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Visual Diagnosis in Emergency Medicine

FEVER UNMASKING BRUGADA-PATTERN ELECTROCARDIOGRAM IN PATIENT WITH CORONAVIRUS DISEASE 2019 (COVID-19)

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INTRODUCTION

Brugada syndrome is an inherited form of dysrhythmia characterized by the presence of a specific electrocardiogram (ECG) pattern that is associated with a higher risk of sudden cardiac death (1). If patients are asymptomatic, it is called Brugada pattern. We present a case of a patient with coronavirus disease 2019 (COVID-19) unmasking Brugada pattern.

CASE REPORT

A 43-year-old Filipino man, who was otherwise healthy and without previous history of cardiovascular disease, presented to the Emergency Department (ED) with fever, nonproductive cough, myalgia, and shortness of breath for 3 days. He also noted having mild pleuritic chest pain with inspiration, however, this resolved prior to arrival in the ED. He had been working on a cruise ship for the past 2 months and was quarantined along with other members of the ship who had similar symptoms. His review of systems was otherwise unremarkable, specifically denying palpitations. His vital signs showed a blood pressure of 129/69 mm Hg, heart rate 95 beats/min, respiratory rate 24 breaths/min, O₂ saturation of 95% on room air, and a temperature of 39.4°C (102.9°F). His physical examination revealed a well-

developed, well-nourished man in no distress, slightly tachypneic but speaking full sentences, and no use of accessory muscles. Lung examination was significant for equal breath sounds bilaterally without wheezing, stridor, rales, or rhonchi. Cardiac examination showed regular rate with normal S1 and S2 without murmurs, rubs, or gallops. His ECG was significant for 3–4-mm ST elevations, specifically, rSR' pattern, followed by an inverted T wave in leads V1 and V2, consistent with Type 1 Brugada pattern (Figure 1). After the patient's fever resolved with acetaminophen in the ED, the repeat ECG showed sinus tachycardia but the Brugada pattern had resolved (Figure 2). His high-sensitivity troponin was normal (8 ng/L), blood natriuretic peptide was normal (<50 pg/mL), and electrolytes were normal, other than mild hypokalemia (3.4 mmol/L). Chest x-ray study was significant for focal perihilar and patchy airspace opacities concerning for multifocal pneumonia. A nasopharyngeal swab was performed with a positive result for COVID-19, and he was admitted and treated for acute hypoxemic respiratory failure and pneumonia. The patient required intubation and was enrolled in the remdesivir trial at our institution. His respiratory status and pneumonia improved, and he was eventually discharged with plans to follow up with Cardiology as an outpatient. Ultimately, it was determined that the patient did not require an implantable cardioverter defibrillator (ICD).

