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EDITORIAL COMMENT

CMR-determined scar volume: predictive for ventricular tachycardias?

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Abstract The interesting data reported by Bernhardt et al. strengthen the diagnostic benefit of CMR in patients with ischemic cardiomyopathy. Consequently, the presence, location and size of the CMR-determined scar tissue may be used for better risk stratification in patients with ischemic cardiomyopathy eligible for ICD therapy.

Keywords Cardiovascular magnetic resonance · Scar tissue · Ventricular tachycardias · Implantable · Cardioverter defibrillator

Over the past years considerable progress has been made in the field of cardiovascular magnetic resonance (CMR), providing accurate evaluation of left ventricular function particularly in patients prone to heart failure due to both ischemic and non-ischemic cardiomyopathy [1–14]. In particular, high-resolution contrast-enhanced CMR has been used to visualize myocardial fibrosis with a high accuracy [15–22]. For instance, in patients with acute myocardial infarction, the injured myocardium shows increased CMR contrast compared to normal myocardium when imaged by delayed gadolinium enhancement. The transmural extent of delayed gadolinium enhancement predicts functional outcome after interventional procedures performed in patients with acute myocardial infarction and chronic ischemic heart disease [23–32]. Not only in the setting of an acute myocardial infarction, but also in patients with various manifestations of cardiomyopathy, evidence of delayed gadolinium enhancement may have important clinical and prognostic implications [32–36]. In patients eligible for cardiac resynchronization therapy, the presence of scar tissue may have consequences for implanting a biventricular device and subsequent accurate lead positioning [37–51]. In patients with hypertrophic cardiomyopathy, the presence of fibrotic tissue may result in increased occurrence of ventricular arrhythmias [52–57]. Recently, it has been shown that presence and quantity of late enhancement is correlated with cardiovascular events and arrhythmias in patients with dilated cardiomyopathy [58–61].

In the present issue of the *International Journal of Cardiovascular Imaging*, Bernhardt et al. [62] address the hypothesis that infarct size in patients with ischemic cardiomyopathy is related to spontaneous monomorphic ventricular tachycardia which may be determined by contrast-enhanced CMR. The purpose of the study was to establish the relationship between

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functional and contrast-enhanced CMR findings and spontaneous ventricular tachy-arrhythmias in patients with ischemic cardiomyopathy who underwent implantable cardioverter-defibrillator (ICD) therapy. A total of 41 patients with ischemic cardiomyopathy and an indication for ICD therapy underwent delayed gadolinium enhancement CMR for quantification of left ventricular volumes, function and myocardial scar tissue before subsequent implantation of an ICD device. During a follow-up period of 1,184 ± 442 days 68 monomorphic and 14 polymorphic types of ventricular tachycardia could be observed in 12 patients. Patients with monomorphic ventricular tachycardia had more extensive scar tissue than patients with polymorphic ventricular tachycardia (mean 25% vs. mean 12% of myocardial mass). Moreover, myocardial infarction involved more segments in the region of the left anterior descending coronary artery (LAD) than in patients with polymorphic ventricular tachycardia (86 vs. 20%). During the long-term follow-up period, patients with spontaneous monomorphic ventricular tachycardia had more infarcted tissue, being more often present in the LAD region, than patients with polymorphic events. As a result, the most important finding of the study was a significant difference between patients with monomorphic and polymorphic events. Patients with monomorphic ventricular arrhythmias during follow up had more dilated ventricles, decreased ejection fractions and larger scar volumes than patients with polymorphic events. More segments in the LAD region showed scar patterns in patients with monomorphic events. This larger extent of scar tissue in the LAD region was independent of the indication for ICD implantation compared to patients with polymorphic ventricular arrhythmias.

Several studies have recently focused on the relation between the extent of myocardial scar tissue and the incidence of ventricular arrhythmias in patients with ischemic cardiomyopathy. Fernandes et al. [63] determined whether mechanical behavior of left ventricular wall segments containing different degrees of scar tissue and localized at different distances from the interface between infarcted and noninfarcted myocardial tissue could assist in predicting inducibility of monomorphic ventricular tachycardia in 46 patients with ischemic cardiomyopathy. They showed that inducible patients had more infarcted and border zone sectors and a shorter time to peak circumferential shortening strain than did noninducible patients in the border zone of adjacent and infarcted regions. Yokokawa et al. [64] sought to clarify the relationship between the scar characteristics and occurrence of sustained ventricular tachycardias in 47 patients with advanced heart failure. The volume of the hyperenhanced areas and total number of hyperenhanced segments were greater in patients with sustained ventricular tachycardias than in those without. The presence and magnitude of the nontransmural scar tissue predicted sustained ventricular tachycardias in patients with advanced heart failure. Di Bella et al. [65] investigated the relationship between nonsustained ventricular tachycardia and left ventricular dilatation, function, remodeling, and CMR-assessed scar tissue extent in patients with previous myocardial infarction. It was shown that necrotic and viable myocardium coexistence within the same wall segments predicted occurrence of nonsustained ventricular tachycardia in patients without left ventricular dilatation, whereas left ventricular mass and end-systolic volume were predictors of nonsustained ventricular tachycardia in those with left ventricular dilatation. Roes et al. [66] recently studied 91 patients with ischemic cardiomyopathy for a mean duration of 8.5 months after ICD implantation for ICD therapy in spontaneous ventricular tachycardias. The authors showed that patients with appropriate ICD therapy had larger myocardial scar detected by CMR than patients without appropriate ICD therapy. They concluded that infarct tissue heterogeneity on contrast-enhanced CMR was the strongest predictor of spontaneous ventricular arrhythmia with subsequent ICD therapy (as surrogate of sudden cardiac death) among other clinical and CMR variables in patients with previous myocardial infarction. However, the authors did not distinguish between monomorphic and polymorphic ventricular tachycardia, and rather addressed ICD therapy itself.

Apart from the value of scar identification in patients with left ventricular dysfunction, delayed gadolinium enhancement CMR can also be used to assess the long-term arrhythmia recurrence rate and outcome in patients with scar-related ventricular tachycardia of right ventricular origin [67–69]. Wijnmaalen et al. [69] studied 29 (45%) patients diagnosed with arrhythmogenic right ventricular dysplasia according to Task Force criteria (TF+)

and 35 (55%) with right ventricular scar of undetermined origin (TF−) at the end of follow-up (64 ± 42 months). The authors demonstrated that scar-related right ventricular tachycardias have a high recurrence rate in TF+ and TF− patients. Patients presenting with a fast index ventricular tachycardia are at high risk for recurrence of fast ventricular tachycardias and may benefit most from ICD therapy.

To summarize, the interesting data reported by Bernhardt et al. [62] strengthen the diagnostic benefit of using CMR in patients with ischemic cardiomyopathy. Consequently, the presence, location and size of the CMR-determined scar tissue may be used for better risk stratification in patients with ischemic cardiomyopathy eligible for ICD therapy.

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