

Brugada ECG Sign & Chest Pain Mimicking ST Elevation Myocardial Infarction

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Abstract

Background: Management of patients with the brugada ECG sign who have no previous history of syncope is still negotiable. We present a case of a 57 year-old Caucasian lady who presented to the emergency department with substernal chest pain. **Results:** Her past medical history showed that she had two previous episodes of lightheadedness, but no syncope. She had a family history of sudden death secondary to unknown cause in her aunt at the age of 61. Physical exam was unremarkable except for diaphoresis. Electrocardiography (ECG) showed ST elevation in the right precordial leads (V1-V2) with T inversion, mimicking a STEMI. Emergent cardiac catheterization revealed normal coronary arteries. Echocardiogram was normal. Again, interpretation of ECG revealed a Brugada type 1 pattern, characterized by coved-type, gradually descending ST-T segment, elevated J point of more than 2 mm and T wave inversion. Electrophysiological (EPS) testing with a Sodium channel blocker challenge showed a persistent Brugada type 1 pattern with non-inducible ventricular tachycardia. This patient had Brugada type 1 ECG pattern with no previous history of syncope (asymptomatic). Thus she was considered at low risk of developing a serious arrhythmogenic event in the future. **Conclusion:** A history of syncope remains the best available predictor for arrhythmogenic events. EPS testing in such patients, to stratify the risk and predict for any future events, is still controversial. It is still unjustified to place an implantable cardioverter-defibrillator in asymptomatic non-inducible individuals with the Brugada pattern. These patients should follow up closely with a cardiologist and be aware of the risk of possible triggers of ventricular arrhythmias.

Key Words: Brugada syndrome, sudden cardiac death, myocardial infarction, sodium channels, cardiac electrophysiologic technique (Source: MeSH-NLM)

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Introduction

The Brugada pattern is characterized by ECG changes alone, without any of the clinical features of Brugada Syndrome. The ECG changes can be one of three types and can be dynamic. However, the patient should have the type 1 ECG pattern “coved-type” to make the diagnosis of Brugada. The following case report and discussion focuses on the presentation, diagnosis, management and prognosis of the asymptomatic patient with a Brugada ECG pattern.

The Case

A 57-year-old Caucasian lady presented to the emergency department (ED) with substernal chest pain of one day duration. It was associated with headache, backache and mild dyspnea. She reported 2 episodes of lightheadedness in the past month when she felt she was about to pass out. The episodes lasted for a few seconds and were relieved spontaneously. She denied syncope, dizziness, cough, palpitations, nausea or vomiting.

Her past medical history included hypertension, migraine, depression, attention deficit hyperactivity disorder, chronic sinusitis and asthma. Her home medications included lisinopril, gabapentin, trazodone, adderall, celebrex and flovent diskus. She has history of allergy to codeine sulfate. Her family history included hypertension and coronary artery disease, however it was significant for sudden death of unknown cause in her aunt at the age of 61. She also had a 48-pack-year history of smoking, but quit 7 years ago. She used to work as a hairstylist and consumed alcohol occasionally.

On physical examination, the patient was diaphoretic and in moderate distress in the ED. The ECG was interpreted by the on-call ER physician as a STEMI (Figure 1) and the cardiac catheterization team was activated. Coronary angiography revealed normal coronary arteries. Laboratory results revealed hypokalemia $K = 2.9$, and otherwise normal CBC, cardiac enzymes and other electrolytes. Transthoracic echocardiography showed a normal left ventricular ejection fraction and no structural heart disease.

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Figure 1. Electrocardiography of 57 years-old lady showing a Brugada type 1 pattern, characterized by the coved-type, gradually descending ST-T segment elevation, J wave and T inversion in leads V1-V2 and incomplete RBBB in the emergency department.

