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A Case of STEMI Masquerading Brugada Syndrome: Emphasizing the Importance of Clinical Decision Making in Emergencies

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Abstract

Brugada syndrome is a rare arrhythmogenic syndrome that is associated with an increased risk of ventricular fibrillation and sudden cardiac death. Electrocardiographic findings include patterns similar to a right bundle branch block (RBBB) and persistent ST-segment elevation in precordial leads (V1 and V2). There are numerous reports of Brugada syndrome mimicking ST-segment elevation myocardial infraction (STEMI); however, we describe a case of 47-year-old male who presented with STEMI mimics Brugada syndrome with preexisting RBBB. The patient developed polymorphic ventricular tachycardia generating into ventricular fibrillation right before catheterization making the diagnosis more challenging. The patient, eventually, was found to have obstructive coronary artery disease and no evidence of abnormal sodium channelopathy on further testing. This case highlights the importance of meticulous history taking and appropriate diagnostic test in establishing proper diagnosis of STEMI in a patient with preexisting RBBB, which can mimic Brugada syndrome.

Keywords

Brugada, right bundle branch block, ST-elevation myocardial infraction, ventricular fibrillation

Introduction

Brugada syndrome is a rare arrhythmogenic syndrome that displays an autosomal dominant mode of inheritance with incomplete penetrance and is associated with an increased risk of ventricular fibrillation and sudden cardiac death (SCD).[1-3] The incidence of Brugada syndrome is estimated to be between 5 and 20 per 10 000 persons and is most common in middle-aged patients and has an 8-fold male predominance.[4,5] Although a number of genes have been studied, this syndrome has been mostly linked to a mutation in sodium voltage-gated channel α subunit 5 (SCN5A) gene that results in defective myocardial sodium channels.[6] Electrocardiographic (EKG) findings include patterns similar to a right bundle branch block (RBBB) and persistent ST-segment elevation in precordial leads (V1 and V2). There are numerous reports of Brugada syndrome mimicking ST-segment elevation myocardial infraction (STEMI); however, we describe a case in which STEMI mimics Brugada syndrome in a patient with preexisting RBBB.

Case

Our patient is a 47-year-old male with a past medical history of uncontrolled type 2 diabetes mellitus, who presented to the emergency department with complains of nausea and vomiting associated with dizziness. The patient denied chest pain, shortness of breath, cough, or diaphoresis. Physical examination including vitals were unremarkable. Laboratory tests on admission was significant for a troponin level of 2.03 ng/mL (normal value $<$ 0.5 ng/mL), serum glucose of

