, cardiac computed tomography (CCT) has an increasing number of emerging applications beyond its recognized utility as a noninvasive diagnostic tool for imaging coronary arteries and assessing CAD.[9] In particular, CCT has demonstrated its usefulness in identifying structural intracardiac pathology and measuring parameters of cardiac anatomy and function. As such, CCT has potential for helping identify myocardial substrate in patients who present with VT/SCD, or for identifying patients who are at risk for developing these substrates. It has also been shown to be a highly accurate measure of ejection fraction (discussed below), which may be of interest in determining the need for an ICD.

In this article, we review different substrates for VT that are identifiable by cardiac CT. We performed an extensive literature search of the Medline database (through PubMed) to identify relevant clinical trials and case studies demonstrating the ability of cardiac CT to identify various pathologies that predispose to VT. Titles and abstracts were screened for relevance, and reference lists from relevant articles were also crosschecked to identify additional studies. It should be noted that finding substrate for VT/SCD in and of itself does not necessarily predict risk for developing it, and with the exception of ICD placement in systolic heart failure, it is still unclear whether therapy or prophylaxis improves outcomes in patients with known substrate. Nevertheless, an awareness of CCT's full potential and range of applications, as well as the wide range of pathologies it can identify, can make it another valuable tool in the workup for VT/SCD.

2. Ischemic cardiomyopathy/obstructive coronary artery disease

Ischemic cardiomyopathy (ICM) is the most common cause of ventricular arrhythmia and sudden cardiac death. In addition to impairing systolic function, it is thought that extensively healed myocardial infarctions (MI) leading to myocardial scar can lead to electrical heterogeneity, becoming a substrate for electrical instability and precipitation of VT.[10]

Characteristic changes of ICM can be seen on imaging, and are well-described in the literature as seen on MRI. However, a number of techniques can be utilized with CCT in order to characterize areas of acute and chronic MI, as well as to accurately quantify scar volume (see Figure 1). Multidetector CT has been found to have contrast patterns similar to first-pass perfusion and delayed enhancement MRI, and scar size as quantified by delayed imaging CT has been shown to correlate well with MRI.[11,12] For both multidetector CT and MRI, delayed hyperenhancement is thought to result from the slow washout of contrast from the region of infarcted myocardium. To perform a delayed enhancement scan, scanning is performed between 5 and 10 minutes after contrast material injection.

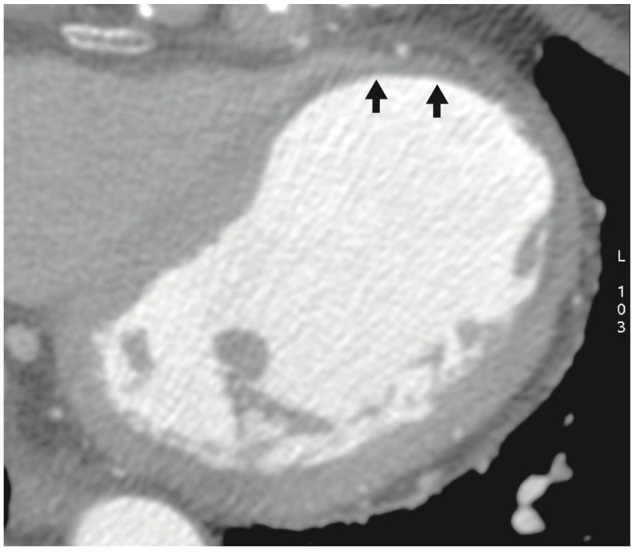

Figure 1. A patient with an old anterior myocardial infarction, with a markedly dilated left ventricle, apical thinning and fibrofatty metaplasia (arrows depict apical thinning and dark region represents fibrofatty metaplasia)

Nieman *et al.* used myocardial CT attenuation values and ventricular dimensions to show that multidetector CT can differentiate a recent (< 7 days) from chronic (> 12 months) MI.[13] In patients with long-standing MI as compared to recent MI, they found significantly lower CT attenuation values (-13 +/- 37 Hounsfield units [HU] vs. 26 +/- 26 HU, p

