Brugada ST elevation masquerading as ST elevation myocardial infarction

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ABSTRACT

Brugada syndrome (BS) is a genetic cardiac syndrome first described in 1992 that is characterized by distinct abnormalities noted in leads V1 and V2 on electrocardiogram. It often leads to syncope or even sudden death in affected patients due to its propensity for ventricular tachycardia and fibrillation. The characteristic ST elevation seen in these patients can be confused with ischemic ST elevation. We present a case where a febrile illness unmasked BS and the ST changes seen were believed to be ischemic.

KEYWORDS Brugada syndrome; electrocardiogram; ST elevated myocardial infarction

Multiple potential triggers of arrhythmia and syncope have been described in Brugada syndrome (BS), including medications, electrolyte derangements, and autonomic changes. Fever is a less commonly cited triggering factor. Here we describe the initial diagnosis of BS in a man who presented with fevers and electrocardiographic changes.

CASE REPORT

A 47-year-old man with no known past medical history presented to a local emergency department with chest pain, coughing, and fever up to 104°F for 5 days. His father had died in his 50s of what was assumed to be a myocardial infarction. He did not smoke or use drugs and drank minimal alcohol.

Shortly before the onset of his illness, his school-aged son had been sick with strep throat. The patient's primary care provider had given him a course of amoxicillin prior to his emergency department admission, assuming that he had also developed strep throat. The patient did not improve on amoxicillin and after being switched to amoxicillin-clavulanate, his fevers did not subside.

With his symptoms not improving, the patient went to a local emergency room. An electrocardiogram showed ST changes that were concerning for an acute ST-elevation myocardial infarction (*Figure 1*). ST elevation was prominent in leads V1 and V2. He had chest pain, which was more consistent with chest wall pain from recent intractable cough, but given the patient's family history, electrocardiographic changes, and symptoms, he was transferred for a higher level of care and an urgent invasive cardiac workup. Coronary angiography demonstrated no significant coronary narrowing, and the electrocardiographic pattern was felt to be most consistent with a diagnosis of type I BS.

Echocardiography showed no structural or functional abnormalities. Acetaminophen and ibuprofen were scheduled to lower his ongoing fever. Tests were sent for strep throat and influenza, along with blood cultures. Augmentin was started to cover for potential strep throat. On hospital day 2, influenza B polymerase chain reaction testing returned positive and antibiotics were discontinued.

The patient was outside the window for Tamiflu, and his fever gradually subsided with supportive care. With resolution of his fever, his electrocardiogram evolved over the next several days, demonstrating a type II Brugada pattern

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Figure 1. Initial electrocardiogram.

Figure 2. Electrocardiogram evolution over time.

with saddleback changes noted in lead V2 (Figure 2). The patient was counseled on aggressive treatment of fevers in the future and avoidance of potential arrhythmia-inducing drugs. Electrophysiology was consulted, and the patient was deemed to be low risk for sudden death. No implantable defibrillator was indicated. After discharge, the patient underwent genetic testing, which did not reveal any known genetic abnormalities associated with BS.

DISCUSSION

This case illustrates how undiagnosed BS may be unmasked by fever in an adult patient—something that is

more commonly seen in the pediatric population. Our patient presented with an electrocardiogram consistent with a type I Brugada pattern in the setting of fever. A 2013 study found that fever-induced BS is significantly more likely to present as a type I electrocardiographic pattern than other triggers of Brugada signs and symptoms. This same study found that febrile patients are less likely to have findings of arrhythmia or history of syncope at presentation; rather, the discovery of the Brugada pattern on electrocardiogram is an incidental finding in most of these patients.¹

In general, BS develops in patients with a cardiac channel mutation that causes a transmural voltage gradient between

the epicardium and endocardium.² More than 70 mutations have been identified that result in BS; the causative abnormality is typically in a cardiac sodium channel, and about 20% of those identified are in the SCN5A channel.³ Early inactivation of the sodium channel has been demonstrated at higher temperatures; this is thought to be the reason for the accentuation of symptoms in febrile BS patients.⁴

Several studies have noted that BS is one of many diagnoses that can be misdiagnosed as ST elevation myocardial infarction (STEMI). In a 2008 study of 19 patients with various disorders misdiagnosed with STEMI, 1 of 19 actually carried a diagnosis of BS rather than acute coronary syndrome.⁵ The typical electrocardiographic pattern of type I Brugada is a down-sloping ST-segment elevation peaking at 2 mm with J wave amplitude in leads V1–V3 and a negative T wave in the same leads; right bundle branch or pseudo-right bundle branch block is typically seen in two or more of V1–V3.^{6,7} This should be contrasted with the typical electrocardiographic findings in a patient presenting with STEMI, in which ST segments tend to be convex upward, forming a dome, often exceeding the height of QRS, with widened or inverted T waves, reciprocal ST depression, and Q waves.⁷ As this case demonstrates, patients presenting with ST elevation that does not fit the classic pattern associated with STEMI should be evaluated for other causes of electrocardiographic changes.

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