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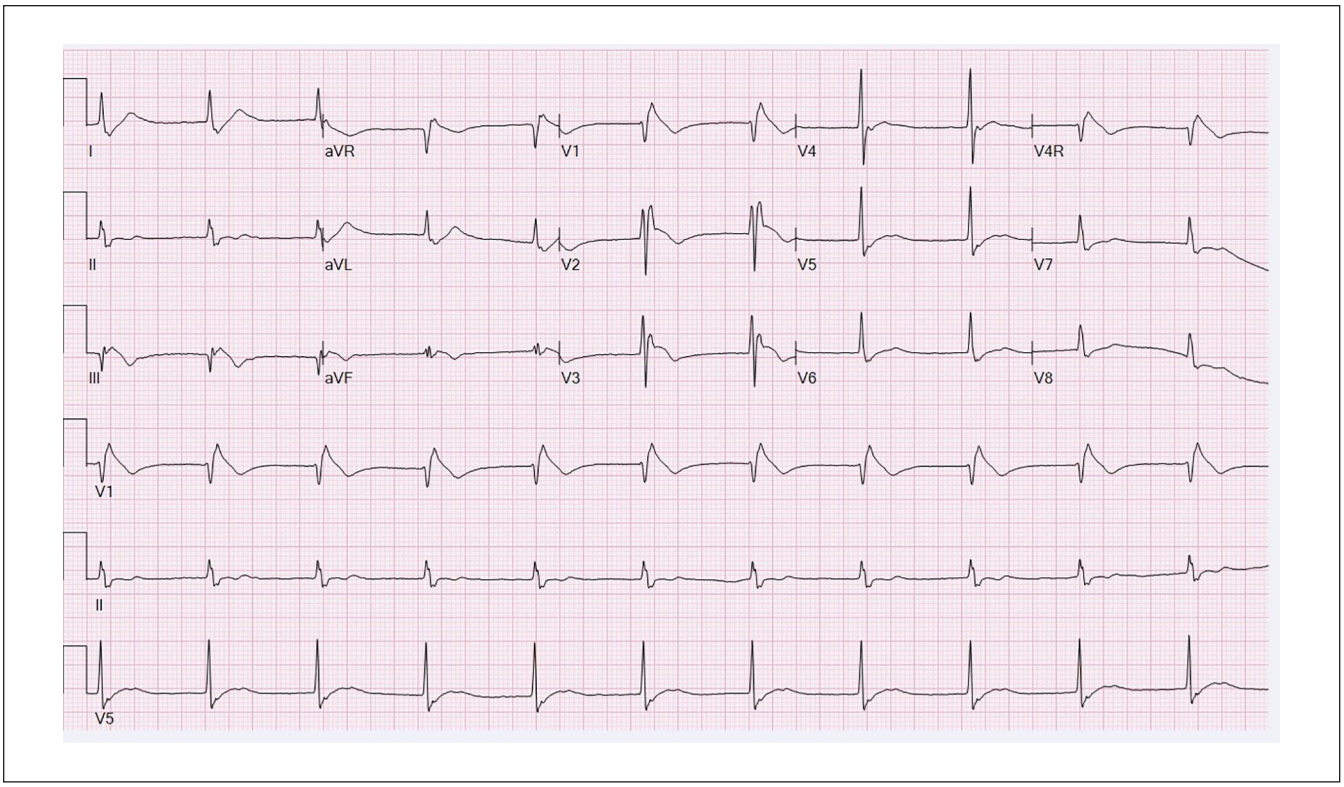

Figure 1. Electrocardiograph showing coved ST-segment elevation in lead V1 suggesting Brugada pattern, and ST-segment elevation in lead V2, V3 suggesting anterioseptal myocardial infraction.

493 mg/dL, an anion gap of 13, and sodium of 133 mEq/L. Other laboratory tests, including electrolytes, blood urea nitrogen, creatinine, and thyroid-stimulating hormone, were all within normal range. Additionally, urine toxicology was unremarkable. Initial EKG showed ST-segment elevation in V2-V4, concerning for anterior wall STEMI (Figure 1). The patient was then emergently taken to the cardiac catheterization laboratory. However, the patient developed polymorphic ventricular tachycardia generating into ventricular fibrillation right before cardiac catheterization and was quickly defibrillated. Cardiac catheterization revealed 80% stenosis in the proximal left anterior descending artery (LAD), 80% stenosis in distal LAD, and diffuse spasm of the LAD (Figures 2 and 3) that was treated with 2 drug-eluting stents: one in proximal LAD and the other in the distal LAD (Figure 4). Two-dimensional echocardiogram revealed left ventricular ejection fraction (LVEF) of 55% to 60% without obvious wall motion abnormalities. Patient sustained 2 further episodes of polymorphic ventricular tachycardia during cardiac catheterization requiring defibrillation and brief period of chest compressions.

Etiology of ventricular arrhythmia was unclear at this point. Angiographic findings were suggestive of ischemic etiology; however, a closer evaluation of the patient's EKG revealed a coved ST segment elevation suggestive of Brugada pattern. This EKG pattern, along with absence of regional wall motion abnormality, raised concern for possible sodium channelopathy. The gastrointestinal upset, low sodium levels, and metabolic abnormalities due to diabetic ketoacidosis may have precipitated Brugada syndrome causing ventricular fibrillation. The patient was evaluated for an implantable defibrillator