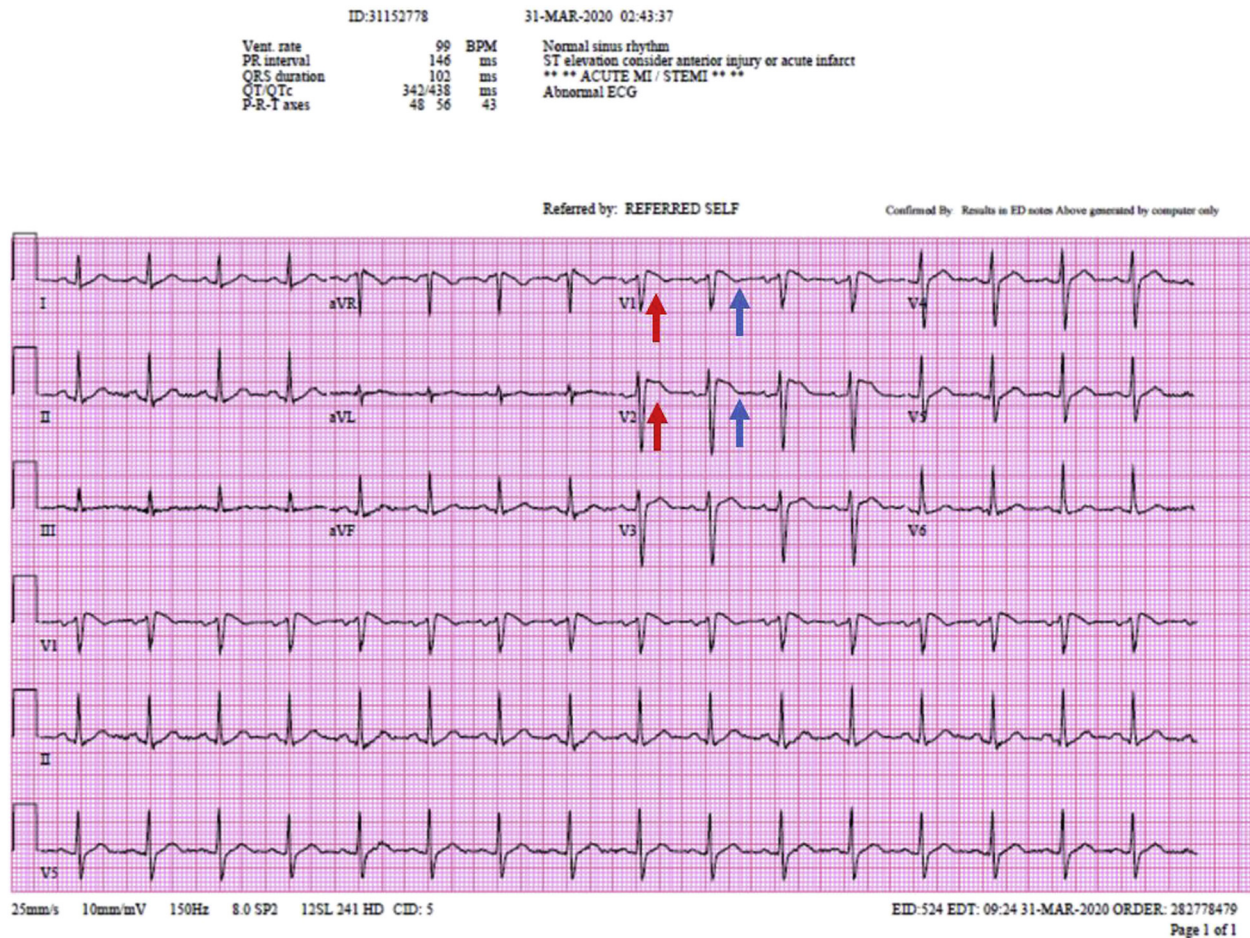


Figure 1. Patient's electrocardiogram (ECG) at presentation with fever of 39.4°C (102.9°F). ECG was significant for 3–4-mm ST elevations, specifically rSR' pattern (red arrow), followed by an inverted T wave (blue arrow) in leads V1 and V2, consistent with Type 1 Brugada pattern.

DISCUSSION

Brugada syndrome is a sodium channelopathy characterized by the presence of ECG showing right bundle branch block and ST-segment elevation in the precordial leads (1). Brugada syndrome is diagnosed by the specific ECG pattern along with either family history of sudden cardiac death in a family member that is < 45 years old or type 1 ECG in relatives, dysrhythmia-related symptoms (syncope, seizure, nocturnal agonal respiration), or ventricular tachycardia or ventricular fibrillation (VF), and patients are considered to have Brugada pattern if asymptomatic (1,2). Brugada syndrome is categorized into three types. Type 1 (coved type) involves ECG showing ≥ 2 -mm ST-segment elevation in one or more right precordial leads (V₁ to V₃) followed by an inverted T wave, occurring either spontaneously or after provocative drug test with sodium-channel blocker (flecainide, procainamide); type 2 (saddle-back type) has ≥ 0.5 -mm ST-segment elevation in one or more precordial leads

followed by a saddle-back morphology to the ST elevation; and type 3 can show either coved or saddle-back morphologies, but with < 2-mm ST-segment elevation (2,3). Type 1 is diagnostic of Brugada syndrome, whereas Types 2 and 3 are suggestive (3). Fever is one of the common triggers of Brugada pattern (4). When Brugada syndrome is suspected, physicians should consult a cardiologist and consider ICD placement, depending on their risk of VF, though this risk stratification continues to be a controversy (5).

COVID-19 is a newly recognized infectious disease that has spread across the world, presenting mainly with fever, pneumonia, and respiratory failure. Recent studies in Wuhan, China have linked this viral infection with increased risk of dysrhythmia and was associated with higher risk of fatal outcomes and in-hospital mortality (6,7). To the best of our knowledge, it is a new finding that fever associated with COVID-19 can unmask Brugada pattern. A recent case report in New York described similar findings in a COVID-19-infected patient with

