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Rare Case of Spontaneous Type I Brugada Syndrome Secondary to Drug Overdose

Walid Omer*, Nagat Mudabal

Hospital medicine department, Advent Redmond hospital
*Corresponding author: Walid_Omer@teamhealth.com

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Abstract The Brugada syndrome is an autosomal dominant genetic disorder with variable expression characterized by abnormal findings on the surface electrocardiogram (ECG) in conjunction with an increased risk of ventricular tachyarrhythmias and sudden cardiac death . Typically, the ECG findings consist of a pseudo-right bundle branch block and persistent ST segment elevation in leads V1 to V2. We present a rare case of spontaneous Type I Brugada secondary to drug overdose with cocaine and benzodiazepines in a young male patient who eventually received **automated implantable cardioverter defibrillator** (AICD).

Keywords: *Type I Brugada, spontaneous Brugada, Drug overdose*

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1. Introduction

The Brugada Syndrome (BrS) is a heterogeneous genetic disease characterized by persistent or transient ST-segment elevation in the right precordial ECG leads and a high incidence of sudden death and lifethreatening ventricular tachyarrhythmias in patients with structurally normal hearts [1]. It is estimated to cause 4–12% of all sudden cardiac deaths [2]. Persons with either the Brugada pattern or the Brugada syndrome can have identical findings on the surface electrocardiography; the electrocardiography will have one of two distinct patterns of ST elevation described below [3]:

Type 1 Brugada electrocardiography **pattern** – The elevated ST segment (≥2 mm) descends with an upward convexity to an inverted T wave. This is referred to as the "coved type" Brugada pattern (Figure A).

Type 2 Brugada electrocardiography **pattern** – The ST segment has a "saddle back" ST-T wave configuration, in which the elevated ST segment descends toward the baseline and then rises again to an upright or biphasic T wave (Figure B).

The syndrome is inherited as an autosomal dominant trait. Since it was linked to mutations in the SCN5A gene that encodes the α subunit of the cardiac sodium channel protein, more than 80 mutations have been linked to the syndrome in the SCN5A gene .2 Although the genetic mutation is equally distributed between the sexes, the clinical phenotype is 8–10 times more prevalent in males than in females [4]. We report a case of Brugada syndrome whose electrocardiography pattern during electrolytes abnormalities converted to normal rhythm after electrolytes correction.

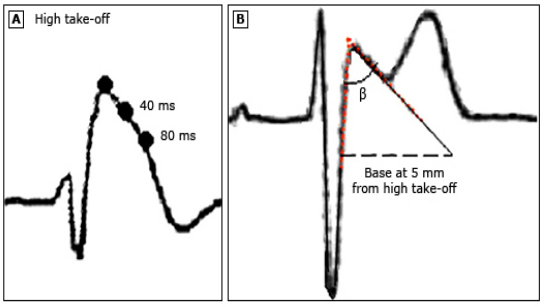

Figure 1. A Type 1 Brugada (coved type) B.Type II Brugada (saddle back)

2. Case Report

This case report describes about a young, male patient with past medical history significant for polysubstance abuse, seizure disorder, and alcohol abuse who was admitted to the intensive care unit for a respiratory failure, drug overdose (cocaine and benzodiazepine) and found to have pneumonia, acute renal failure, and rhabdomyolysis. A few hours after the admission the patient have developed transient electrocardiography changes suggestive of Brugada syndrome. The patient was found unresponsive at home and was brought to the electrocardiography where he was intubated because of respiratory failure; after intubation and he was transferred to the intensive care department. The patient was found to have Urine drug screen positive for Cocaine and Benzodiazepine for which he was given Narcan. On presentation, Pt had Hyperkalemia at 8.3, elevated phosphorus at 14, elevated Mg at 2.8, elevated total CPK at 902, and elevated Troponin at 0.7. The Pt's renal function was indicated acute pre-renal failure with Cr of 3.1 (baseline 0.8). Upon his presentation, part of his

