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COVID-19 Disease and its Electrocardiographic Manifestations: Our Experience

To the Editor:

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) responsible for the recent pandemic is clinically manifested as Coronavirus disease 2019 (COVID-19). Although COVID-19 has a predominant effect on the respiratory system, it also has deleterious impacts on other organs, especially the cardiovascular system.[1] Cardiovascular manifestations of COVID-19 include cardiac arrhythmias, heart failure, cardiomyopathy, myocarditis, cardiac injury, myocardial infarction, cardiogenic shock, and venous thromboembolism.[2] We are presenting these comments to highlight our experience about the effect of COVID-19 on electrocardiograms (ECGs) observed at an academic center. Apart from the obvious ECG findings of, for example, arrhythmias and ST-elevation myocardial infarction in COVID-19 patients, we have noticed 3 different patterns of ECG in 3 different categories of COVID-19 disease.

Category 1 includes COVID-19 patients with a mild disease; symptoms are mostly cough, fatigue, and low-grade fever, without imaging evidence of pneumonia. These patients are stable and managed on the medical floors or at home. ECG in mild disease is usually benign and may show sinus tachycardia, as is shown in Figure 1. Sinus tachycardia is likely the compensatory response of the body to the mild febrile illness. These patients carry a good prognosis if they remain mildly symptomatic without developing pneumonia.

Category 2 includes patients in whom there is evidence of COVID-19 pneumonia; these patients are sicker and usually are managed in the intensive care unit. The ECG in this group of patients usually shows the pattern of ST elevation in lead III/aVF and V1, along with S1Q3 morphology in lead I and lead III. This is likely due to the rightward vector shift in the frontal plane as a result of right ventricular volume or pressure overload due to COVID pneumonia,

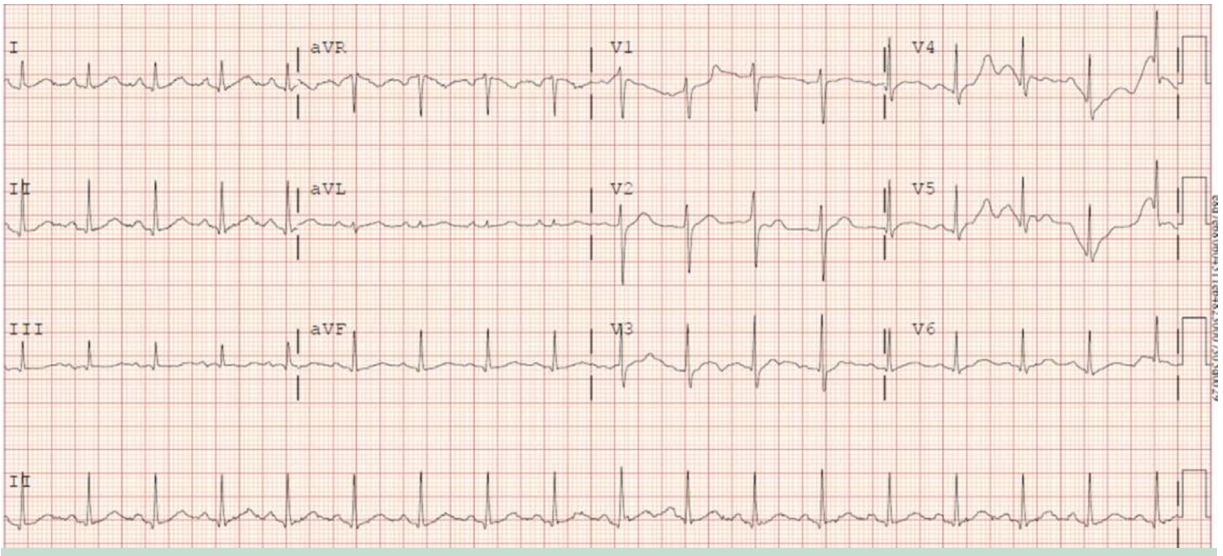

Figure 1 Sinus tachycardia with nonspecific ST-T wave changes in COVID-19 disease without pneumonia.