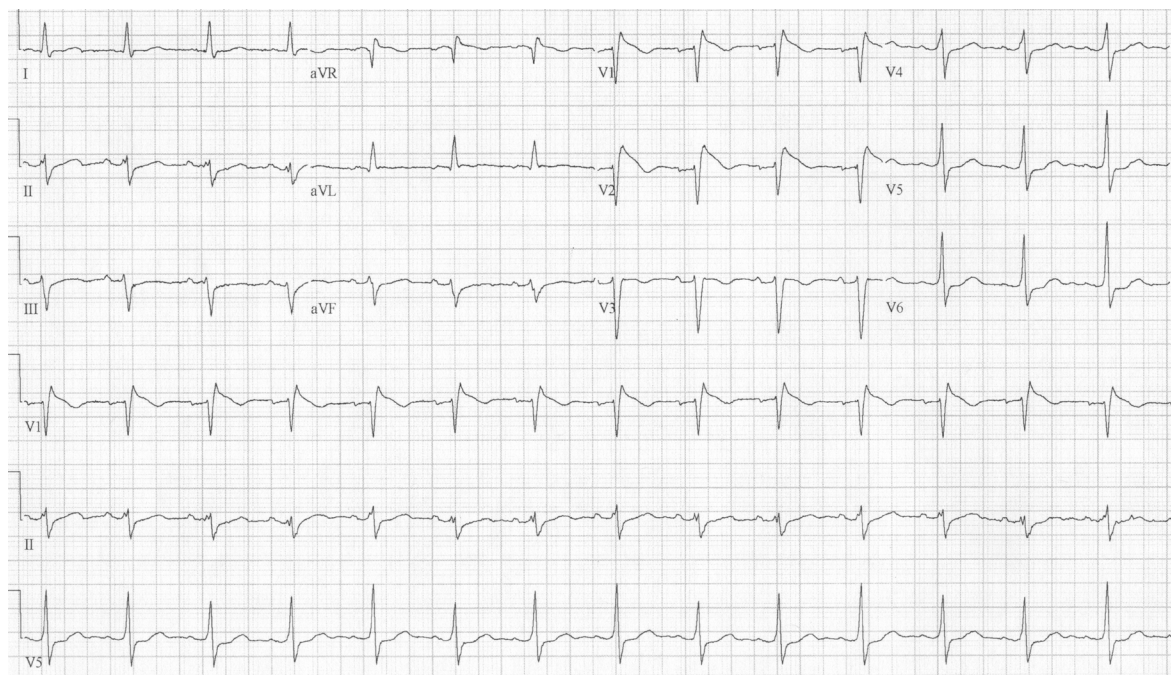
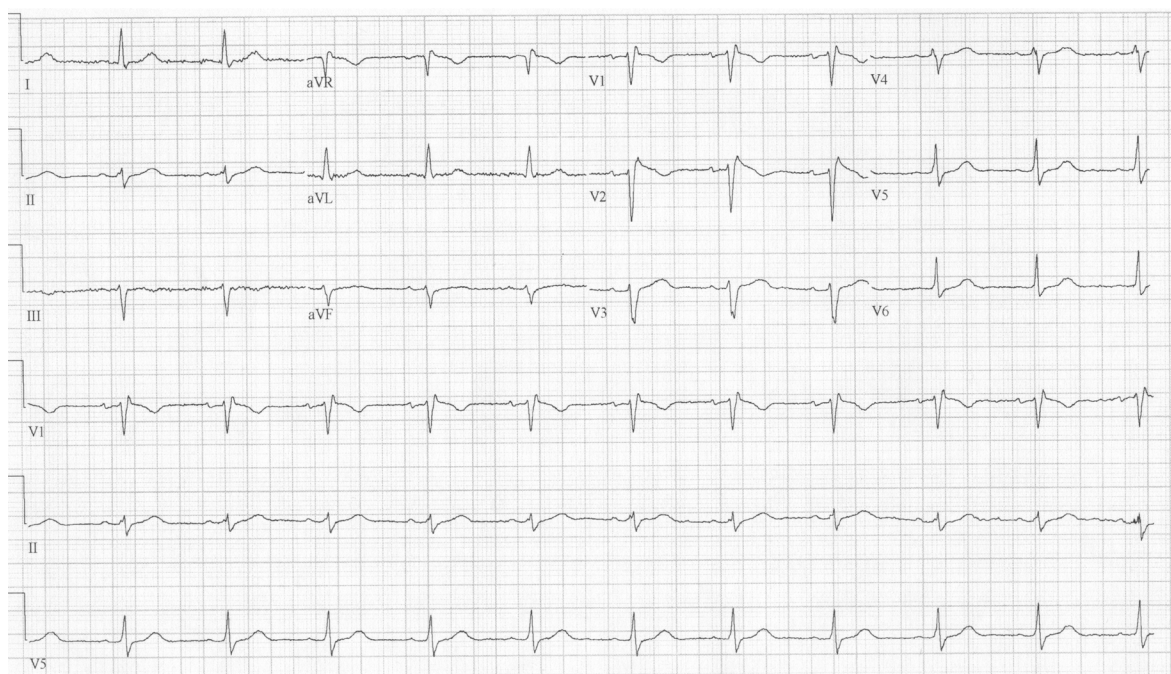


