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CASE REPORT

Transient Brugada-Like Electrocardiogram Configuration Provoked by COVID-19 Fever

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ABSTRACT

There are varieties of cardiac complications which can occur in COVID-19 encompassing acute coronary syndrome, myocarditis to arrhythmia. We report a case of COVID-19 infected patient who presented with a high fever which provokes transient Type 1 Brugada-like ECG configuration. Brugada syndrome is a potentially life-threatening arrhythmia which could lead to sudden cardiac death secondary to ventricular arrhythmias. Being commonly asymptomatic, there are many triggers which could lead to its manifestations and subsequent complications. While fever, being one of the triggers, is the most common presentation of COVID-19. Besides, it could be multifactorial, either from the variant systemic complications in COVID-19 or from the management itself, especially common drugs used in resuscitation which could provoke the Brugada pattern. Looking at this association, ECG monitoring then becomes important in COVID-19, especially in those with chest pain and fever. Proper treatment for fever in COVID-19 is prudent to avoid malignant arrhythmia.

INTRODUCTION

Brugada syndrome was first published in 1992 by Pedro and Josep Brugada with a characteristic Electrocardiogram (ECG) pattern of right bundle branch block (RBBB) and persistent ST = segment elevation. It commonly presents with syncope and sudden

cardiac death due to ventricular arrhythmias (Brugada & Brugada, 1992). Mutations in SCN5A and SCN10A genes account for more than 50% of Brugada syndrome (Mizusawa & Wilde, 2012). Though its inheritance form is an autosomal-dominant trait with incomplete penetrance, most patients are undiagnosed until middle age and the average age is 40 (ranging from 1 – 77 years). It is more endemic in the Southeast Asia population and males are more prone to have this syndrome than females (8:1) (Hermida et al., 2000). Hallmark of ECG pattern in leads V_{1-3} for Brugada syndrome type 1 is coved ST-segment elevation ≥2mm followed by T wave inversion, type 2 is ≥2 mm of saddleback shaped ST-elevation or ≥1 mm of J-point elevation and type 3 is < 1mm of ST-segment elevation without any underlying structural heart disease, ischemia or electrolyte imbalance (Vohra & Rajagopalan, 2015). Fever is a very well-known cause to unmask Type 1 Brugada pattern in symptomatic patients which can lead to ventricular arrhythmia (Adler et al., 2013). COVID-19 is the current pandemic disease which is known to result in many systemic complications, including the cardiovascular system, in which there are many reports of cardiac complications encompassing acute coronary syndromes, myocarditis to arrhythmias (Wu et al., 2020). Fever is one of the most common presentations of COVID-19 infection. We report a case of COVID-19 infected patient who presented with a high fever which provokes a transient Type 1 Brugada type ECG pattern.

CASE PRESENTATION

A 49-year-old Chinese man was admitted to the hospital after presenting with a high fever, non-productive cough, breathlessness and sharp central chest pain for six days. He did not have any history of syncope, light-headedness or palpitation. He has a background history of bronchial asthma, hypothyroidism and hypercholesterolemia. He regularly takes Levothyroxine 75 micrograms once a day, Omeprazole 20 mg once a day, Simvastatin 40 mg once a night orally, Beclomethasone 100 micrograms twice a day and Salbutamol 100 micrograms four times a day metered dose inhalers. He is an ex-smoker and occasionally drinks alcohol less than 14 units per week. There is no family history of sudden cardiac death at a young age. He was born in the United Kingdom whereas his parents were originally from Hong Kong. On examination, he was fully conscious and not in respiratory distress. His initial vital signs were as follows: temperature of 38.6°C, blood pressure of 132/89 mm Hg, heart rate of 107 beats per minute, respiratory rate of 20 per minute, and oxygen saturation of 91% under room air. He needed 2 L oxygen support via nasal prong oxygen to maintain saturation above 96%. Lung examination showed coarse crackle in bilateral lower lobes. Heart sounds were normal and there was no murmur. Other system examinations were unremarkable.

