The Known into the Unknown: Brugada syndrome and COVID-19

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Coronavirus Disease 2019 (COVID-19) infection outbreak has been recently declared as pandemic by the World Health Organization (1). The healthcare organizations of numerous countries in the world are currently stretched in order to offer the best care to the patients who contract the infection of COVID-19, fighting at the same time with the penury of both resources and knowledge. The amount of scientific data which have been collected from the spread of the infection and, in particularly, in the last month, on the pathophysiology and on possible medical treatments of this frequently lethal viral infection is remarkable, but still insufficient to declare victory against the virus. That’s the reason why all major scientific journals, including *JACC: Case Reports*, have opened quickly dedicated sessions to the research and the investigations performed on this very hot topic.

We have therefore to thank all the Authors who have found the time in this difficult moment to sit in front of a computer and share their experience on COVID-19, on top of their very undoubtedly demanding clinical duties. In line with this, we congratulate Prof. Vidovich to have published recently on *JACC: Case Reports* a case of Brugada syndrome (BS) associated with COVID-19 infection (2). The known into the unknown, precisely.

COVID 19 infection presents mainly as respiratory syndrome which include pneumonia and in the worst scenario acute respiratory distress syndrome (ARDS) (3). We have learnt also that, in a not negligible amount of cases, the virus can provoke myocardial ischemia and/or inflammation, in association or also without the respiratory syndrome (4). There are already numerous cases of COVID-19 infections presenting as STEMI, which have triggered the activation of the primary percutaneous coronary intervention (PCI) protocols. The cause of this ST elevation is unknown: it has been linked to the traditional plaque rupture in those cases which have required coronary angioplasty, but it has been suggested that myocarditis or microvascular thrombosis could be the
cause when no obvious thrombus or coronary flow interruption is detected. If all this was not sufficient, here you go also Brugada type I pattern interfering and complicating the life of our interventional cardiologists. In the case presented by Vidovich, indeed, patient presented with shortness of breath, substernal chest pain and fever. The ECG showed a Brugada-type I pattern in the right precordial leads with no reciprocal changes; the presence of chest pain, shortness of breath and reduction of systolic left ventricular function assessed with a 2-D echocardiogram imposed to perform an urgent coronary angiography, which excluded an ongoing acute coronary syndrome. No significant electrolyte imbalance was found. The conclusion of the Authors was that the Brugada type I pattern, completely unknown to the patient until this admission, was unmasked by the COVID-19 viral infection and the ongoing fever. Confirming that when it rains it pours, the patient suffered also from an episode of supraventricular tachycardia, which is also another clinical feature of the BS and confirmed the final diagnosis.

A link between fever and Brugada type I pattern is very well known and has been described extensively (5,6,7). The international guidelines on sudden cardiac death recommend in fact lowering as soon as possible the body temperature in those patients with an established diagnosis of Brugada syndrome and also in carriers of the mutations with proved inducible Brugada type I pattern (8). The increase in the body temperature has indeed been proven to cause a higher degree of inactivation of Na+ channels, both mutated and wild ones: in the subjects who are genetically predisposed, this reduced Na+ flow can result into a dangerous transmural heterogeneity which is the basis for phase 2 reentry ventricular arrhythmias and sudden death (9,10). It would be of interest as well to understand if the virus itself could interact directly with the myocardial ion channels and provoke the ECG modification typical of Brugada syndrome.
Take home message is therefore that patients with Brugada and concomitant COVID-19 infection should be monitored in ICU or in the telemetry ward till the fever is resolved, regardless of their respiratory conditions. Further research would be needed to help clinicians to navigate this uncharted sea.
REFERENCES


