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The arrhythmogenic face of COVID-19: Brugada ECG pattern during acute infection

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Case description

A 53-year-old female was admitted to our hospital with fever up to 39.7°C, cough, myalgia, fatigue, and diarrhoea. She had received an allogeneic renal transplant in 2017 due to minimal change nephropathy without any signs of transplant dysfunction since then. Quantitative reverse transcription–PCR (RT–qPCR) analysis of naso- and oropharyngeal swabs provided evidence of SARS-CoV-2 infection, and chest CT scan showed bilateral pulmonary parenchymal ground-glass opacities of the dorsal lower lobes (Supplementary material online, *Figure S1*). Laboratory abnormalities comprised lymphopenia, elevated ferritin levels, and signs of cytokine activation. Under medical treatment with ceftriaxone, caspofungine, and hydrochloroquine, fever declined to subfebrile temperatures, and the patient

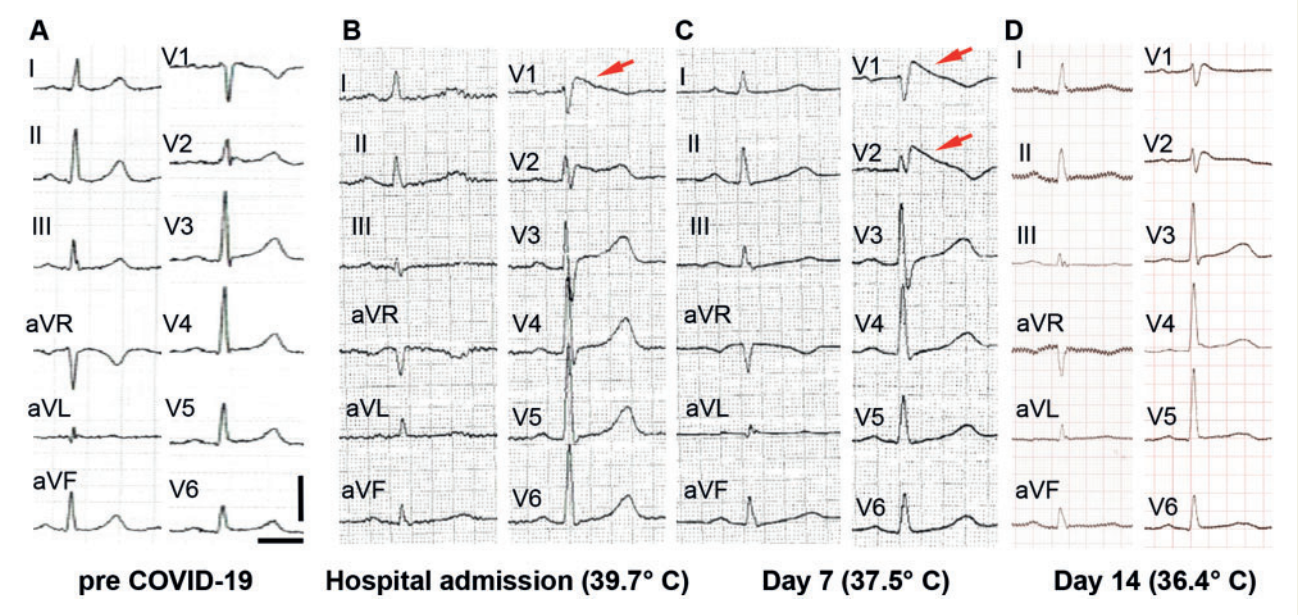

Figure 1 12-lead ECGs 3 months prior to COVID-19 infection (*A*), at hospital admission (*B*), and at follow-up day 7 (*C*) and day 14 (*D*). Note aggravation of the Brugada type I ECG pattern in leads V1 and V2 with disease progression (red arrows). Specific body temperature at the time of each ECG are depicted at the bottom, if applicable. Scale bars: horizontal 200 ms, vertical 1 mV.

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stabilized on a light to moderate disease level, not requiring respiratory support.

While high-sensitivity cardiac troponin T (8 pg/mL; normal range <14 pg/mL), and N-terminal probrain natriuretic peptide (NT-proBNP) (75 ng/L; normal range <125 ng/L) levels were normal, 12-lead ECG revealed coved ST-segment elevation in lead V1 at the day of admission with a rise of the J-point by 0.25 mV indicative for Brugada type 1 ECG (*Panel B*). Due to potential proarrhythmic side effects, therapy with hydrochloroquine was discontinued immediately after ECG diagnosis. Typical ECG changes were absent from previous ECGs (*Panel A*), suggesting an association with the infection. The patient denied syncope, dizziness, or palpitations in the past, and there was no history of arrhythmic diseases in her family. The Brugada ECG pattern aggravated with disease progression, and the J-point elevation increased up to 0.45 mV in leads V1 and V2 at 1 week after admission (*Panel C*), although temperature remained on a subfebrile level. Simultaneously, interleukin-6 (IL-6) levels increased continuously (Supplemental material online, *Figure S1*). Circulating IL-6 is known to be closely linked to the severity of COVID-19. Continuous ECG monitoring did not reveal ventricular arrhythmias, and echocardiography displayed no overt structural, valvular, or functional changes. In the further course, IL-6, ferritin, and NT-proBNP levels decreased and a second SARS-CoV-2 RT–qPCR revealed a negative result. Accordingly, Brugada ECG pattern resolved (*Panel D*).

In the present case, Brugada ECG pattern appeared first after COVID-19 infection and was not clearly related to fever. The authors speculate that the known cytokine flush in symptomatic COVID-19 patients or direct cardiotoxicity of the virus may contribute to the typical ECG changes. The patient was monitored until the J-point elevation disappeared, and appointments for ambulatory follow-up visits were made. In conclusion, thorough ECG and cardiological monitoring of COVID-19 patients is clearly advocated to recognize potentially life-threatening cardiac involvement of the disease.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

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