Editorials

Electrocardiographic risk stratification in dilative cardiomyopathy: an unfulfilled promise

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Idiopathic dilated cardiomyopathy represents the substrate for approximately 10% of sudden cardiac deaths in the adult population[1]. Mortality in patients with dilated cardiomyopathy ranges between 10% and 50% annually, mainly determined by the severity of the disease. Tamburro and Wilber compiled 14 studies including a total of 1432 patients and demonstrated an average mortality rate over 4 years of 42%; 28% of all fatalities were classified as sudden deaths[2]. According to other investigators, sudden death may indeed be responsible for up to 50% of fatalities particularly in patients with NYHA class II or III heart failure[3].

Recently, it has been demonstrated that in patients with a history of sustained ventricular tachycardia or aborted sudden death, the implantable defibrillator is superior to antiarrhythmic pharmacotherapy in prolonging life[4,5]. In these studies, a significant proportion of enrolled patients suffered from dilated cardiomyopathy as the underlying heart disease. Even in dilated cardiomyopathy patients without aborted sudden death but with syncope as the initial presentation in whom no arrhythmias could be provoked at electrophysiological testing, there was a high incidence of appropriate device therapy during a follow-up of 24 ± 13 months[6]. It appears conceivable, therefore, that device therapy may similarly be capable of reducing sudden death in patients with dilated cardiomyopathy without prior ventricular tachyarrhythmic episodes or syncope. However, accurate identification of patients with dilated cardiomyopathy being at high risk of arrhythmogenic death is mandatory since prophylactic ICD implantation in all patients presenting with this type of structural heart disease will not be feasible. Moreover, widespread application of such an approach requires non-invasive, inexpensive, and easy to perform risk stratification methods.

Accordingly, in recent years numerous studies have been published examining this important issue. In general, the results of many of these studies have been contradictory. For instance, the presence of spontaneous non-sustained ventricular tachycardia has been reported to be a risk marker in dilated cardiomyopathy patients in some studies, whereas others could not confirm this[3]. Simple ECG-derived findings, such as the presence of atrial fibrillation[7] or left bundle branch block[8] have also been suggested as risk markers but their predictive value was low. Similarly, several studies used the signal averaged ECG in order to detect non-invasively areas of slow conduction within the diseased ventricular myocardium of dilated cardiomyopathy which are thought to represent the substrate for reentrant arrhythmias. However, results obtained by conventional time-domain analysis of the SAECG were rather disappointing. For instance, in one of the largest studies, Turitto et al. reported a positive predictive value of SAECG analysis of 17% with a sensitivity of only 22% in 80 patients with dilated cardiomyopathy[9]. In another study in 64 patients, these values were 13% and 20%, respectively[10]. Similar to risk stratification in survivors of acute infarction[11], conventional analysis of the SAECG was of limited value in the prediction of arrhythmic events in patients with dilated cardiomyopathy.

In this issue, Yi and associates report their findings in 82 patients with dilated cardiomyopathy using a more sophisticated analysis of the SAECG, namely wavelet decomposition[12]. This novel technique has several advantages over conventional SAECG evaluation since it can detect small and transient irregularities hidden within the QRS complex. Accordingly, it can for instance be used in patients with left bundle branch block which has been found in dilated cardiomyopathy to be a marker of risk by itself. Yi et al. followed their 82 patients for an average of 23 ± 18 months, during which time five patients died from sudden death and 23 developed progressive heart failure which resulted in heart transplantation in 14. Kaplan–Meier analysis demonstrated that significantly more patients with an abnormal result of wavelet decomposition showed progressive heart failure compared to those with normal findings. At 3 years, survival free from progressive heart failure was
44.6% in patients with, compared to 85.5% in patients without abnormal findings on wavelet decomposition[12]. However, even this sophisticated SAECG analysis technique was unable to identify patients prone to sudden arrhythmic death. Although the number of arrhythrogenic end-points in this study was relatively low, it adds further evidence that SAECG analysis is highly unlikely to yield much prognostic information with respect to arrhythmogenic death in patients suffering from dilated cardiomyopathy. This lack of prognostic value of the ECG-derived risk assessment is similar to that obtained in survivors of acute myocardial infarction in the era of acute reperfusion therapy[11].

Despite this negative result, careful and sophisticated analysis of the ECG should continue to be explored in order to establish other parameters which could potentially be used as risk stratifiers. There is now growing evidence that disturbances in the ventricular repolarization process may be a common feature in heart failure[13] and may be caused by altered expression of various ionic channels responsible for repolarization[14]. Accordingly, research efforts should focus on evaluation of changes in repolarization, as assessed from the ECG. Perhaps the most simple approach in this direction is represented by the analysis of interlead QT dispersion in the surface ECG. However, it has already been demonstrated that this analysis is subject to numerous technical limitations. Accordingly, its predictive value for arrhythmic events in patients with dilated cardiomyopathy[15] or coronary disease[11] is limited. However, other methods, most notably analysis of subtle changes in T wave morphology[16] or amplitude[17] may represent a research direction worthwhile to pursue.

To ascertain the yield of prognostic information of these new ECG-derived risk stratifiers, well-designed studies with prospectively defined cut-off values of the various risk parameters and end-points are very much needed. In summary, therefore, we should not slacken our effort to identify patients with dilated cardiomyopathy at high risk of arrhythrogenic death since the implantable defibrillator provides a very potent therapeutic strategy. Further analysis of the surface ECG, applying sophisticated computer technology to assess the ventricular repolarization phase, may eventually result in more accurate risk stratification in dilated cardiomyopathy.

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References