Letter by Assenza et al Regarding Article, “Electrocardiographic Features of Arrhythmogenic Right Ventricular Dysplasia”

To the Editor:

We read with interest the article by Jain et al 1 about the ECG features of arrhythmogenic right ventricular cardiomyopathy (ARVC). The authors tested the diagnostic performance of different surface ECG features in a cohort of patients with a previous clinical diagnosis of ARVC based on 1994 Task Force criteria (recently, revised Task Force Criteria have been proposed). 2 They included 15 patients with so-called incomplete right bundle-branch block and 17 patients with complete right bundle-branch block. Including patients with right ventricular conduction delay is very relevant because the 1994 Task Force consensus statement on ARVC diagnosis does not specify how to apply ECG criteria to patients with complete or incomplete right bundle-branch block.

Recently, Steriotis et al 3 published a study on ECG patterns in ARVC. Their study showed that a pathognomonic ECG pattern in ARVC does not exist. A wide spectrum of ECG abnormalities can be found in this disease. They are likely to be related to a unique balance of progressive and heterogeneous loss of electric forces secondary to myocardial atrophy, anisotropic conduction caused by fibrofatty replacement, abnormal electric gradient, and RV dilatation. One new finding of this study is that low QRS voltages, previously considered consistent with the severe form of the disease, were present even in patients with milder ARVC phenotypic expression.

This aspect was not addressed by Jain et al but is of interest in that all the representative ECG samples in their article show low QRS voltages in the precordial leads compared with control. Additionally, the authors do not address the fact that right precordial T-wave inversion is an age-dependent finding and can be found in normal teenagers. 4 Thus, the algorithm would have to be used with caution in those <18 years of age.

The presence of low QRS voltages in ARVC is likely the consequence of progressive myocardial atrophy, fibrofatty replacement, and RV dilatation. This possible pathophysiological link between this ECG finding and myocardial substrate is also suggested by the analysis of ECG in Ebstein anomaly. In this disease, apical displacement of the tricuspid valve orifice is associated with an atrialized portion of the RV that is thin and fibrotic. The ECG pattern in Ebstein anomaly is similar to ARVC, although in the former severe atrial enlargement is often present 5 The association between low QRS voltage in precordial leads in 2 very different diseases, both characterized by RV myocardial atrophy and RV dilatation, is intriguing in regard to its potential as a prognostic marker. In ischemic and nonischemic cardiomyopathy, ventricular arrhythmias have been shown to be caused by reentry, which occurs in scar zones characterized by low-amplitude, fractionated endocardial electrograms. Low QRS voltage can be considered, in this setting, the macroscopic consequence of this microscopic disruption of the electric myocardial activity and a potential predictor of disease progression.

Further analysis of this ECG parameter in ARVC could help identify a possible relevant clinical association between low voltages with specific morphological features or clinical outcome.

Disclosures

None.

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