SARS-CoV-2 RNA polymeric change reaction test was positive on admission. Chest radiograph and computed tomography of the pulmonary artery showed moderately extensive peripheral patchy ground glass opacities in all lobes and segments with upper lobe and posterior predominance which is consistent with COVID-19 pneumonitis (Figure 1). Initial resting ECG showed sinus tachycardia with RSR' pattern RBBB morphology, >2 mm cove-shaped J point ST-elevation with T wave inversion in V_{1-2} (Type 1 Brugada pattern) and borderline prolonged QT_C interval of 0.45 sec [normal range for male: 0.36 – 0.44 sec] (Figure 2). The subsequent ECGs were similar to admission ECGs during the intermittent spiking of fever. After three days of admission when temperature sided below 38°C, repeated ECG showed a resolution of coved-shaped ST-elevation but persistent RSR' pattern, T wave inversion in V_1 and prolonged QT_C interval of 0.49 sec (Figure 3). Initial blood investigation showed a white cell count of 5.6 (4 – 11 × 10^9/L), haemoglobin of 153 (115 – 160 g/L), platelet count of 162 (150 – 400 × 10^9/L), lymphocyte count of 1.2 (1.0 – 4.5 × 10^9/L), prothrombin 12.9 (10 – 14 s), INR 1.2 (1), D-dimer 257 (0 – 250 ng/mL), ferritin 3127 (12 – 200 mcg/L),

sodium 132 (135 – 145 mmol/L), K 3.7 (3.5 – 5.3 mmol/L), Urea 2.4 (2.5 – 6.7 mmol/L), creatinine 87 (70 – 100 μmol/L), eGFR 81 (>90), albumin 43 (35 – 50 g/L), LDH 958 (70 – 250 U/L), troponin <13 (<13 ng/L), CRP 32 (<10 mg/L), free T4 14.0 (9 – 22 pmol/L), TSH 7.46 (0.5 – 4.2 mU/L). A transthoracic echocardiogram revealed normal biventricular function with a normal size of all four chambers, and no regional wall motion abnormalities or valvular abnormalities.

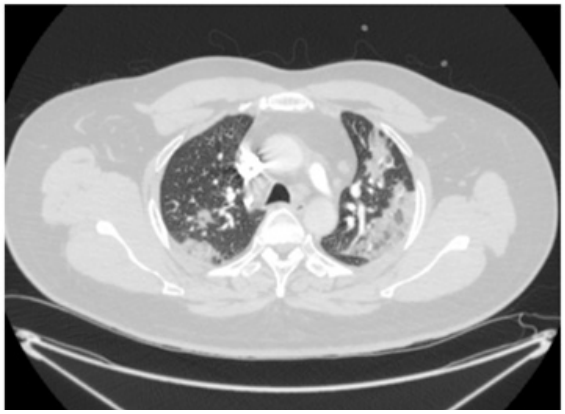

Figure 1 Computed tomography of the pulmonary artery showed moderately extensive peripheral patchy glass ground opacities in both lungs which is consistent with COVID-19 pneumonitis

