COVID-19: An Emerging Etiology for the Brugada Pattern on EKG

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Abstract

Brugada syndrome is an inherited arrhythmogenic disorder characterized by autosomal dominant mutations in several identified genes encoding for cardiac voltage-gated sodium channels. The underlying mutations predispose individuals to ventricular tachycardia, ventricular fibrillation, and sudden cardiac death. Oftentimes, the condition is asymptomatic until a triggering event occurs, with sodium channel blockers, fever and alcohol being common risk factors for appearance of the Brugada pattern on EKG. COVID-19 diagnosis can also present a risk for Brugada presentation. COVID-19 is known to exacerbate other cardiac arrhythmias and channelopathies such as Long-QT syndrome, in addition to being known to cause fever and electrolyte imbalance. We present the case of a patient exhibiting the Brugada pattern on EKG with symptoms of chest tightness, shortness of breath, and tachycardia shortly after testing positive for COVID-19 in the Emergency Department. Due to the potential for unmasking the Brugada pattern as well as other cardiac arrhythmias during COVID-19 infection, patient and family history should be thoroughly considered to rule out potential genetic conditions when examining patients who test positive for SARS-CoV-2. This case is presented to highlight COVID-19 as a potential etiology of the Brugada pattern on EKG in genetically predisposed individuals.

Introduction

Brugada syndrome is an inherited arrhythmogenic disorder characterized by autosomal dominant mutations in several identified genes with about 50% of cases carrying loss of function mutations in the SCN5A and SCN10A genes which encode for cardiac voltage-gated sodium channels [1-4]. When loss of function occurs in these genes, there is a weakened sodium current in conjunction with an unchanged potassium current. This may lead to complications including ventricular tachycardia, ventricular fibrillation, and eventually progressing to sudden cardiac death in some cases [1]. The Brugada pattern manifests on an EKG as coved ST-elevation with an accentuated J-wave [5]. The pattern can precede episodes of ventricular tachycardia and/or ventricular fibrillation.

The mutations described above predispose individuals to sudden cardiac death, though patients are typically asymptomatic prior to an event that ‘unmasks’ their condition. Risk factors for symptomatic events include fever, alcohol use, sodium channel blockers, and inflammatory responses [1,6,7]. Additionally, there is some evidence COVID-19 is a risk factor for Brugada unmasking in genetically predisposed individuals. COVID-19 is a known risk factor for other cardiac arrhythmias and channelopathies and can trigger additional cardiovascular pathologies such as Long QT syndrome [4-8,12]. COVID-19 symptoms also include several confounding risk factors for Brugada presentation including fever, diarrhea and associated electrolyte imbalance. Fever has been shown to decrease cardiac sodium channel capacity, exacerbating existing deficits seen in patients with sodium channel mutations associated with Brugada [8,13]. As a result, there is significant concern for patients diagnosed with COVID-19 who have a genetic predisposition to Brugada syndrome. We present the case of a patient exhibiting the Brugada pattern on EKG with symptoms of chest tightness, shortness of breath, and tachycardia shortly after testing positive for COVID-19 in the Emergency Department (ED). This case is presented to offer insight into the emerging risk for COVID-19 associated Brugada presentation.

Case Presentation

Case Report

A 61-year-old man with a past medical history of type 2 diabetes, DVT, pulmonary embolism, and CVA presented to the ED after two days of worsening subjective fever, chest tightness and shortness of breath. The patient tested positive for COVID-19 on admission to the ED, reporting multiple instances of vomiting and diarrhea. The patient has a history of palpitations and presyncope, with episodes of light-headedness and dizziness occurring when he is working as a painter but more recently occurring at rest as well. He had no prior EKGs indicating potential cardiac arrhythmias or ectopic foci. The patient has a family history of cardiomyopathy (father), fatal myocardial infarction (mother), and unknown conditions requiring all of his siblings to have pacemaker or ICD implantation. Cardiac exam showed tachycardia (105-112 beats per minute) with a normal rhythm and no murmur or gallop. Peripheral pulses were present and capillary refill was normal.

Physical Examination

The patient was alert and oriented upon arrival, appeared well nourished and in no acute distress. Upon admission, the patient was afebrile (36.8°C), had a blood pressure of 107/58 and an oxygen saturation of 97%. Skin examination showed several healing abdominal wall incisions from a recent laparoscopic inguinal hernia repair. Cardiac exam showed tachycardia (105-112 beats per minute) with a normal rhythm and no murmur or gallop. Peripheral pulses were present and capillary refill was normal.
Laboratory results, Imaging, and EKG

Laboratory results are insignificant barring a troponin that is slightly elevated (Table 1).

<table>
<thead>
<tr>
<th>Laboratory test</th>
<th>Patient Values</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>6,700 cells/mm3</td>
<td>4,500-11,000 cells/mm3</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>16.5 g/dL</td>
<td>14-18 g/dL</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>50.10%</td>
<td>42-50%</td>
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<tr>
<td>MCHC</td>
<td>32.9 g/dL</td>
<td>33-37 g/dL</td>
</tr>
<tr>
<td>Sodium</td>
<td>135 mmol/L</td>
<td>136-142 mmol/L</td>
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<tr>
<td>Glucose</td>
<td>149 mg/dL</td>
<td>70-140 mg/dL</td>
</tr>
<tr>
<td>Monocytes</td>
<td>1.27x10^3/mcL</td>
<td>2-8x10^2/mcL</td>
</tr>
<tr>
<td>Troponin 1</td>
<td>&lt;0.015 ng/mL</td>
<td>0-0.04 ng/mL</td>
</tr>
</tbody>
</table>

Diagnosis and treatment

Based on the evidence of the clinical findings, past medical history, family medical history, and EKG results, the patient was diagnosed with COVID-19 infection preceding Type-1 Brugada pattern. The patient was given subcutaneous enoxaparin daily and received acetaminophen for COVID-19 symptoms. He was monitored via telemetry until COVID-19 symptoms availed. The patient was recommended for concurrent with COVID-19 diagnosis require extensive monitoring throughout hospital admission. In Brugada patients, the sodium gated ion channels function at a reduced efficiency and increased temperature further diminishes sodium channel capacity– exacerbating the condition [8,13]. As a result, reducing and controlling fever in COVID-19 patients with the Brugada syndrome or associated risk factors is vital to preventing complications of Brugada pattern on EKG. For patients diagnosed with COVID-19 inducing the Type 1 Brugada pattern on EKG, risk factor management is key– reducing fever, electrolyte imbalance, and avoiding medications that can exacerbate cardiac symptoms. We present this case to bring clinical awareness to the risk that COVID-19 has on unmasking the Brugada pattern on EKG in patients with significant past medical and/or family history.

Conclusion

In patients diagnosed with COVID-19 inducing the Type 1 Brugada pattern on EKG, risk factor management is key– reducing fever, electrolyte imbalance, and avoiding medications that can exacerbate cardiac symptoms. We present this case to bring clinical awareness to the risk that COVID-19 has on unmasking the Brugada pattern on EKG in patients with significant past medical and/or family history.

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References