Figure 2. Electrocardiography of the same lady showed a persistent Brugada type 1 ECG pattern on the 5th day of admission. The patient was afebrile and recovering from a community acquired pneumonia on antibiotics.



The electrophysiology service was consulted and reevaluation of the ECG revealed coved-type J point and ST elevation in the right precordial leads (V1-V2) with incomplete right bundle branch block (RBBB), illustrating Brugada type 1 pattern. Cardiac MRI study showed no evidence of arrhythmogenic right ventricular dysplasia (ARVD). Genetic testing for SCN5A mutation wasn't performed.

The patient was transferred to telemetry for observation. Electrophysiological study (EPS) was planned for the next day due to a Brugada type 1 pattern on ECG with non-sustained ventricular tachycardia, when the patient was found to have fever (101.2 F), tachycardia, shortness of breath and cough.

Physical examination and chest X-ray showed left lower lobe infiltrate with left pleural effusion. Labs showed leukopenia and a positive urine test for the Streptococcus pneumonia antigen. She was diagnosed with community acquired pneumonia and antibiotics were started.

On the 6th day of admission, the patient was doing well and afebrile. A persistent Brugada type 1 ECG pattern is shown in (Figure 2). EPS was performed with provocative testing using procainamide. It revealed Brugada type 1 ECG pattern but no VT was induced with up to triple ventricular extra stimuli from right ventricular apex and base, on and off isuprel. The patient was asymptomatic and no implantable cardioverter defibrillator (ICD) was implanted. She was discharged home and scheduled to follow up closely with her cardiologist.

Discussion

Sudden cardiac death (SCD) in patients with normal hearts is an uncommon occurrence.¹ Brugada syndrome (BrS) was described in 1992 as one of the causes of SCD in patients with normal heart structure.^{2,3} This syndrome of high incidence of life-threatening ventricular arrhythmias is associated with ECG changes consistent with ST segment elevation in leads V₁ to V₃ and incomplete right bundle branch block.^{2,4-6} Due to the availability of a preventable mode of therapy, an implantable cardioverter-defibrillator (ICD) should be considered when definitive diagnosis of BrS is made.⁷

The Brugada pattern is characterized solely by the typical Brugada ECG changes, excluding the following clinical features: documented ventricular fibrillation (VF), self-terminating polymorphic ventricular tachycardia (VT), family history of SCD at the age <45 years, type 1 ST segment elevation in family members, electrophysiologic inducibility of VT, unexplained syncope suggestive of tachyarrhythmia or nocturnal agonal respiration. The Brugada ECG pattern has a coved-type, gradually descending ST-T segment, J wave amplitude of more than 2mm and T wave inversion.^{8,9} Characteristics of patients with the Brugada ECG pattern included higher prevalence in the Asian population.⁶ However, the exact prevalence varied among studies from different countries.^{13,14,18-21} It was mainly observed in adult males^{19,22} and a mutation in SCN5A gene on chromosome 3p21-24, that codes for the alpha-subunit of cardiac sodium channels, was found in about 18-30% of families with BrS.^{11,15,23,24}

