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# A Classic Pattern of Type 1 Brugada Syndrome on ECG: A Case Report

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CASE REPORT

# A Classic Pattern of Type 1 Brugada Syndrome on ECG: A Case Report

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**Abstract**

**Brugada syndrome is an inherited disorder characterized by a channelopathy of cardiac sodium, potassium, and calcium channel. The pathophysiology of this disorder is not completely elucidated yet, however, most of the reported cases are caused by a pathogenic alteration in the SCN5A gene, leading to the malfunction of cardiac sodium channels. Several stressors are well known to unmask this pathology including fever and electrolytes imbalance.**

**Three ECG patterns are frequently described in the literature, type 1, type 2, and type 3. However, only the type 1 pattern is considered diagnostic of Brugada syndrome in the appropriate clinical context. Therapeutic strategies can range from conservative medical management with antiarrhythmic medications to Automatic Implantable Cardioverter Defibrillator (AICD) placement. Prompt recognition is of utmost importance since this pathology can rapidly evolve into life-threatening arrhythmias and sudden cardiac death.**

**Here we present a case of a 22-year-old male who presented after a syncopal episode and was found to have Brugada syndrome in the setting of Influenza A infection.**

*Keywords:* Brugada syndrome, Channelopathy, Automatic implantable cardioverter defibrillator, Sudden cardiac death

## 1. Introduction

Brugada syndrome is an inherited cardiac disorder often associated with mutations in the SCN5A gene which encodes for cardiac sodium channels. It has been found that this pathology is more prevalent in Southeast Asians with a strong male correlation with a 4:1 ratio as compared to females. There are three ECG patterns, type 1 is characterized by a coved-shaped ST-segment elevation along with a negative T-wave in the right precordial leads. Type 2 pattern is characterized by a saddleback morphology and ST-segment elevation. Type 3 pattern can be identified by a coved pattern or a saddleback along with an ST-segment elevation of less than 1 mm. This syndrome is estimated to be responsible for approximately 4% of sudden cardiac death in the general population, and around 20% of deaths among individuals with no known structural heart disease.[1] An implantable cardioverter defibrillator remains to be a safe therapeutic strategy and reduces cardiovascular mortality in patients with Brugada syndrome.[2,3]

Here we present a case of a young gentleman who presented with Brugada syndrome in the setting of Influenza A infection.

## 2. Case presentation

The patient is a 22-year-old male with a past medical history of schizophrenia who presented to the Emergency Department (ED) after an unwitnessed syncopal episode. The patient described the presence of prodromal symptoms characterized by flushing, palpitations, lightheadedness, and dizziness prior to the syncopal episode the morning of the presentation. He is not aware of the exact time duration of the episode, but he denied confusion or disorientation after the event. Afterward, the patient

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went to work when he started to experience headaches, myalgias, nasal congestion, sore throat, nausea, and 2–3 episodes of non-bloody, non-bilious emesis. He recalled a similar episode 6 years prior to the presentation.

He denied chest pain, shortness of breath, orthopnea, paroxysmal nocturnal dyspnea, tongue biting, abdominal pain, diarrhea, fever, or chills. He had a significant family history of unspecified heart conditions. He had a paternal uncle who died suddenly at the age of 16, and a sister who had pacemaker placement at 16 who is currently 24 years old and recently diagnosed with heart failure.

In the ED, the patient was febrile with a temperature of 100.7° Fahrenheit, blood pressure of 136/82 mmHg, heart rate of 98 beats per min, respiratory rate of 18 per min, and saturating at 98% on room air. On physical examination, the patient had a regular rate and rhythm, S1 and S2 were present without extra sounds, and no murmurs, rubs, or gallops were appreciated. Pertinent labs included mild hypokalemia of 3.4 [3.6–5.1 MMOL/L], troponin of <0.01 [0.5 ng/ml], and lactic acid 1.05 [0.5–2.2 mmol/L]. He tested positive for Influenza A. Chest X-ray was negative for any acute or chronic cardiopulmonary disease. ECG showed Brugada Type 1 pattern raising concern for Brugada Syndrome (Fig. 1).

The patient was admitted for further management with Tamiflu and Tylenol. Arrangements were made and the patient was transferred to Newark Beth Israel for an electrophysiology study and assessment for possible AICD placement.

