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CASE REPORT

Brugada Syndrome: When Strict Treatment of Febrile Episodes Really Matters

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Abstract

We present a case of Brugada syndrome (BrS) diagnosed in a 32-year-old male during a febrile episode. This syndrome has characteristic ECG findings and predisposes patients to ventricular tachyarrhythmias and sudden cardiac death. We would like to highlight the necessity of aggressively treating febrile episodes in patients with BrS. The degree of risk for malignant arrhythmias in asymptomatic patients diagnosed with BrS is not clear. However, the potential for malignant arrhythmia is still there and increases in the setting of febrile episodes.

Keywords: Brugada syndrome, Brugada pattern, Ventricular arrhythmia

1. Introduction

Type 1 Brugada pattern on ECG is defined by a coved-type ST-segment elevation greater than or equal to 2 mm in 1 or more of the right precordial leads (V1−V3) occurring either spontaneously or induced by sodium channel blocking agent.[1] Usually this is also associated with a right bundle branch block.[2] Furthermore, patients with Brugada syndrome are nearly always found to have a structurally normal heart.[1]

There is debate on when to diagnose Brugada Syndrome (BrS). However, the generalized consensus is that with the pattern already described, the diagnosis of BrS can be made.[1] The patient does not have to have a malignant arrhythmia such as ventricular fibrillation for the diagnosis of BrS.[1] Therefore, for simplification, we will discuss type 1 Brugada pattern on ECG as Brugada syndrome (BrS) in this case report.

The prevalence of BrS in the general, asymptomatic population is small, though there is some variation in presumed percentage. The prevalence ranges from 0.4% in Quan et al., to 0.64% in Mizusawa et al., to 0.8% in Probst et al.[2−4] That there is geographic variation has been established.[2,3] In Mizusawa et al., BrS can be seen in up to 0.36% of Asian population, up to 0.25% of European population, and 0.03% of United States population.[3]

Despite this seemingly small incidence of BrS, this syndrome accounts for a large portion of sudden cardiac death.[1] Approximately 20% of sudden cardiac death cases in patients with structurally normal hearts have been attributed to BrS.[1] Even in patients diagnosed with BrS without an initial arrhythmic event, there is an estimated 12% risk of malignant arrhythmia within 10 years of diagnosis.[1]

2. Case report

A 32-year-old male with a history of HIV AIDS, daily marijuana use, and asthma presented to the hospital for worsening shortness of breath. He had been short of breath for one week. He was working in the kitchen of a restaurant and noticed he was having more difficulty completing tasks he could normally do without issue. His breathing became progressively worse and because of that he presented to the hospital.

Medications: atovaquone 10 mL/1500 mg by mouth daily, diphenhydramine 25 mg capsule every 6 h as needed for itching, and Dolutegravir-Rilpivirine 50-25 mg tablet daily.

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On physical exam his maximum temperature was 102.4 F (39.1C), heart rate ranged from 98 to 116 beats per minute, respiratory rate ranged from 11 to 36 breaths per minute, and blood pressure was 102–136/60–83 mmHg. Otherwise, there was an unremarkable cardiopulmonary exam. No lower extremity edema.

His initial ECG revealed coved ST segment elevation >2 mm in V1 and V2 followed by a negative T wave (Fig. 1). Initial chest x-ray showed patchy bilateral opacification concerning for multifocal pneumonia.

The initial troponin was <0.03 ng/mL. COVID-19 testing was negative. CD 4 count was 14%.

He was started on Vancomycin and Cefepime for pneumonia. His fever overnight was treated with acetaminophen 650 mg every 6 h as needed. Vancomycin was discontinued with negative MRSA nares swab.

The second troponin was <0.03 ng/dL. His febrile episodes were treated with acetaminophen. He did continue to have some temperature elevations into the second day. The temperature taken before the second ECG was performed was 37.7C (99.8 F). The second ECG no longer showed the Brugada pattern (see Fig. 2). Several hours after the second ECG, he did have one episode where temperature was 39.1C (102.1 F) but then returned and stayed around 37.1C (98.7).

He had a transthoracic echocardiogram (TTE). This showed ejection fraction 61% with normal left ventricular size, thickness, and preserved systolic function. Mild tricuspid regurgitation with elevated estimated pulmonary pressure of 50 mmHg, otherwise structurally normal heart.