The relation between the Brugada ECG sign in asymptomatic patients (no previous history of syncope) and the future risk of developing arrhythmogenic events has been investigated, especially over the past decade. One study revealed that the cardiac event rate per year in asymptomatic patients was 0.5%, compared to 1.9% in patients with a history of syncope and 7.7% in patients with aborted SCD.¹⁰ Further

reports of extended follow up of the asymptomatic population over a period of 3 to 7 years showed none to a very low arrhythmogenic event rate.^{4,9,11-15} Variations in the baseline characteristics of asymptomatic patients over a mean follow up of 2 to 3 years, revealed different rates of arrhythmias, with events arising in 8% of the asymptomatic population studied by Brugada et al,^{16,17} compared to 0.8% in a later study by Eckardt et al.¹⁷

Multiple predictors of future arrhythmic events in patients with the Brugada pattern were studied, with male gender, mutation of the SCN5A gene and a positive family history of SCD found to be non-predictive.^{10,16,30} The prognostic value of the clinical, ECG and EPS variables was analyzed in a population of spontaneous type 1 Brugada ECG patterns and no previous cardiac arrest. The cohort with negative EPS (non-inducibility of VT/VF) had a 1.8% risk of developing arrhythmic events (SCD or documented VF), compared to 14% in those with a positive EPS.³⁰ Further studies revealed a 0.9% rate of significant cardiac events in patients with non-inducible arrhythmias.^{16,31} A recent prospective multicenter study showed that a positive EPS is not predictive for arrhythmic events, and data reported that the spontaneous ECG pattern and the history of syncope are the best available predictors of such events.^{10,32} It also showed that QRS fragmentation and a ventricular effective refractory period of less than 200ms are risk indicators. These findings are imperative indicators for the need of an implantable cardioverter defibrillator in such populations.³² Additional literature review revealed the low significance of EPS in stratifying the risk of future arrhythmic events, and that its role remains an area of investigation and debate.^{9,11,15-17,26,33-39}

Currently, pharmacological therapy for the prevention of SCD in Brugada patients is not well established, however some reports showed that quinidine and hydroquinidine may be beneficial in such patients.⁴⁰⁻⁴³ The ICD has multiple complications, including the relatively high rate of inappropriate shocks (6-9% per year), thus it seems to be unjustified to give an ICD to the asymptomatic non-inducible individual with an abnormal ECG pattern.^{16,23,44-46} There is no definite evidence to suggest that individuals who have no personal history of syncope or any family history of SCD are at a higher risk of SCD than the general population.^{4,9,11-13,15}

In our case, genetic testing was deferred as only a small percentage of patients with the BrS do test positive for the SCN5A gene mutation,^{9,11} and recent reports showed that it will not guide the management of such cases.²⁵ We excluded other important causes of SCD, including ARVD, where the Brugada ECG pattern can be an early subclinical manifestation of this genetic disorder.²⁶ Moreover, our patient was afebrile on presentation and didn't have any of the known provoking factors that induce a Brugada-like

ECG pattern.^{27,28,29} There was no previous history of syncope along with non-inducible VT on provocative EPS testing. Lack of such possible predictive factors reflects a low risk of developing arrhythmic events and SCD.³⁰ Thus ICD therapy wasn't recommended in this lady as the weight of evidence didn't support it.⁷ To conclude, our patient was advised to follow up carefully with the cardiologist and to be aware of the risks of possible triggers of ventricular arrhythmias.

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