THE ANATOLIAN JOURNAL OF CARDIOLOGY

Official journal of the TURKISH SOCIETY OF CARDIOLOGY

Fragmented QRS Pattern in Patients with COVID-19: Further Insights into Its Temporal and Mechanistic Features

LETTER TO THE EDITOR

Fragmented QRS (f-QRS) pattern has been regarded as a promising electrocardiographic (ECG) risk-stratifier in various clinical conditions including dilated cardiomyopathy, etc.[1,2] Pathophysiologically, this phenomenon is uniformly agreed to denote an existing myocardial injury and scar formation leading to an eventual derangement in ventricular depolarization.[1,2] In their recently published article, Özdemir et al[1] have documented the prognostic value of f-QRS in the prediction of all-cause mortality in patients with severe coronavirus disease 2019 (COVID-19). Within this context, we would like to make further comments on their study findings and potential implications of f-QRS in the setting of COVID-19.

First, temporal emergence of f-QRS pattern might significantly differ in patients with COVID-19: Accordingly, f-QRS might be regarded as a pre-existing (serving as a marker of underlying cardiovascular disease) or a new-onset phenomenon (emerging as a consequence of COVID-19-related factors) in these patients. In this regard, pre-existing f-QRS might be predominantly ascribed to clinical[2] or subclinical myocardial disease. However, as the authors mentioned in the section of study limitations, there were no previous ECG data of study participants.[1] Of note, we hold the opinion that f-QRS pattern might be considered as a pre-existing ECG sign in a portion of patients with COVID-19 potentially suggesting that increased mortality in these patients is largely due to the aggravation of underlying myocardial disease during the course of COVID-19 (mostly manifesting as acute heart failure, malignant arrhythmias, etc.). However, clinical studies are strongly warranted to make a comparison between pre-existing and new-onset f-QRS patterns in terms of their prognostic value in patients with COVID-19.

Second, new-onset f-QRS pattern might arise as a reversible (transient) or irreversible (permanent) phenomenon in patients with COVID-19. Of note, hyperinflammation might be considered as the key trigger of new-onset f-QRS pattern[1] regardless of whether it is transient or permanent. Pathophysiologically, irreversible f-QRS is mostly associated with scar tissue formation due to myocardial injury associated with COVID-19-related factors including hyperinflammation, hypoxia, etc.[1,2] On the other hand, reversible f-QRS in patients with COVID-19 might possibly be attributable to transient disturbances in cardiac ion channels (leading to interruption of myocardial depolarization and possibly repolarization currents) potentially associated with the direct impact of certain cytokines on myocardium usually in the absence of scar formation. In this context, certain cytokines including tumor necrosis factor-α, etc., were previously suggested to be associated with abnormal calcium handling (with prolonged action potential durations) and sodium channel dysfunction (associated with disturbed myocardial depolarization) along with abnormalities in repolarization parameters including corrected QT interval (QTc) and transmural repolarization of myocardium).[3,4]

In the study by Özdemir et al[1], association of f-QRS pattern with more prolonged QTc intervals might possibly suggest a predominant pattern of reversible f-QRS due to cytokine impact across their study population. Accordingly, did the authors

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Cite this article as: Taylan G, Öztürk C, Yalta K. Fragmented QRS pattern in patients with COVID-19: Further insights into its temporal and mechanistic features. Anatol J Cardiol 2022;26(3):239-240.

DOI:10.5152/AnatolJCardiol.2021.1265

plan a long-term serial ECG examination of survivors with a f-QRS pattern following their discharge to determine the incidences of reversible and irreversible f-QRS patterns? Moreover, prolonged QT interval might have significantly contributed to arrhythmic mortality particularly in patients without significant myocardial scar formation (hence with a reversible f-QRS pattern). In this context, evaluation of certain novel indices including index of cardiac electrophysiological balance index (ratio of QTc/QRS) might help determine the relative contribution of QTc interval to arrhythmic mortality in patients with COVID-19.[5] On the other hand, hyperinflammation is also a well known trigger of coronary microvascular dysfunction[4] that might potentially be associated with certain ECG changes including ST segment depression in the present study.[1] Importantly, coronary microvascular dysfunction generally presents with a coronary slow flow (CSF) kinetics as demonstrated on coronary angiogram (CAG).[6] Therefore, we wonder whether the authors performed any emergent CAG with a subsequent diagnosis of CSF in their study population.

Finally, the COVID-19 assessment for survival at admission (CASA) has been recently suggested as a useful index for the prediction of in-hospital mortality in patients with COVID-19.[7] This index comprises certain parameters on admission including levels of albumin and troponin, D-dimer, white blood cell count, glomerular filtration rate, PaO_2/FiO_2, and respiratory rate.[7] Of note, CASA index, despite its high sensitivity (98.6%), was shown to have a moderate specificity (69%) in the prediction of COVID-19-related mortality.[7] Therefore, integration of f-QRS evaluation into CASA index might significantly enhance the power of its specificity and hence, positive predictive value in the evaluation of COVID-19 mortality.

In conclusion, f-QRS pattern might have important prognostic implications in patients with COVID-19. On the other hand, this phenomenon might also have diverse temporal and mechanistic characteristics in these patients. However, further studies are still needed to confirm the clinical implications of f-QRS pattern in the setting of COVID-19.

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