< .001), larger attenuation difference between infarcted and remote myocardium, and increased wall thinning and ventricular dilation. CCT's potential to assess myocardial perfusion in addition to coronary anatomy has also been investigated by Rubinshtein *et al*., who studied dual-source CT using single-photon emission CT as the reference standard.[14] MI on CT was defined as a transmural or subendocardial hypoenhancement that persisted in both diastolic and systolic reconstructions and was concordant with a coronary territory. Compared with quantitative single-photon emission CT, dual source CT evaluation for MI showed moderate sensitivity (75%) and positive predictive value (68%), but excellent specificity (98%) and negative predictive value (99%).

Peri-infarct heterogeneous zones are areas of scar with islets of viable cells interspersed among fibrosis which have been shown to be critical substrates for VT and increase cardiac arrhythmia susceptibility.[10] Their appearance on MRI is well-described,[10] and larger heterogeneous zones appear to be associated with inducible VT, with successful electrophysiology ablation of these zones causing VT to be no longer inducible.[15] Schuleri *et al*. demonstrated the appearance of heterogeneous zones on CCT in mini-pigs with induced chronic MI's.[16] They found excellent correlation of scar size as seen on CT with both post-mortem pathology (r = 0.97) and MRI (r = 0.92), and were able to detect peri-infarct heterogeneity in all 15 animals. Their findings suggest that CT may provide a more detailed assessment of the heterogeneous zones than MRI, being less susceptible to some of the partial volume effects that MRI suffers. The accuracy of CCT in this regard has yet to be borne out in human subjects.

CCT's ability to accurately identify the peri-infarct heterogeneous zone as a substrate for VT has been demonstrated in several case reports to help identify targets for electrophysiology ablation therapy, especially in patients with ICD's in whom MRI is contraindicated. Bello *et al*. reported a patient with ICM who presented with VT, in whom cardiac CT demonstrated a large transmural inferolateral infarction that correlated with positron emission tomography (PET) and voltage mapping as the VT substrate.[17] The scar was ablated, with subsequent resolution of VT and no further recurrence to date. Tian *et al*. compared CT-derived characteristics of myocardial scar and heterogeneous zone with standard voltage mapping in 11 patients with ICM and ICD scheduled for VT ablation, and assessed the feasibility of integrating CT data into clinical mapping systems to guide VT ablations.[18] Abnormal LV myocardial areas correlated well between CCT and voltage map (r = 0.77), which suggests that CCT may allow correct prediction of abnormal voltage locations in advance of the voltage mapping procedure.

Adipose tissue is also thought to contribute to the heterogeneity of post-MI LV tissue, and can be visualized on CT.[19] A significantly higher proportion of MI patients have been found to have fat deposition within the LV myocardium detectable on CCT,[20] and CT findings of LV adipose tissue have been shown to correspond to sites of prior MI's in post-MI patients.[21] Therefore, routinely searching for myocardial fat on CT may present the first opportunity to diagnose a silent MI, and may help with localizing MI sites as possible substrates for ventricular tachyarrhythmia. It may also allow for identification of other causes of myocardial fat such as arrhythmogenic ventricular dysplasia (discussed below). It should be noted that the beam hardening effect, which primarily occurs due to high-density metal or calcium situated near a low-density structure, can result in falsely decreased attenuation of normal tissue and may be misinterpreted as fibrofatty metaplasia. On cardiac CT, this artifact occurs in a very specific location on scans, namely at the posterolateral wall due to the spine. In our experience, we can assess for a typical presentation in that region and avoid interpreting it as metaplasia. The fatty metaplasia diagnosed with RV dysplasia is typically in the RV free wall, far from the posterior wall of the left ventricle.

