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EDITORIAL

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Editorial to QRS fragmentation as a potential new marker for subclinical myocardial dysfunction

Narrow QRS duration (<120 ms) with subtle abnormality presenting morphological fragmentation within QRS complex (fQRS) defined by several ECG features (ie, the presence of an additional R wave as R′, notching in the nadir of the R wave or the S wave, or the presence of >1 R′ fragmentation in two contiguous ECG leads) observed in some patients with ischemic heart diseases signifies conduction disturbances and indicates myocardial scar.[1] Furthermore, it has been proposed that the fQRS location (ie anterior or lateral) may be outcomes relevant.

fQRS determined by using resting 12-lead ECG in clinical practice, a very old and conventional morphological ECG pattern, has recently garnered interest in diverse cardiovascular disorders. fQRS has been identified as a marker or an arrhythmic predictor in subjects with coronary artery disease.[1,2] The existence of fQRS is strongly associated with the presence of local or scattered myocardial scars, which possibly explains the inhomogeneous ventricular activation pattern observed in fQRS.[1] The rationale underlying these findings might be that fractionated electrograms from altered electrical property or patchy myocardial scar result in intra-myocardial conduction delay through the muscle bundles separated by interstitial fibrosis that may contribute to re-entry substrate formation. As the frequency of fQRS is significantly higher in hypertensive patients compared to normotensives and likely serves as a predictor of left ventricular hypertrophy accompanied by worse systolic and diastolic functions even in the absence of other structural heart diseases, the existence of fQRS might only be clinically meaningful as a marker of myocardial fibrosis in certain population with higher cardiovascular risks.[3]

Notably, almost all of these studies assessed patients with declined and reduced ejection fraction or impaired LV contractile function rather than healthy individuals or in those with relatively preserved ejection fraction. Dehghani et al[4] reported in a case-control study (age- and sex-matched) of a healthy population that fQRS was associated with lower subclinical myocardial contractility marker of global ventricular longitudinal strain (GLS). By using speckle-tracking, a highly sensitive marker for detecting systolic myocardial dysfunction even prior to overt chamber dilation. In this study, both worsened GLS and smoking history independently predicted the presence of fQRS. Overall, findings from the study of Dehghani et al highlight the diagnostic and prognostic significance of fQRS as an emerging clinical risk marker for identifying preclinical myocardial damage. Given the fact that the presence of fQRS in apparently healthy middle-aged population may not confer greater cardiovascular risks except for those presenting fQRS in lateral leads with known structural cardiac diseases, it is unclear whether the observed attenuated subclinical systolic dysfunction associated with fQRS signifies certain cardiovascular risks.

What remains unanswered in this study would be, whether the observed functional decline of longitudinal strain may reflect the possible greater amplitude of intra-myocardial dys-synchrony because of altered electromechanical coupling. Dys-synchronized myocardial contraction pattern has shown to cause inefficient global ventricular pump function leading to are reduced peak global LV longitudinal strain value even when global LV ejection pump function remains preserved.[5] Furthermore, the authors could not perform subgroup analysis for fQRS locations because of their limited sample size. Therefore, further studies in larger populations are necessary to confirm whether fQRS can be used in clinical practice to detect myocardial dysfunction in individuals without prevalent heart failure or known cardiovascular diseases.

CONFLICT OF INTEREST

Authors declare no conflict of interests for this article.

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