The patient had no history of lightheadedness, pre syncope, or syncopal episodes. His family history was rather unclear. He believed he had a brother who had a pacemaker or some sort of cardiac device inserted at 23 years old. His father had a pacemaker or some sort of cardiac device placed at 55 years old. His grandfather died "of a heart attack" at 48 years old.

He was asymptomatic without history of ventricular arrhythmia or syncopal episode. Therefore, decision was made to hold off on intervention. The goal was to treat fevers aggressively during hospitalization and in the future and avoid medications that could cause the pattern such as sodium channel blockers.

He was seen in the cardiology clinic several weeks after hospital discharge. He remained asymptomatic. Intervention was not performed. ECG at that time again showed no significant ST segment or T wave changes such as were seen on the ECG on initial presentation. Furthermore, repeat TTE ejection fraction 64% with no wall motion abnormalities. At that time, trace tricuspid regurgitation and now with an estimated right systolic pressure of 23 mmHg.

3. Discussion

Fever is known to unmask BrS.[1,5] One study performed by Adler et al. found the incidence of type 1

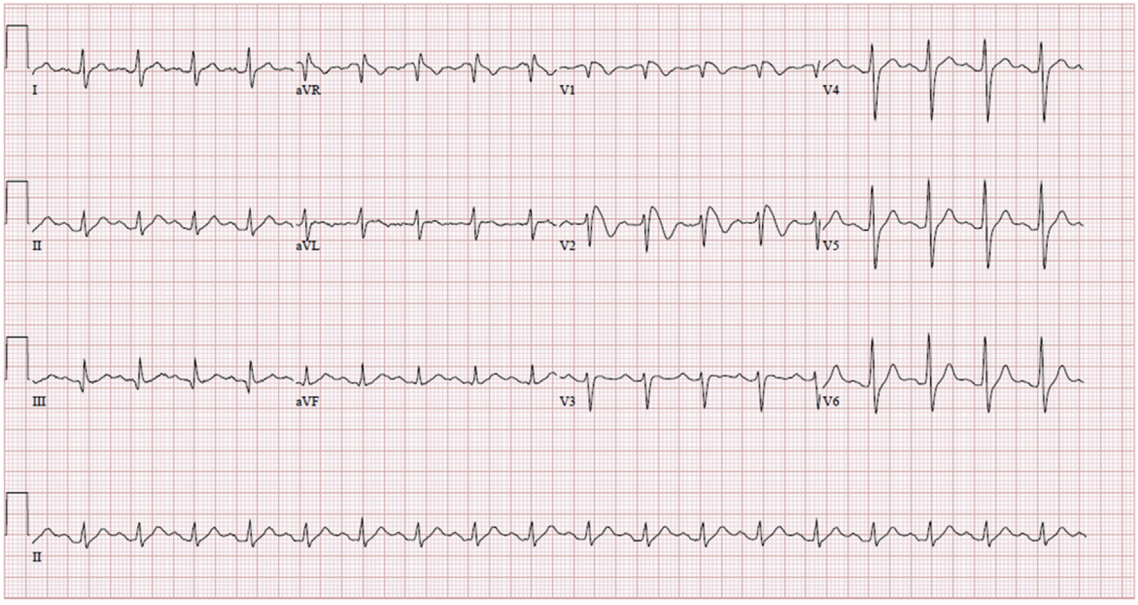

Fig. 1. ECG with coved ST segment elevation >2 mm in V1 and V2 followed by a negative T wave.