Finally, CCT's ability to noninvasively workup obstructive CAD is a significant advantage of this modality, especially in light of the fact that 75% of sudden cardiac death is associated with CAD.[4] It has been demonstrated in large studies that CCT has a very high sensitivity for obstructive CAD, allowing non-invasive assessment with a negative predictive value that often exceeds 99% compared with invasive angiography.[9] Therefore, CCT provides the unique ability to effectively rule out obstructive CAD as an important etiology for VT. Furthermore, patients who initially undergo CCT to rule out CAD may later be evaluated for other structural etiologies of VT without the need for further re-imaging.

3. Heart failure (impaired systolic function)

Heart failure is an important etiology for ventricular tachyarrhythmias, and as briefly mentioned above, decreased ejection fraction is an important prognostic factor for patients with heart failure from any etiology.[5] Both LV dysfunction and NYHA functional class are powerful risk factors for SCD in patients with either ischemic or nonischemic cardiomyopathy, and ICD placement in these high-risk patients has been shown to improve survival.[5] In the workup for heart failure and LV dysfunction, TTE is usually the first-line imaging modality given its convenience and safety, while MRI is regarded the reference standard for measurement of global and regional myocardial function, though not widely

used given its cost and time demand.

Acquisition of CCT images via retrospective ECG gating allows for image reconstruction in any phase of the cardiac cycle, making it possible to calculate ventricular functional parameters such as LVEF and ventricular volumes. Numerous studies show excellent correlation and concordance between LV volumes and EF obtained by CCT and other modalities such as TTE and MRI.[22,23] For example, Greupner *et al*. performed a head-to-head comparison of LV function assessment between 64-row cardiac CT and 3 other imaging modalities (2D and 3D echocardiography, and cineventriculography), using MRI as a reference standard.[24] They found CCT to be superior to the other 3 modalities in assessing global LV functional parameters, showing significantly higher correlation between CT and MRI in terms of LVEF (r = 0.89) and LV volumes: end-diastolic volume (r = 0.90), end-systolic volume (r = 0.96), with the exception of stroke volume which was significantly overestimated. Cardiac CT also provides adequate visualization of right ventricular (RV) volume and function, with studies showing good correlation of RV functional parameters obtained by CCT with MRI, angiography and echocardiography.[25,26]

An important caveat to CCT's ability to workup cardiac function is that prospective ECG gating is increasingly employed in order to reduce the amount of radiation exposure, and it is not possible to assess ejection fraction and wall motion with this technique. In addition, the temporal resolution of CCT is limited compared to MRI and may impair its ability to obtain images in patients with higher heart rates.

4. Nonischemic cardiomyopathies

4.1 Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is defined as a diffuse or segmental LV hypertrophy with a nondilated and hyperdynamic chamber. The usual diagnostic criterion for HCM is a maximal LV wall thickness greater than or equal to 15 mm in the end-diastolic phase.[27] Echocardiography is the usual modality used for screening. However, cardiac CT has proven useful for evaluating cardiac morphology and function in HCM (see Figure 2). CCT's excellent spatial resolution enables detailed characterization of the cardiac chambers, which allows characterization of different subtypes of HCM, measurement of LV outflow tract diameter (though not gradient) and detection of systolic anterior motion in asymmetric HCM.[27]

CCT may also be useful for stratifying risk of HCM patients for sudden cardiac death. Important risk factors include LV maximal wall thickness of 30 mm or more, LV dilatation and depressed EF (end-stage or burned-out phase of HCM), and possibly perfusion defect.[28] In addition, CCT may be useful for screening family members of HCM patients for preclinical HCM, namely by detection of LV crypts, which are defined as blind pits extending into but confined by the myocardium.[29,30] LV crypts have been identified with a high rate of occurrence of 81% in HCM mutation carriers who had not yet developed LV hypertrophy, and are suggested to be one of the early pathologic alterations of the myocardium that ultimately progress into HCM. However, more studies are needed regarding the natural history and prognosis of this particular finding.