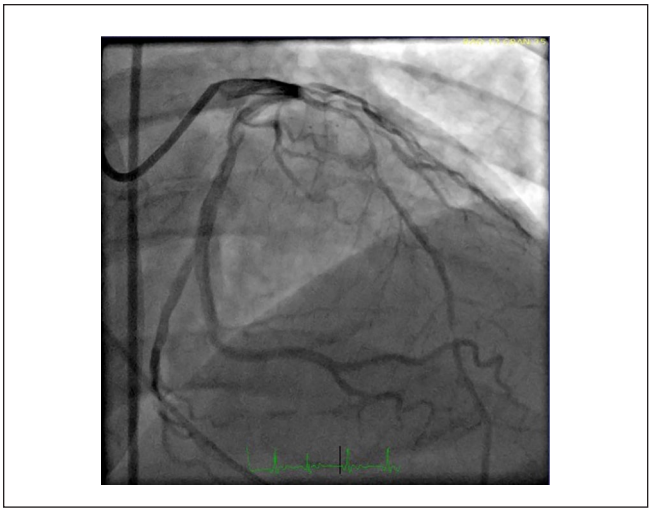

Figure 2. Cardiac catheterization showing 80% stenosis in proximal left anterior descending artery (LAD), 80% stenosis in distal LAD, and diffuse spasm of the LAD.

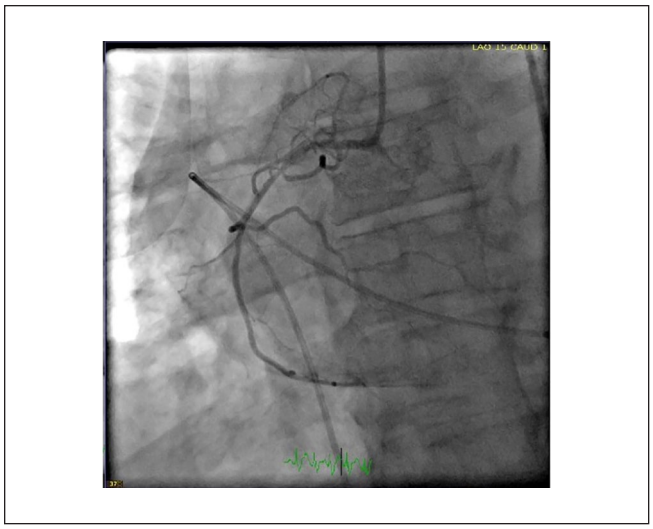

Figure 3. Cardiac catheterization showing nondominant small caliber nonobstructive right coronary artery.

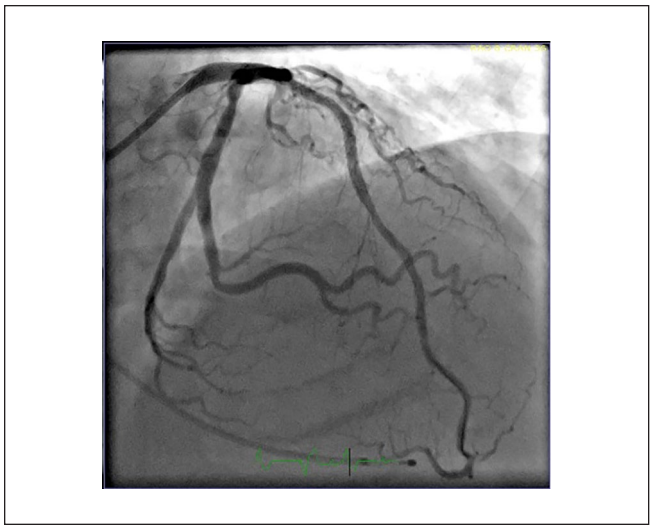

Figure 4. Cardiac catheterization showing 2 drug-eluting stents: one in proximal left anterior descending artery (LAD) and the other in the distal LAD showing resolution of stenosis and spasm.

for secondary prevention of SCD. He recovered clinically in few days and was discharged on LifeVest wearable defibrillator. Subsequent outpatient follow-up revealed that he did not receive any shock from the wearable defibrillator. Follow-up EKG showed disappearance of the Brugada pattern (Figure 5). The patient subsequently underwent electrophysiological study with procainamide challenge test that did not show any sodium channelopathy suggestive of Brugada syndrome.