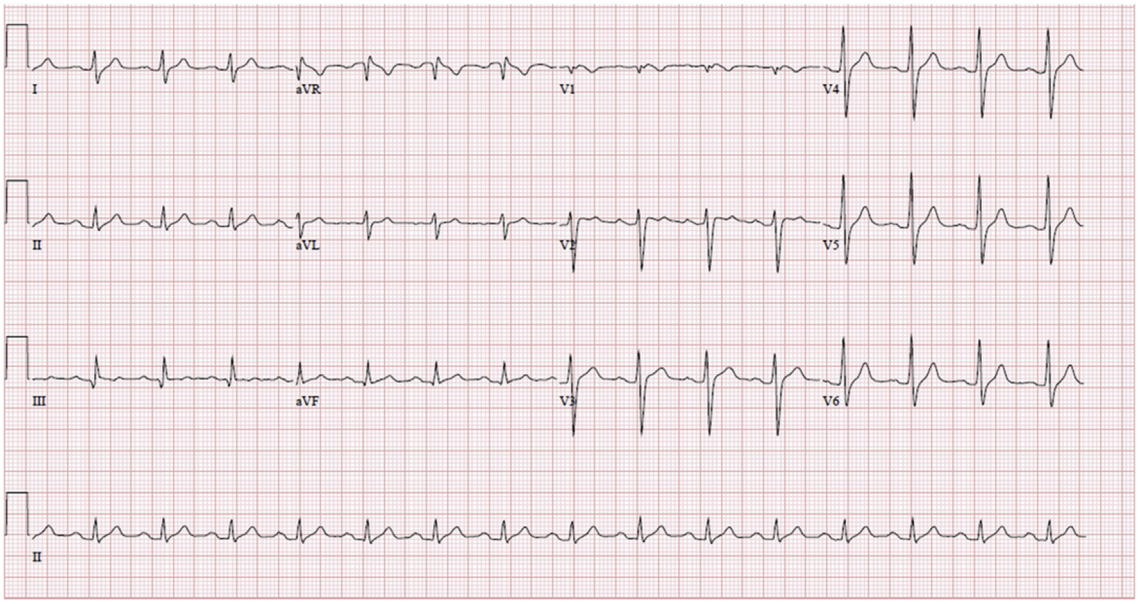

Fig. 2. ECG no longer with the Brugada pattern.

Brugada pattern is 20 times higher in asymptomatic febrile versus afebrile patients. The study raises the concern that the prevalence of BrS in the general public could be as high as 2%, which is higher than previously estimated.[5] If fever is a great revealer of BrS in an asymptomatic public, then it stands to reason that without fever, many patients would not have the diagnostic ECG pattern, and would not be diagnosed with BrS. Therefore, there could potentially be a larger community of undiagnosed BrS.[5]

Most patients with BrS will be men. In the study by Adler et al., the patients with BrS were 87% male and the ages ranged from 31 to 57 years old.[5] In a European registry looking at BrS patients, the characteristics of the patients were very similar.[4] In an asymptomatic subgroup, 69% were male and the median age was again 45 years old.[4]

Our patient was newly diagnosed with BrS after an ECG taken while he was febrile fit the diagnostic criteria for BrS. He does fit the young, male demographic, though he is not of Asian descent.

There is an increased risk for malignant arrhythmias such as ventricular fibrillation in BrS patients who have febrile episodes.[1,3] Multiple studies have looked at patients who had fever and malignant arrhythmia on initial presentation. In Amin et al., 22 of 111 patients who were diagnosed with BrS had cardiac arrest and 18% of those patients who had cardiac arrest had been febrile.[6] In Junttilla et al., 47 patients were found to have type 1 Brugada pattern on ECG. 18 developed sudden cardiac death. Out of those 18 patients, 6 had febrile episodes.[7]

Therefore, malignant arrhythmia is an ongoing concern for patients diagnosed with BrS.[6,7] For symptomatic BrS patients, ICD implantation is indicated.[1] However, for asymptomatic BrS patients, ICD is not necessarily indicated.[3] It is unclear in this subset of patients who are febrile, found to have BrS, but are otherwise asymptomatic, what risk they have of developing malignant arrhythmia in the future.[5]

Besides malignant arrhythmias, another question remaining is how often does Brugada pattern on ECG even occur in patients diagnosed with BrS. Richter et al. looked at 89 patients after diagnosis of BrS was made. Greater than 95% of the patients who initially presented with type 1 Brugada pattern on ECG had resolution of pattern on future ECG. They followed patients every 3–6 months. Overall, in these follow ups only every fourth ECG was again diagnostic of BrS. It can be inferred that an ECG again diagnostic for BrS may occur only every 2 years.[8]

For our patient, the Brugada pattern on ECG resolved once the febrile episodes dissipated. His hospital course was not complicated by malignant arrhythmia. He had outpatient follow up 2 months after initial presentation and ECG at this appointment again did not reveal the Brugada pattern.

4. Conclusion

The ultimate management of Brugada syndrome is still being debated. And the exact impact of the syndrome on those who remain asymptomatic is unknown. As more asymptomatic patients are diagnosed with BrS based on ECG findings, studies

should be done to look at long term arrhythmia risk. Discovering specific genetic markers especially in those patients who are or become symptomatic would also be of great utility. Both could help with future risk stratification and management. For now, what is definitive and well documented is that febrile episodes can precipitate BrS. The ultimate concern is that this can lead to potentially fatal arrhythmias. Therefore, in BrS patients, treating febrile episodes is imperative.

Conflict of interest

We have no conflicts of interest to disclose.

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