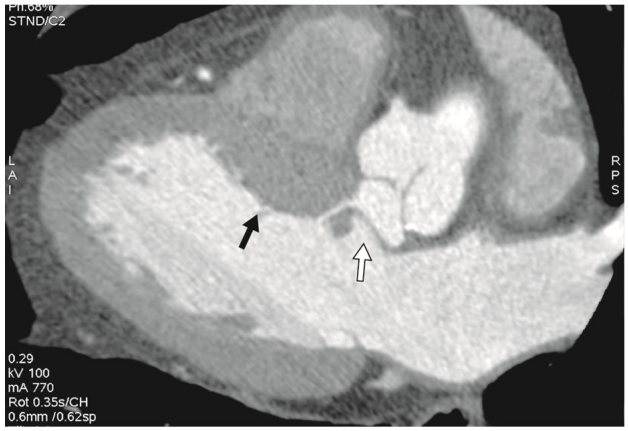

Figure 2. A three chamber view demonstrating marked thickening of the septum (black arrows) in a patient with hypertrophic cardiomyopathy and systolic anterior motion of the anterior leaflet of the mitral valve (white arrows)

4.2 Arrhythmogenic right ventricular dysplasia

Arrhythmogenic right ventricular dysplasia (ARVD) is a heart muscle disease of unknown cause that predominantly affects the right ventricle. It is characterized by myocardial atrophy and fibro-fatty replacement of the RV myocardium. Patients present clinically with RV electrical instability which leads to ventricular tachycardia or fibrillation. This may then precipitate sudden cardiac arrest, particularly in young people and athletes.[31]

An important component of the diagnosis of ARVD includes imaging of the right ventricle, which will typically reveal dilatation and reduction of RV ejection fraction with regional RV akinesias/dyskinesias, dyssynchronous RV contractions, or RV aneurysms.[32] The detection of fatty replacement of the RV myocardium by imaging is also useful in helping diagnose ARVD.[31] The 2010 ARVC/D revised task force criteria list echocardiography, MRI and contrast ventriculography as standard imaging techniques to arrive at the diagnosis of ARVD.[32] CCT's accuracy in assessing RV size and function, as well as ability to detect ventricular fatty infiltration, make it a good tool for assessing ARVD[33] (see Figure 3).

Characteristic findings on CCT include RV wall thinning, fatty infiltration and excessive trabeculations, as well as signs of RV failure such as reduced RV systolic function, regional hypokinesia and dilatation.