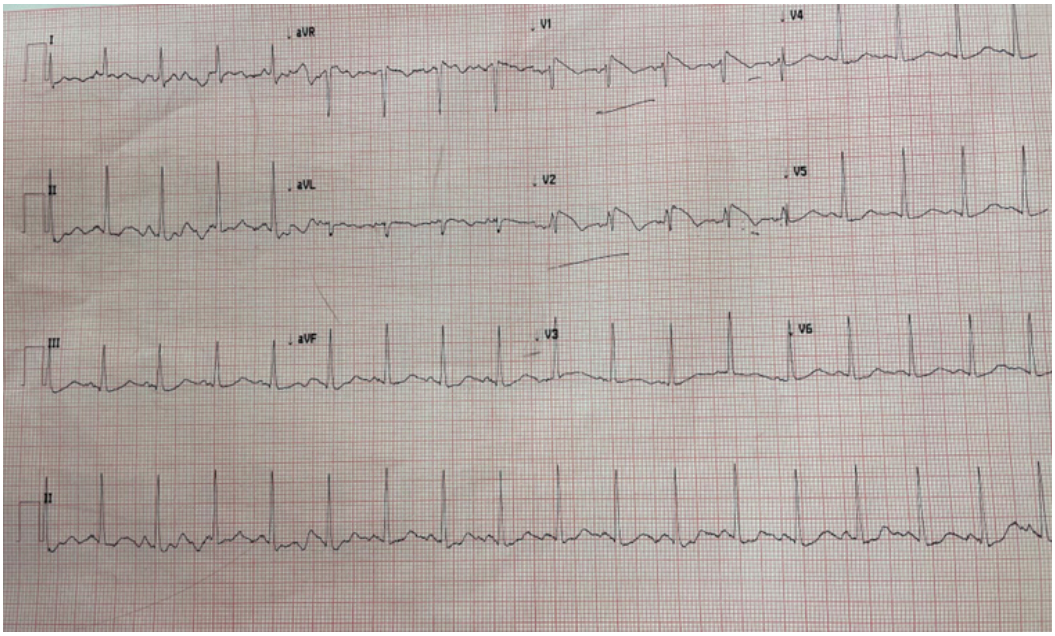

Figure 2 ECG shows sinus rhythm with partial right bundle branch block (RBBB) pattern and cove-shaped ST-elevation followed by T wave inversion in V_{1-2} (Type 1 Brugada pattern)

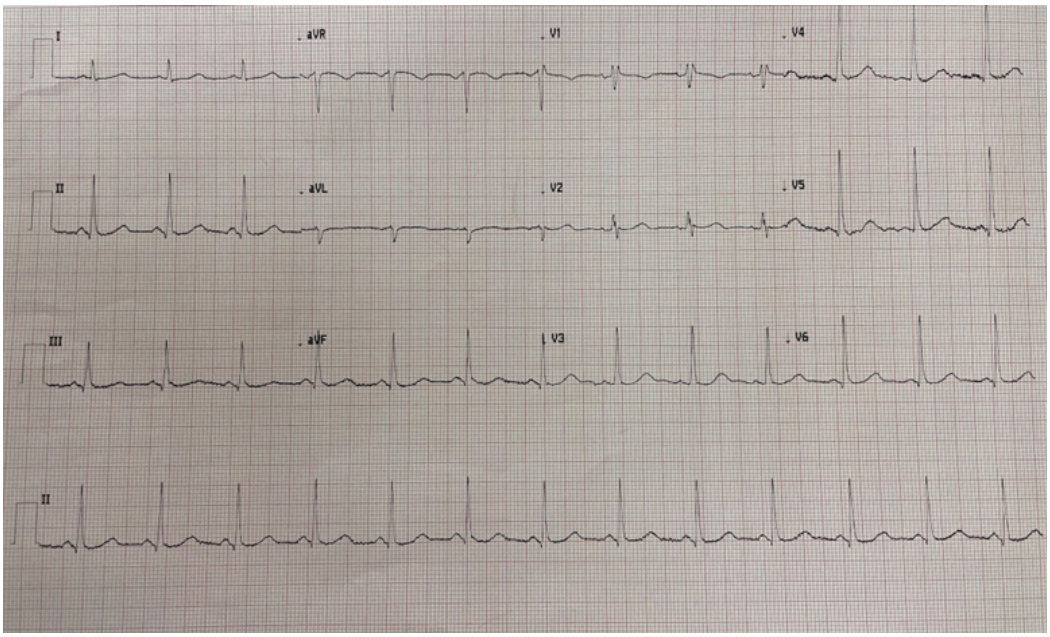

Figure 3 ECG shows normal sinus rhythm with RBBB morphology and borderline prolonged QT interval