## 3. Discussion

Brugada syndrome is an unusual cardiac disorder with an autosomal dominant pattern of inheritance associated with a mutation in multiple genes encoding for calcium, sodium, and potassium cardiac channels. Most of the reported cases are caused by a pathogenic alteration in the SCN5A gene, leading to the malfunction of cardiac sodium channels. However, other less frequent mutations of genes encoding for sodium channel subunits (SCN1B, SCN2B, SCN3B) have been reported, as well as, in genes associated with potassium channels (KCNE3, KCNJ8, KCND3, and KCNE5).[4]

Three patterns have been described in the literature that can be differentiated by EKG findings. Type 1 pattern is characterized by a coved-type ST-segment elevation (>2 mm) associated with a negative T wave in two or more right precordial leads (V1–V3). Type 2 pattern is recognized by the evidence of saddleback morphology followed by an ST-segment elevation of at least 1 mm along with a 2 mm J-point elevation.[4] Type 3 pattern can be identified by a coved pattern or a saddleback along with an ST-segment elevation of less than 1 mm.[3] However, only the type 1 pattern is diagnostic of Brugada syndrome while the presence of type 2 or 3 patterns is suggestive of this disorder.[1]

The ECG changes in this pathology may be triggered by a wide variety of stressors (electrolyte imbalances, fever, drug intoxication) or medications (anesthetics, sodium channel blockers, psychotropics). Similar to our case, Manohar et al., reported a case of an elderly lady who had a type 1 Brugada

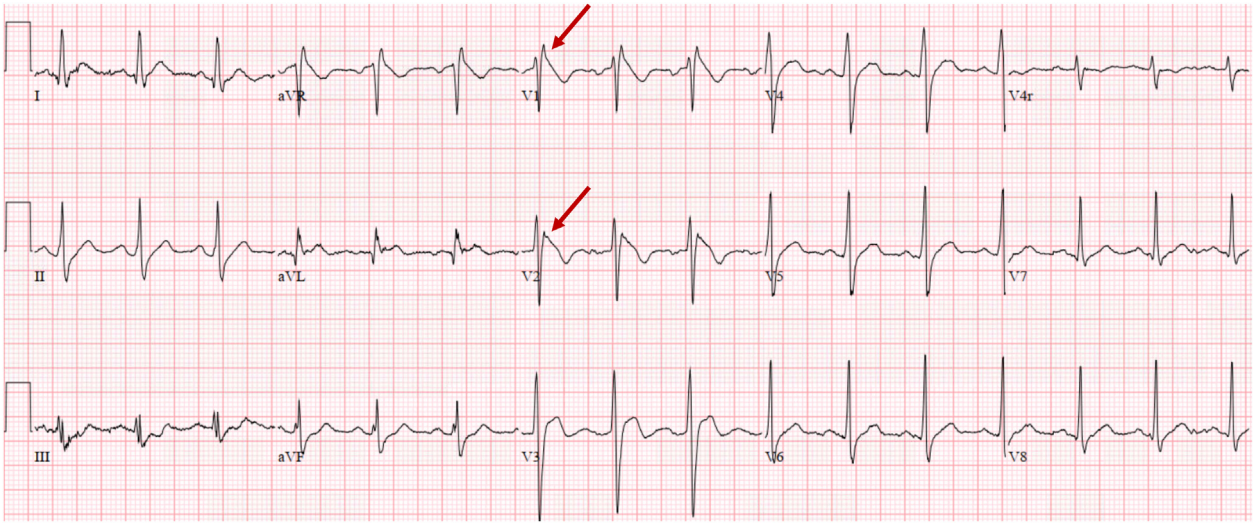


*Fig. 1. ECG showing coved ST-elevation in anterior leads.*

pattern on ECG after she was found febrile in the setting of sepsis secondary to urinary tract infection.[5] Furthermore, Adler et al., found that this ECG pattern was 20 times more likely to be evidenced in febrile vs afebrile patients; Amin et al., reported that fever was a relevant precipitating factor (18%) of the cardiac arrests in patients with Brugada syndrome.[6,7]

Currently, there are multiple therapeutic strategies for patients with Brugada syndrome from conservative management with antiarrhythmic medications (quinidine, bepridil) or phosphodiesterase III inhibitors (cilostazol) to more invasive management such as an AICD insertion. AICD placement has been proven to be a safe long-term therapeutic tool that reduces cardiovascular mortality in these patients, however, it is associated with complications including infections or lead failure.[2]

Clinicians should remain vigilant and have a low threshold for diagnosing this pathology since these patients are at increased risk for sudden cardiac death despite the absence of structural heart abnormality. Even though this syndrome has no known cure, the prevention of life-threatening arrhythmias is of utmost importance.

## Conflict of interest

There is no conflict of interest.

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