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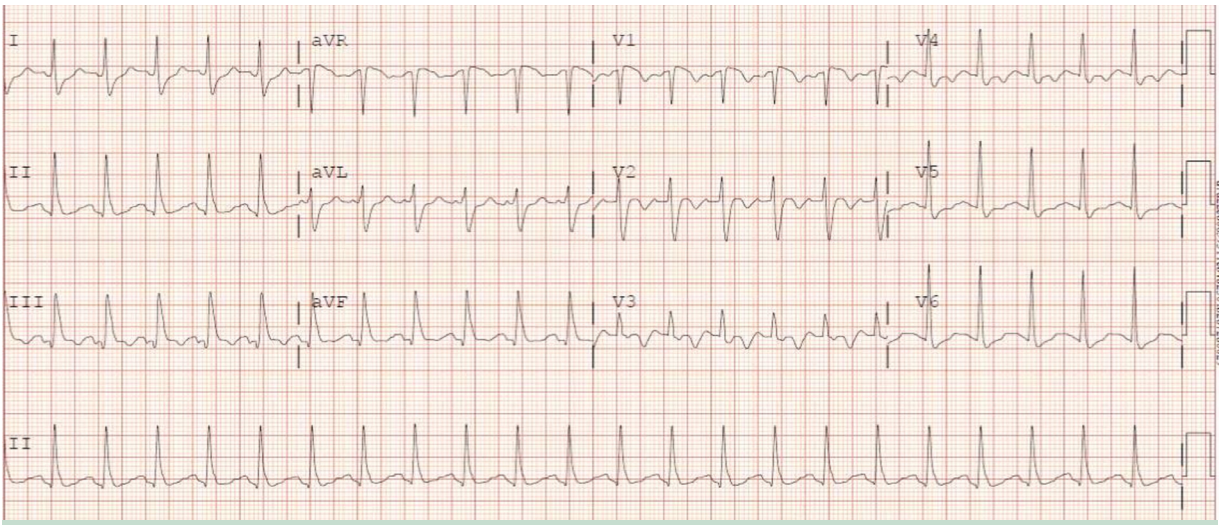

Figure 2 Sinus tachycardia with ST elevation in lead III/aVF and V1, along with S1Q3 morphology in lead I and lead III, in a patient with COVID-19 pneumonia.

and is shown in Figure 2. In patients with venous pulmonary embolism the S1Q3T3 pattern is observed in lead I and lead III, but when COVID-19 pneumonia has coexisting venous pulmonary embolism, the ECG in these patients can show a pattern of ST elevation in lead III/aVF and V1, along with S1Q3 morphology in lead I and lead III, as is shown in Figure 3. Searching the medical literature, we found this pattern has been reported before and portends a bad prognosis, and is also a predictor of cardiac arrest in patients with pulmonary disease.[3]

Category 3 includes patients with evidence of myocarditis along with COVID-19 pneumonia; in our experience this category has the worst prognosis. The ECG in this group shows diffuse ST-segment elevation with a prolonged QTc interval. The diffuse ST elevation is likely due to involvement of the left ventricle in myocarditis and does not have localization to coronary distribution. The ECG shown in Figure 4 shows precordial and inferior leads ST elevation with hyperacute T waves. This patient had nonobstructive coronary artery disease on coronary angiography, and cardiac magnetic resonance imaging demonstrated myocarditis.

There are also other potential mechanisms of QTc prolongation in COVID-19 patients, possibly due to