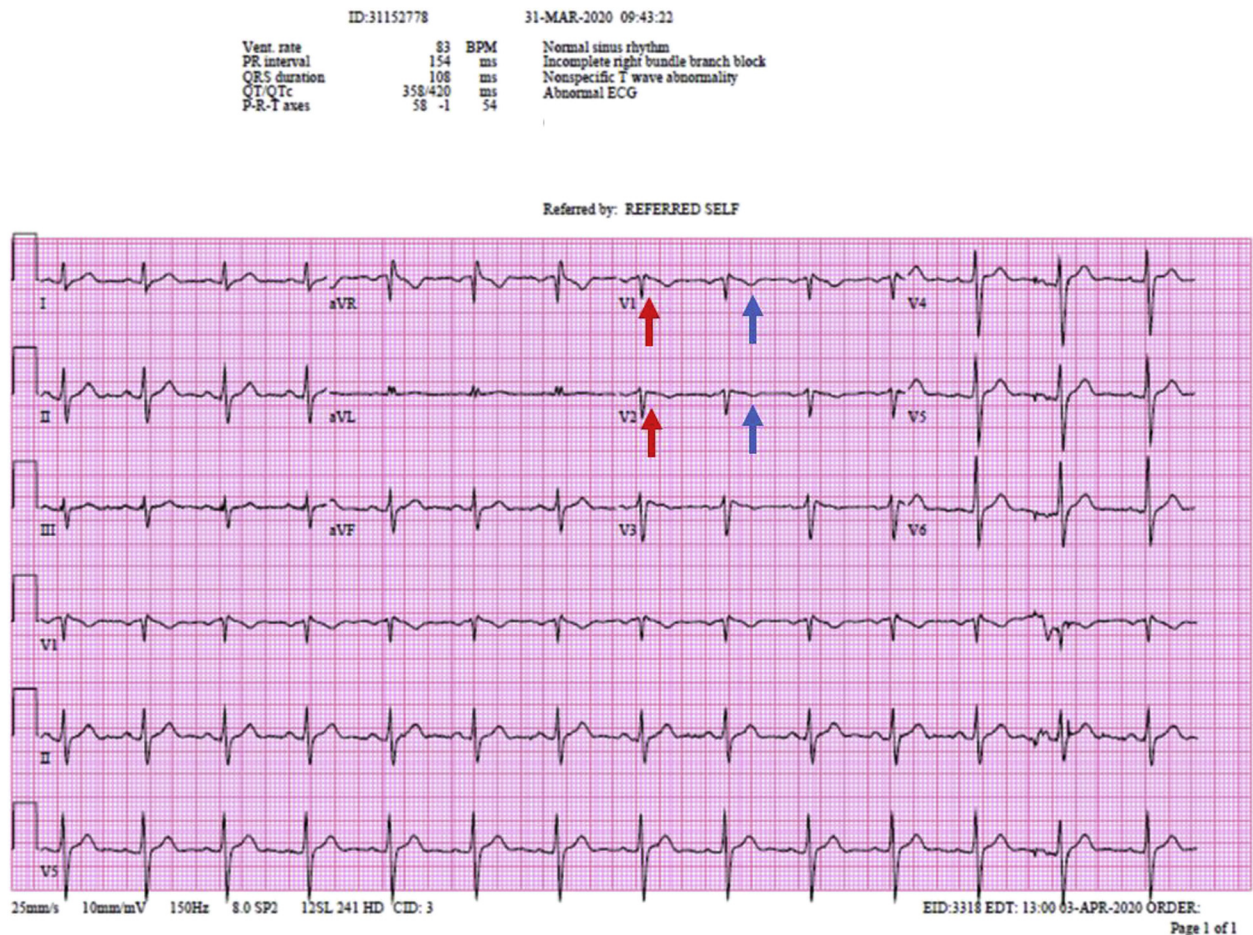


Figure 2. Patient's electrocardiogram (ECG) after fever resolved. ECG was significant for sinus tachycardia, but the Brugada pattern had resolved. Specifically, the ST elevation in V1 and V2 is no longer present (red arrow). Residual T-wave inversion is still present at V1 and V2 (blue arrow).

fever who was found to have Brugada syndrome (8). Undiagnosed Brugada syndrome has an estimated mortality rate up to 10% per year, and spontaneous Brugada pattern in an asymptomatic patient has a cumulative risk of VF up to 12% at 10 years (9,10). It is unclear at present whether patients who develop Brugada pattern in association with COVID-19 are at similar risk.

CONCLUSION

We present this case to increase awareness of the potential association between COVID-19 and the development of Brugada pattern on ECG. It is important to detect increased risk of dysrhythmia in these patients, as it seems to be associated with a higher risk of in-hospital mortality.

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