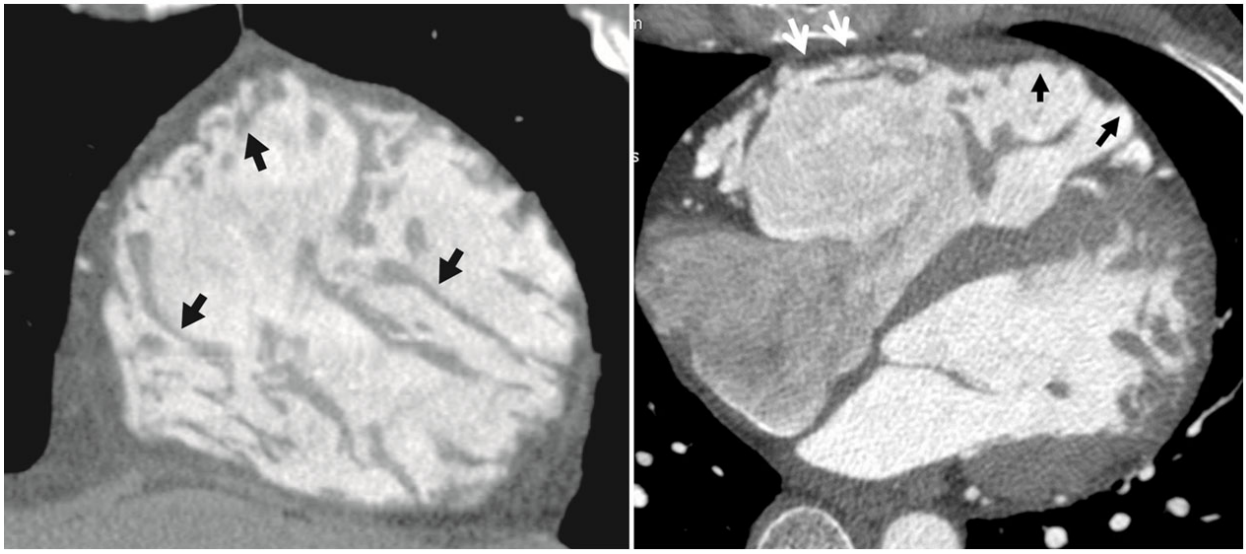

Figure 3. A patient diagnosed clinically with arrhythmogenic right ventricular dysplasia (ARVD). The image on the left (3a) demonstrates a dilated right ventricle (RV) with multiple trabeculations throughout (arrows depict trabeculations in right ventricle). The image on the right (3b) shows the right ventricle with fat in the RV free wall (white arrows), and scalloping of the RV free wall (black arrows).

4.3 Noncompaction cardiomyopathy

Noncompaction cardiomyopathy (NCM) is a rare congenital form of cardiomyopathy that results from the arrest of the normal compaction process during embryogenesis. This causes marked trabeculation of the ventricular wall, with deep recesses that communicate with the ventricular cavity. Patients present with congestive heart failure, atrial/ventricular arrhythmias, or thromboemboli.[34] Echocardiography is considered to be the diagnostic test of choice, while MRI diagnostic criteria have been published proposing that a noncompacted-to-compacted myocardial thickness ratio of > 2.3 can be used to diagnose NCM.[35]

CCT has been demonstrated by numerous case reports to be an effective modality for detecting NCM[36, 37] (see Figure 4). It is able to detect the characteristic ventricular trabeculations with deep intertrabecular recesses in noncompacted LV myocardium, and may be able to provide better visualization of trabeculations relative to echocardiogram.[38] No formal criteria exist for diagnosing NCM on CCT, although MRI criteria have been extrapolated to arrive at the diagnosis via CCT.[39]

5. Anomalous coronary arteries

Anomalous coronary arteries are an uncommon but important cause of sudden cardiac death. The mechanism behind SCD is thought to be proximal severe spasm, usually in the context of severe exertion, leading to ischemia and subsequent arrhythmia.[40] Patients in whom the proximal segment of an anomalous coronary artery courses between the aorta and pulmonary artery are at a particularly increased risk of fatal arrhythmias and sudden death,[41] while other possible indicators of risk from SCD also include acute angle of takeoff of the anomalous vessel from the aortic sinus, a slit-like orifice, an interarterial course and an intra-mural aortic segment.[7]

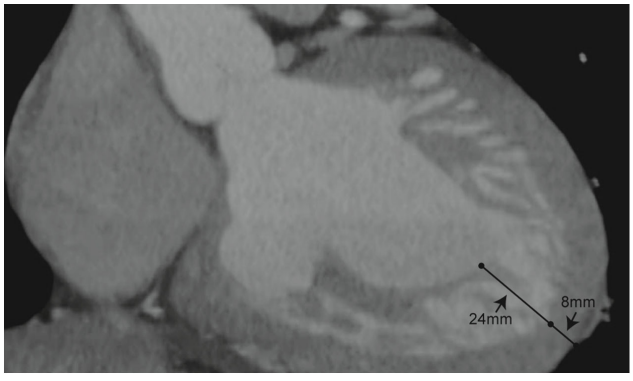

Figure 4. A coronal view of a ventricle demonstrating multiple striations consistent with noncompaction. The noncompacted tissue was 24 mm in diameter, the compacted (normal) myocardium was 8 mm.