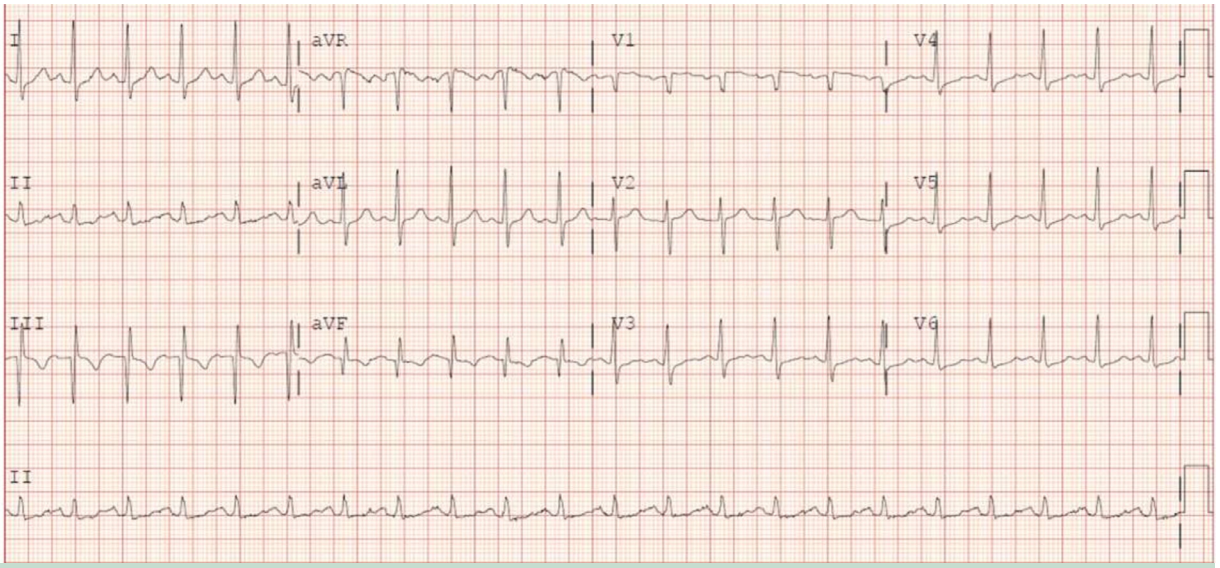

Figure 3 Sinus tachycardia with ST elevation in lead III/aVF and V1, along with S1Q3 morphology in lead I and lead III, in a patient with COVID-19 pneumonia and coexisting pulmonary embolism.

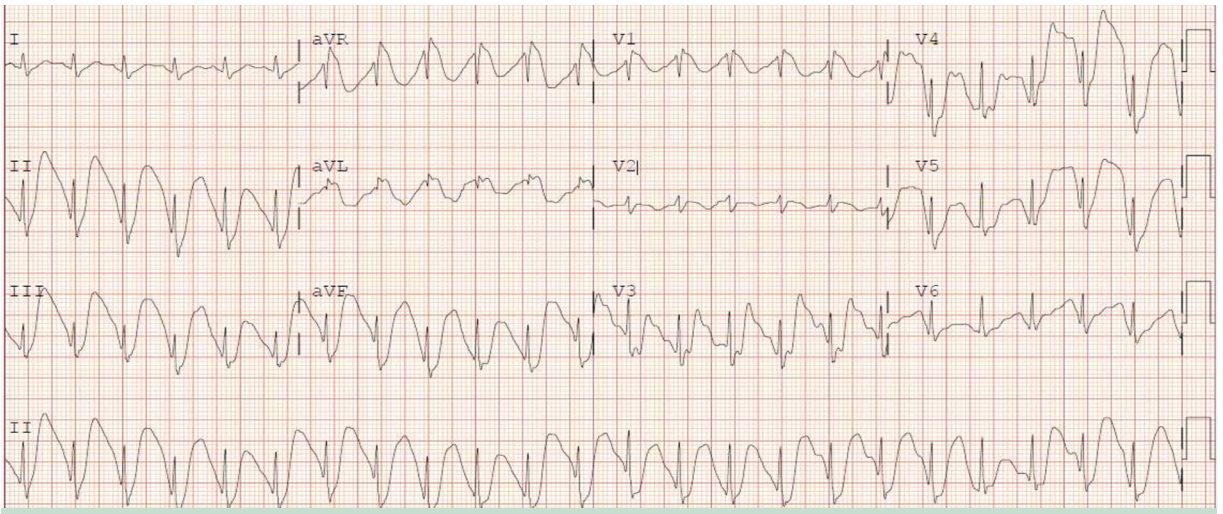

Figure 4 Sinus rhythm with inferior and precordial ST-segment elevation with hyperacute T waves in COVID-19 myocarditis.

inflammation and drugs. The interleukin-6 was observed to block the rapid inward rectifying channel (IKr) by inhibiting the hERG (Ether-à-go-go-related gene; alternative nomenclature, KCNH2) leading to prolonged QTc interval.[4,5] The same mechanism of IKr blockade also is reported in drug-induced QTc prolongation, and in COVID-19 patients the use of hydroxychloroquine and azithromycin along with other drugs prolongs the QTc interval, probably by this mechanism.[6]

In short, we report that various severities of COVID-19 disease have different presentations on the surface ECG. COVID-19 disease without pneumonia causes sinus tachycardia, while COVID-19 pneumonia causes rightward vector shift on the surface ECG and of ST elevation in lead III/aVF and V1, along with S1Q3 morphology in lead I and lead III. The myocarditis that causes the diffuse ST elevation and prolonged QTc interval on ECG has the worst prognosis, in our experience.

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