Discussion

Brugada syndrome is a rare inherited arrhythmogenic syndrome in the absence of ischemia, electrolyte disturbances, or identifiable structural heart disease, and is associated with an increased risk of ventricular fibrillation and SCD.[1-3] Currently, it is believed that this syndrome is responsible for 12% of SCD and 20% of SCD in populations with no structural heart abnormality. The incidence of Brugada syndrome is estimated to be between 5 and 20 per 10 000 persons and is most common in middle-aged patients (40 years) and has an 8-fold male predominance.[4,5] More than 70 mutations have been identified, and of these, 20% to 30% of cases are due to a mutation in the sodium voltage-gated channel α subunit 5 (SCN5A) gene, which impedes function of myocardial sodium channels.[6] Multiple potential triggers have been described including febrile illness, hypothermia, alcohol, medication, electrolyte derangements such as hypokalemia, autonomic changes, and cocaine abuse.[7,8]

Patients often present with repeated episodes of syncope and in some cases, resuscitated SCD. Additionally, Brugada syndrome has a higher incidence of supraventricular tachyarrhythmias, including atrial and atrioventricular reentrant tachycardia and rarely monomorphic ventricular tachycardia. Precordial lead ST-segment elevations on electrocardiogram constitute the hallmark of Brugada syndrome due to the associated depolarization and repolarization abnormalities. Several studies have shown that Brugada syndrome is one of the many conditions that can be misdiagnosed as ST-elevation myocardial infarction.

Although the pathophysiology of ST-segment elevation in Brugada syndrome is not well understood, 2 potential mechanisms have been studied. The first hypothesis revolves around heterogeneous expression of transient outward potassium current between the epicardium and other transmural layers.[4,9] A strong potassium current in the epicardium in the presence of dysfunctional sodium channels renders the cardiac myocytes more susceptible to strong repolarizing forces causing the ST-segment elevation. The alternative explanation is based on regional differences in epicardial conduction velocity of the right ventricular outflow tract, which can be further aggravated by a reduction in functional sodium channels. However, neither mechanism has yet been conclusively confirmed. The typical EKG pattern of Brugada syndrome has been classified into 3 types. Type I is characterized by a prominent coved ST-segment elevation with a J-wave amplitude of $\geq$2 mm in leads V1-V3 followed by a negative T-wave. Type II is also characterized by a ST-segment elevation, however, gradually descending ST-segment remaining $\geq$1 mm above the baseline, followed by a positive or biphasic T-wave (saddle-back configuration). Type III is a right precordial ST-segment elevation of $<$1 mm of either saddleback type, coved-type, or both.

Given the high risk of SCD in patients with Brugada syndrome, timely diagnosis and management is crucial to prevent this adverse outcome. Diagnosis of Brugada syndrome revolves around the characteristic ST-segment elevations. However, the EKG can be silent, requiring sodium