He received intravenous antibiotics of amoxicillin 1 gram three times a day, methylprednisolone 80 mg once a day, Remdesevir 100 mg once a day, oral clarithromycin 500 mg twice a day and paracetamol 1 gram four times a day. He was put on a cardiac monitor to observe heart rhythm especially when the fever rises above 38°C. After 10 days of admission, he recovered from COVID-19 infection and was discharged with electrophysiologist follow-up in view of the absence of risk factors. He has been advised to treat fever aggressively and to avoid excessive alcohol. He was provided with a list of medications that he needs to avoid which might trigger arrhythmia. He has been explained the incidental ECG finding, the small risk of dangerous arrhythmias and sudden cardiac death.

DISCUSSION

A literature review conducted by Roomi et al. (2020) revealed that the Brugada ECG pattern (RBBB with cove or saddle-shaped ST-segment elevation in the right precordial leads) is commonly associated with pneumonia (30%) who has a fever (83%), cough (21%), sore throat (10%), and chest pain (7%). Fever can alter the ionic currents during action potential at the cardiac sodium channel during higher temperatures which can unmask the Brugada pattern (Adler et al., 2013). Brugada-like ECG pattern often disappears when the fever subsides (Baranchuk et al., 2012). With COVID-19 becoming a global pandemic and is known to cause high temperatures with respiratory symptoms, it is a new finding that fever associated with COVID-19 can unmask Brugada pattern ECG findings. A study by Wang et al. (2020) showed that arrhythmias occurred in 17% of hospitalized COVID-19 patients, which is quite significant for all clinicians to look into. The relationship between COVID-19 and arrhythmias could be attributed to multiple pro-arrhythmic factors in COVID-19, namely fever, electrolyte imbalance, stress and antiviral use (Korlipara et al., 2021). Brugada syndrome is one of the possible arrhythmias to happen in COVID-19, which should be highlighted more as it could be fatal and especially when most of the pro-arrhythmic factors are possible provoking factors for manifestation of Brugada syndrome. There are few similar case reports in patients with COVID-induced fever found to have Brugada syndrome (Kim et al., 2020;

Korlipara et al., 2021). Our case demonstrates that high fever (>38.5°C) due to COVID-19 pneumonia can induce tachycardia, Brugada pattern ST elevation and prolonged QT interval which warrants the clinician to further investigate for Brugada syndrome. One study thus recommended that in the management of COVID-19 patients, there should be a low threshold for ECG monitoring for any arrhythmias (Van de Poll & van der Werf, 2020). Wu et al. (2020) demonstrated that the risk of developing fever-induced life-threatening arrhythmia in Brugada syndrome is associated with a symptom which is syncope as well as age, in which particularly young and old patients have a higher risk. It has been recommended that patients with Brugada pattern ECG during COVID-19 infection, patients should be monitored intensively until the fever is resolved (Pasquetto et al., 2020). Also, it is important to monitor body temperature regularly and treat it appropriately with regular antipyretics to prevent fatal cardiac arrhythmia (Wu et al., 2020). Nevertheless, asymptomatic patients with fever-provoked Brugada patterns do not need antiarrhythmic drugs or intracardiac defibrillator placement.

CONCLUSION

In conclusion, a history of chest pain in COVID-19-infected patients is crucial as it may indicate COVID-19-related cardiac complications. COVID-19 fever in a middle age male is thought to be the provocative trigger for the Brugada ECG pattern, possibly due to the direct effect of the virus on the myocardial ion channel. ECG monitoring is important in COVID-19 infection, especially during high fever and should be continued monitoring till the fever subsides. Fever should not be taken lightly and should be treated aggressively and promptly in COVID-19 pneumonitis to avoid malignant arrhythmia triggered by Brugada syndrome.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest to publish this case report.

CONSENT

Written consent was obtained from the patient to publish this case report. A copy of the written consent is available for review by the Chief Editor.

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