initial work up, he had electrocardiography checked on 3/21,3/22 and 4/2. The second electrocardiography on 3/21 showed ST elevation in V1, V2, pathognomonic for Brugada syndrome [Figure 2]. The findings initially thought to be from his cocaine intake were not seen on other electrocardiography. Upon further investigation, patient admits that he has no history of angina, Recurrent syncope or presyncope, however, he states that he had very few occasions when he passed out after having drank a lot of alcohol. His history of seizures is unclear as he states that he was put on a Seizure medication but cannot recall the name. (Unclear if the Seizures are related to alcohol intake or were syncopal episodes taken as seizures). Patient denies any family history of heart disease or syncope. One documented coved ST elevation in a patient with multiple co morbidities including polysubstance abuse, and electrolytes abnormalities. Transthoracic echocardiogram showed reduced EF at 35-45% and mild aortic stenosis; Left heart catheterization was negative for any coronaries blockages and **automated implantable cardioverter defibrillator** (AICD) has placed by cardiology team**.**

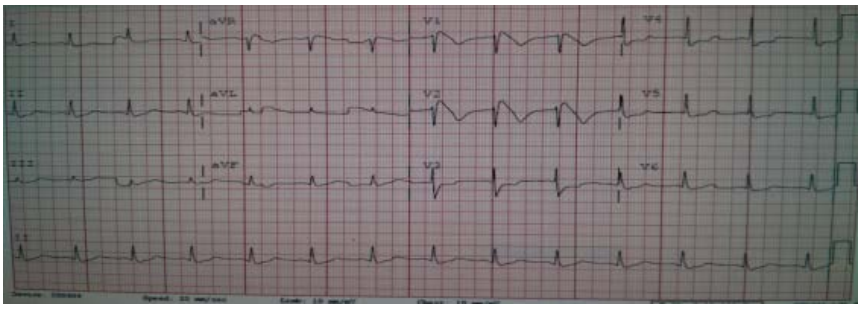

Figure 2. ST elevation in V1 and V2

3. Discussion

Brugada syndrome can be easily diagnosed by electrocardiography. The ST segment elevations in V1-V3 with the characteristic right bundle branch block pattern are seen. The morphology of the ST segment and right bundle branch patterns are different based on the genetic morphology. Clinical presentations of the Brugada syndrome are related to life-threatening ventricular arrhythmias. Males outnumber females with the syndrome [4]. Sudden cardiac death or syncope is often the first clinical event. The syndrome should be suspected in patients with either documented idiopathic ventricular fibrillation, self-terminating polymorphic ventricular tachycardia, a family history of sudden cardiac death in patients less than 45 years of age and/or syncope with the characteristic electrocardiographic change [5,6]. The appearance of the electrocardiographic changes alone represents an idiopathic Brugada electrocardiography pattern and not the Brugada syndrome. The Brugada mutation predisposes individuals to a lifetime risk of sudden cardiac death. There is no effective pharmacologic treatment that has been demonstrated to reduce the risk of sudden death. Both amiodarone and beta blockers are inferior to ICD. Genetic counseling is needed for patients with Brugada syndrome. First-degree relatives with the genetic mutation who are asymptomatic but carry the Brugada type electrocardiogram are still at risk for developing sudden cardiac death. Brugada syndrome can cause sudden cardiac death in structurally normal hearts. This rare genetically predisposed disease should be considered in all patients with the presentation of ST segment elevations in V1-V3 along with a right bundle branch block with symptoms suggestive of ventricular tachyarrhythmias [7].

4. Conclusion

We report Brugada syndrome in an atypical clinical context of a young man with drug overdose (cocaine and benzo) on top on acute renal failure, pneumonia, and rhabdomyolysis. As this is a rare case of Brugada syndrome underage of 30 without any risk factors, we present this case to raise clinician's awareness of Brugada syndrome secondary to drug overdose with cocaine and benzodiazepine and younger patients and promote work-up for these patients with characteristic electrocardiographic changes.

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