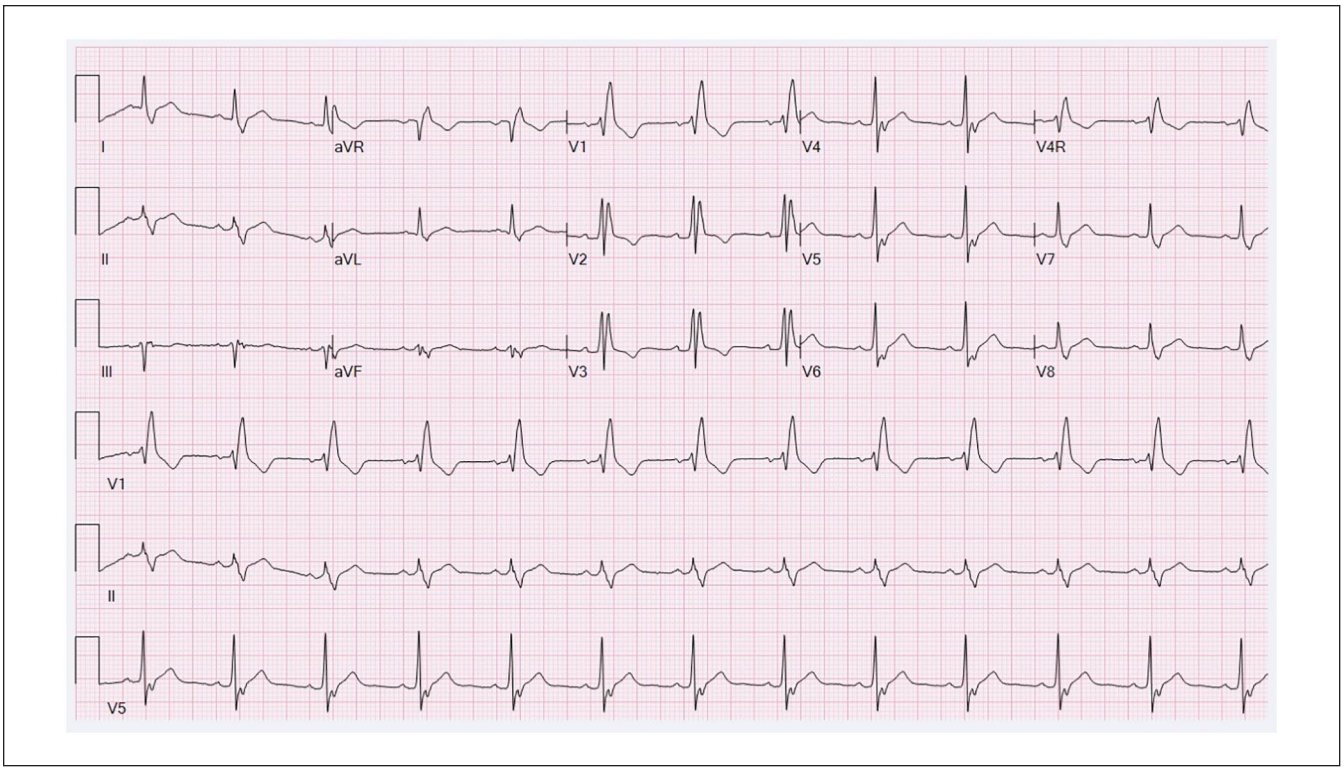

Figure 5. Follow-up electrocardiograph showed right bundle branch block and disappearance of the previously present Brugada pattern.

channel blockers such as flecainide and procainamide to unmask the pathology.[2] Furthermore, 20% to 30% of patients diagnosed with Brugada syndrome have a significant family history of SCD, and therefore can aid in diagnosing this condition.[1,7] Patients with suspected Brugada syndrome must be evaluated for implantable cardioverter defibrillator (ICD) placement as this is the only effective prevention of SCD.[4,8] Quinidine and phosphodiesterase 3 inhibitors among other pharmacological treatments are also being used to manage Brugada syndrome.[4] Furthermore, radiofrequency ablation of ventricular ectopy was postulated as a therapeutic approach in patients with Brugada syndrome. In support of this, Nademanee et al published the first study showing prevention of ventricular fibrillation in these patients by catheter ablation over the anterior right ventricular outflow tract.[10]

In our case, the patient initially presented with STEMI, complicated by cardiac arrest and ventricular fibrillation that was thought to be due to ischemia. He continued to have EKG abnormalities suggestive of Brugada pattern after successful coronary angioplasty with stents placed in culprit vessels. This led to further suspicion of patient having a presumed sodium channel defect that caused the syncope, polymorphic ventricular tachycardia, and cardiac arrest, which are associated with Brugada syndrome. This necessitated further evaluation for ICD with electrophysiological testing. This case demonstrates the dilemma in the management of conditions presenting like Brugada syndrome and shows the importance of electrophysiological studies to confirm the diagnosis.

In conclusion, STEMI in patient with preexisting RBBB can mimic Brugada syndrome and should be suspected especially in the setting of ventricular fibrillation. One should be extra vigilant in monitoring these patients. Meticulous history taking and appropriate diagnostic test is essential to help establish proper diagnosis and guide appropriate therapy.

Declaration of Conflicting Interests

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Ethics Approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed Consent

Written informed consent was obtained from the patient for the anonymized information to be published in this article.

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