

Case Report: A 40 year-old Male with Fever and Syncope

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Introduction

Class I Brugada Syndrome is a disorder characterized by heterogenous, inheritable cardiac ion channel defects [1]. It is believed to account for 4% of all incidences of Sudden Cardiac Death (SCD) and up to 20% of cases of SCD in which there is an absence of structural cardiac defects. Traditional findings on EKG are the classic "sail" appearance of the QRS complex and ST segments in the right pre-cordial leads, characterized by ST segment and J-point elevation without a distinct R-wave which down-slopes into an inverted T-wave [2,3]. Patients often do not experience a syncopal episode until the 4th decade of life. Syncope is many times precipitated by a stressor such metabolic derangement or fever [4]. The patient examined in this clinical case report showed a history of febrile illness along with syncope [5], with subsequent EKG revealing a classic presentation of this rare disease. Further work remains to elucidate the influence of high temperature and metabolic derangements on the development of symptomatic Brugada syndrome and how to best risk stratify these patients.

Case Report

Mr. A is a 40 year-old black male with no past medical history who presented to the emergency room with myalgias and fever of 104°F. He reported additional symptoms such as chills, diaphoresis, headache, malaise, non-productive cough, emesis, and diarrhea. He stated that while riding the public bus en route to the emergency room, he began to have palpitations and then lost consciousness. He awoke shortly after and had no evidence of sequelae from this episode prior to his arrival.

Mr. A reported a similar episode of syncope preceded by palpitations six months before this occurrence, but otherwise had no pertinent medical problems. He stated he awoke that morning with a feeling of nausea and vomited one time. He noticed his heart beating faster than usual and shortly after lost consciousness leaving his bathroom. He woke up on the floor of his living room with no lingering symptoms. This episode of syncope was not witnessed nor did he see a physician after this incident. His family history was remarkable for cerebrovascular accidents and hypertension in both parents, but he clearly stated that no family member had any known heart conditions



Figure 1: An electrocardiogram showing complete right bundle branch block with J-point elevation and steep, down-sloping ST elevations in V1-V2.

or history of sudden cardiac death. He drinks alcohol socially and has no history of illicit drug use. He had a 10 pack/year history of cigarette smoking, but had quit one month prior. He was not sexually active and was employed as a construction worker.

He arrived at the ER where he was found to be febrile at 103.9 F with otherwise normal vital signs. Cardiac exam was unremarkable, including a regular rate and rhythm, with no murmurs, rubs, or gallops. Basic lab work was negative. A chest X-ray performed showed no infiltrates or consolidations. A CT of the head revealed no evidence of acute intracranial pathology.

An electrocardiogram (Figure 1) showed incomplete right bundle branch block with J-point elevation and steep, down-sloping ST elevations in V1-V2. These findings were classically suggestive of Class I Brugada syndrome [2,3]. He was admitted to the hospital for telemetry monitoring and further evaluation of his heart condition as well as his fever.

During his hospital stay, Mr. A was treated with oseltamivir 75 mg PO daily, intravenous hydration, and acetaminophen for a suspected influenza infection. A 2D echo was performed and showed normal cardiac function. Extensive testing revealed no viral etiology of the illness. Blood and urine cultures showed no growth.

After two days, his clinical condition improved to baseline and he remained afebrile. Given his history of syncope, he was deemed to be at a high risk for sudden cardiac death and surgical fixation of an automatic implantable cardioverter defibrillator was indicated per

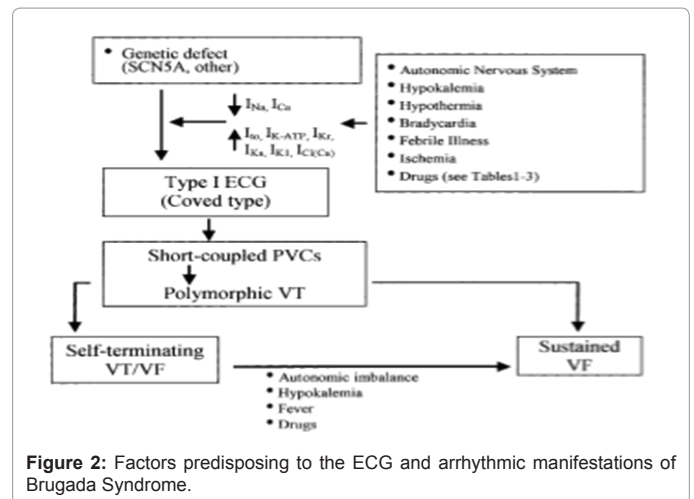


Figure 2: Factors predisposing to the ECG and arrhythmic manifestations of Brugada Syndrome.

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ACC/AHA guidelines (class I indication). The patient desired a second opinion on the placement of an AICD and was discharged from the hospital against medical advice with a LifeVest wearable defibrillator.

Conclusion

Class I Brugada syndrome is a disorder characterized by heterogenous inheritable cardiac ion channel defects first described in 1992 by Pedro and Josep Brugada. The worldwide prevalence is estimated to be 5/10,000 worldwide with a higher incidence (12/10,000) in Southeast Asia where it is endemic. Men are much more likely to be affected than women (4:1 ratio) with a mean diagnosis at 40-45 years of age. It is believed to account for 4% of all incidences of Sudden Cardiac Death (SCD) and up to 20% of cases of SCD in which there is an absence of structural cardiac defects [6].

The pathogenesis of Class I Brugada syndrome is dictated by the particular gene which is mutated. Currently, there are 10 different gene mutations which have been identified, most of which affect the cardiac sodium, calcium, and transient outflow currents. Overwhelmingly, the most common mutation is a loss of function mutation in the sodium channel encoded by the *SCN5A* gene. Traditional findings on EKG are the classic "sail" appearance of the QRS complex and ST segments in the right precordial leads which is characterized by ST segment and J-point elevation without a distinct R'-wave which downslopes into an inverted T-wave [2,3].

Patients usually do not experience their first syncopal episode until the 4th decade of life and is often precipitated by a stressor such as fever (Figure 2).

Indeed, several case reports have emerged demonstrating that febrile illness could unmask Brugada syndrome and precipitate ventricular arrhythmias [7]. Of note, northeastern Thailand, where Brugada syndrome is most prevalent, is known for its hot climate. Arrhythmias can also be induced by Class I anti-arrhythmic agents such as flecainide, and these drugs can be used to confirm diagnosis of Class I Brugada Syndrome.

Risk stratification of patients with Class I Brugada syndrome is typically quite challenging due to both the multiple genes responsible for this condition as well as the high incidence of SCD in this population. Previous episodes of syncope or SCD in patients are associated with

high recurrence rates for ventricular arrhythmias. It is recommended as a Class I indication that these patients, such as Mr. A, undergo AICD placement for future prevention of SCD. Previously asymptomatic patients are also at risk for SCD, with studies reporting 1-8% rate of fatal or near-fatal arrhythmia episodes at 30-50 months follow up. Family history has not been found to be a reliable indicator for risk stratification purposes in patients with Brugada Syndrome.

Multiple case reports have shown that a febrile state can lead to elevation of ST segments and unmask Brugada Syndrome. The possible underlying mechanisms involve the effects of fever on the function of mutant ion channels. Furthermore, the risk of ventricular arrhythmia in these patients seems to be increased during fever [8]. However, the effect of fever on the risk of life-threatening arrhythmias and sudden cardiac death appears to not have been studied systematically with large patient populations [9]. This is an area of study that can help elucidate both the pathogenesis and risk stratification for patients affected by Brugada Syndrome in the future.

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