Given its ability to noninvasively image coronary arteries, CCT is the imaging modality of choice to identify anomalous coronary artery origins or unusual angulations of the coronary artery ostia, with high accuracy and greater ease than by invasive cardiac angiography[42] (see Figure 5). Shi *et al*. reported that anomalous coronary arteries detected on conventional angiography were only 35% of those detected on 16-slice CCT.[43] Karaca *et al*. performed CCT in a group of 52 patients shown to have anomalous coronary arteries by conventional angiography and demonstrated that it easily depicted the anomalous vessels and their courses, even in those that conventional angiography failed to delineate.[44] In particular, it is able to precisely localize anomalous vessels in relation with the aorta and pulmonary artery and detect potentially malignant courses, whereas conventional angiography does not reliably define this course.

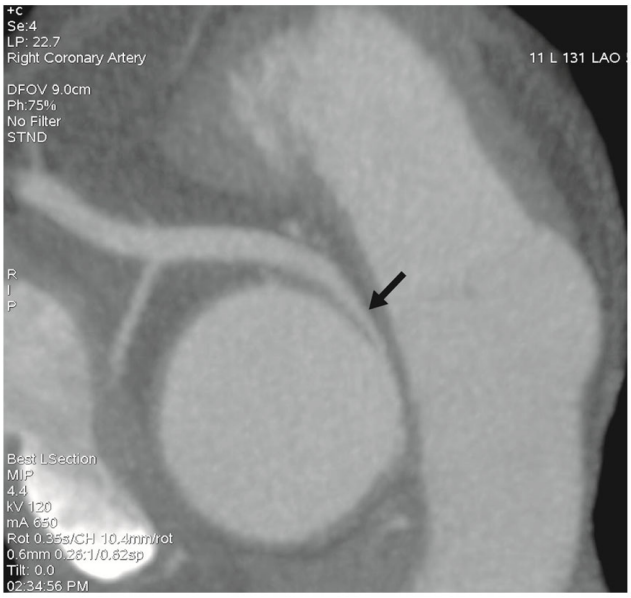

Figure 5. Anomalous coronary artery. This demonstrates a curved multiplanar image of the right coronary artery arising from the left coronary cusp and running between the pulmonary artery and the aorta (arrow depicts anomalous coronary artery).

6. Conclusion

Noninvasive cardiac imaging is a valuable tool for the workup and diagnosis of substrates for ventricular tachyarrhythmias and sudden cardiac death, and may allow for more accurate identification of patients who would benefit from therapeutic intervention to help prevent SCD, such as placement of an ICD or coronary artery revascularization. Our review of the literature suggests that cardiac CT is comparable to MRI or TTE in its ability to workup many of the structural substrates of VT, and even superior in the workup of several etiologies such as obstructive CAD or anomalous coronary arteries.

It is important to be aware that CCT has important limitations that make it unlikely to be first-line for many of the disease processes discussed. CCT requires the use of nephrotoxic contrast as well as significant exposure to radiation. Use of prospective triggering as a dose sparing technique is more commonly employed today, which reduces effective radiation doses by 80%,[45] though this does not permit assessment of ejection fraction and wall motion. On the other hand, most of the etiologies related to SCD/VT are still assessable with prospective imaging, as it still allows visualization of the myocardium, chamber size and CAD.

Although it may not be considered a first-line modality for working up cardiac disease given its limitations and adverse effects, CCT may be a viable alternative to MRI and other imaging modalities for the detection of numerous etiologies of VT, and may be especially valuable in patients who would benefit from cardiac imaging but cannot tolerate MRI or have contraindications such as pacemakers. CCT that is performed to rule out obstructive CAD yields much additional information about cardiac anatomy and function that may lead to the discovery of predisposing risk factors for VT/SCD, and should always be examined carefully with these disease entities in mind.

Conflicts of Interest Disclosure

Dr. Matthew Budoff receives a grant from General Electric. Dr. Danny Yoonsang Lee and Dr. Marc Girsky declare no conflicts of interest.

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