LETTERS TO THE EDITOR

To the Editor—Is it really COVID-19?

We read with great interest the article by Chang and colleagues,[1] in which they reported COVID-19 infection unmasking Brugada syndrome but they report, finally, that it is the fever that is unmasking the Brugada pattern. We want to congratulate the authors on their article, which demonstrates that COVID-19 infection may be another etiology to expose a Brugada pattern.

We agree with most of the statements provided in the present work, but our main concern is the lack of rigor, since it is nothing new that fever is capable of unmasking Brugada pattern and this hallmark has already been previously reported in large cohort series. Dumaine and colleagues[2] were the first to link temperature with the function of a mutant SCN5A sodium channel. In 2002, Porres and colleagues[3] reported that an increase in temperature may be the most important factor in revealing the electrical anomalies and activating ventricular arrhythmias. Two studies demonstrated that in unselected populations with fever, type 1 Brugada electrocardiography may be seen. Mizusawa and colleagues[4] demonstrated that patients who had fever-induced type 1 electrocardiography have an increased risk for syncope and ventricular fibrillation. Michowitz and colleagues[5] reported that premature inactivation of the sodium channel is accentuated at higher temperatures, suggesting that febrile states may unmask certain Brugada syndrome patients or temporarily increase the risk of arrhythmia, mainly in Caucasian male patients. So, we would like it to be learned that any condition that predisposes to fever may unmask a Brugada pattern—not just the COVID-19 infection.

Ivan Hernandez Betancor, PhD, MD* (ivanhbet@hotmail.com), Josué A. Orozco Garduño, MD†, Ruben A. Juarez Prera, PhD, MD*, Francisco Bosa Ojeda, PhD, MD*

**Department of Cardiology, Hospital Universitario de Canarias, La Laguna, Santa Cruz de Tenerife, Canary Island, Spain; †Department of Cardiology, Hospital Clínico San Carlos, Madrid, Spain*

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Author's Reply—Is it really COVID-19?

Thank you for the opportunity to expand on the discussion of Brugada syndrome in patients with Coronavirus 2019 (COVID-19). As Betancor and colleagues astutely pointed out, COVID-19-induced fever was depicted as the potential cause of electrocardiographic changes in our patient.[1]

However, while fever has been unequicovally proven to alter the SCN5A sodium channel and cause Brugada patterns on the electrocardiogram (ECG),[2] there are still many unknowns about the novel virus and how we should manage these vulnerable patients. For instance, why did our patient's first ECG upon admission with no fever show type I Brugada pattern while the last ECG after defervescence, back to his initial temperature on admission, show narrower QRS complex on the right precordial leads with near-resolution of the coved ST elevation in lead V_2? COVID-19 may directly impact the myocardium, manifesting with changes on the ECG and echocardiogram suggestive of myocardial ischemia and/or inflammation.[3] As the Brugada group noted, what if the virus has a higher propensity to directly affect the myocardial sodium channels compared to other previously encountered respiratory virus infections that caused SARS (severe acute respiratory syndrome) and MERS (Middle East respiratory syndrome)?[3] What about potential preexisting myocardial scarring contributing to our patient's presentation in the setting of the infection?

The incidence of Brugada pattern on ECG and the clinical syndrome are expected to increase during this pandemic, in which patients commonly present with febrile illnesses. While fever is without a doubt a major contributing factor—and hence the emphasis on early treatment for defervescence—these are some of the unanswered questions surrounding COVID-19 that need to be further scrutinized.

David Chang, MD (davidchang7787@gmail.com), Laurence M. Epstein, MD

Division of Electrophysiology, Department of Cardiology, Northwell Health, Manhasset, New York

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