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A Perfect Storm: A Case of Transient Brugada Pattern

Gin Den William Chang*, Christabel Nyange, Sana Ahmad, Prateek Baghel, Elizabeth Brown, Sandrine Lebrun, Nicholas Skipitaris

Northwell Lenox Hill Hospital
*Corresponding author: wchang7@northwell.edu

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Abstract Brugada syndrome is a known electrophysiological phenomenon associated with high risk of sudden cardiac death. However, there are a number of published case reports showing a Brugada pattern in patients due to an underlying agent or cause. This case provides the nearly ideal clinical setup for a patient presenting with electrocardiographic findings of Brugada due to multiple factors requiring treatment of the underlying causes without need for further long term interventions.

Keywords: *Brugada syndrome, transient Brugada*

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1. Introduction

Brugada syndrome is an inherited autosomal dominant arrhythmogenic disorder involving the cardiac sodium gene SCN5A associated with sudden cardiac death with an electrocardiographic presence of a right bundle branch block with a coved ST segment elevation or saddleback pattern in the right precordial leads. Affected myocardial sodium channels reduce sodium inflow currents which disrupt normal action potentials. In some studies, the ST segment elevation and T wave inversions in the right precordial leads are hypothesized as a result of altered epicardial cells located in the right ventricular outflow tract, causing a transmural gradient of action potentials and subsequent Brugada electrophysiological findings in the right precordial leads [1]. There are two common types of Brugada: Type 1 is described as x≥2mm down sloping, "coved" ST elevation and Type 2 is a x≥2mm "saddleback" shaped ST elevation, both with a right bundle branch block morphology in the respective leads of V1 - V3.

Patients with a Brugada pattern characterized by symptoms such as sudden cardiac death or sustained ventricular tachycardia are diagnosed clinically as having Brugada syndrome - in most cases requiring an implantable cardioverter defibrillator. However, in some cases transient Brugada pattern can be observed in drug overdoses, fever, infection and polysubstance abuse without need of drastic intervention other than treating the underlying cause. We present a case in which a patient was initially admitted for a suspected ST elevation myocardial infarction but observed with a reversible Brugada pattern in the setting of cocaine, alcohol, fever, heroin and sepsis.

2. Case Presentation

A 53 year-old male with daily cocaine and intermittent heroin abuse presented with generalized malaise, fever and intermittent sharp chest pain without arm radiation for one day. He reported cocaine, excessive alcohol and heroin two days prior to admission. He reportedly drank multiple beers along with snorting heroin and cocaine. However, he denied any previous history of syncope or any family history of sudden cardiac death. On admission, vital signs were significant for a rectal temperature of 101 degrees Fahrenheit, heart rate 110, but blood pressure and oxygen saturation were within normal limits. Labs were notable for a white blood cell count of 12,000, and troponin T, CK-MB and CK were all within normal limits. Chest X-ray revealed a right middle lobe infiltrate. Electrocardiogram (ECG) revealed a coved-type ST elevation ≥ 2 mm (Type 1 Brugada pattern) in V1-V2 along with inferior ST depressions in leads II, III, AvF (Figure 1).

He was admitted to the Cardiac Care Unit for suspected acute coronary syndrome and sepsis secondary to community acquired pneumonia. He was treated with acetaminophen, ceftriaxone and azithromycin for pneumonia and empirically started on aspirin, ticagrelor, atorvastatin and a heparin drip for initial concern for acute coronary syndrome. Repeated cardiac enzymes were negative on two subsequent occasions. ST depressions normalized during his second hospitalization day and his Type 1 Brugada pattern improved during the next few days of his hospitalizations once his fever and chest pain subsided (Figure 2).

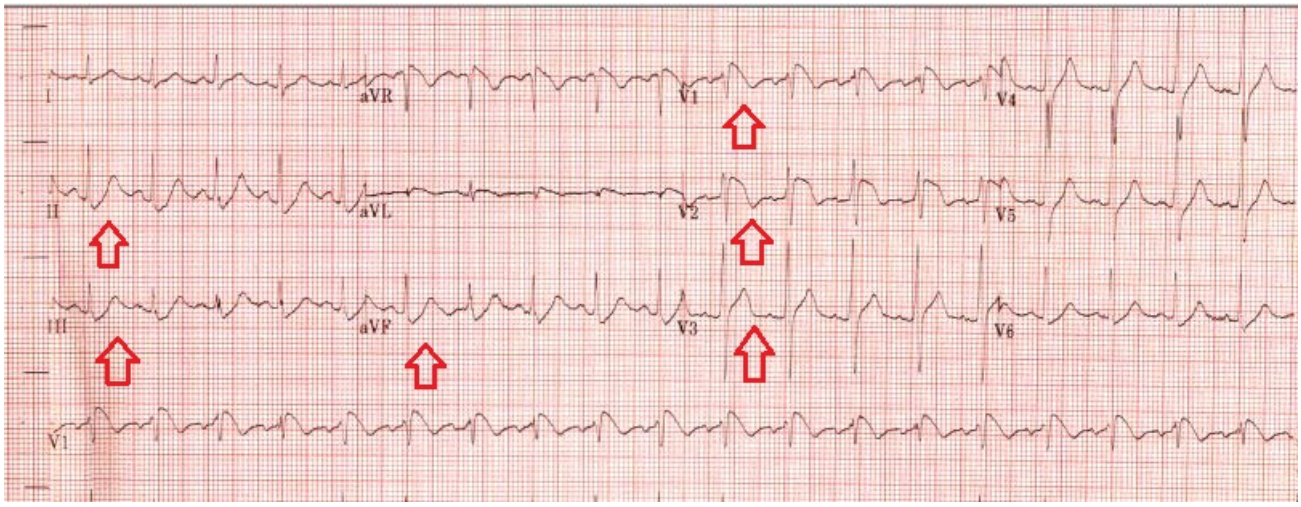

Figure 1. Electrocardiogram on day of admission revealed ST depressions in II, III, AvF, 2mm ST elevation in V3 and Brugada pattern in V1-V2 with characteristic right bundle pattern with coved ST elevations (red arrows)

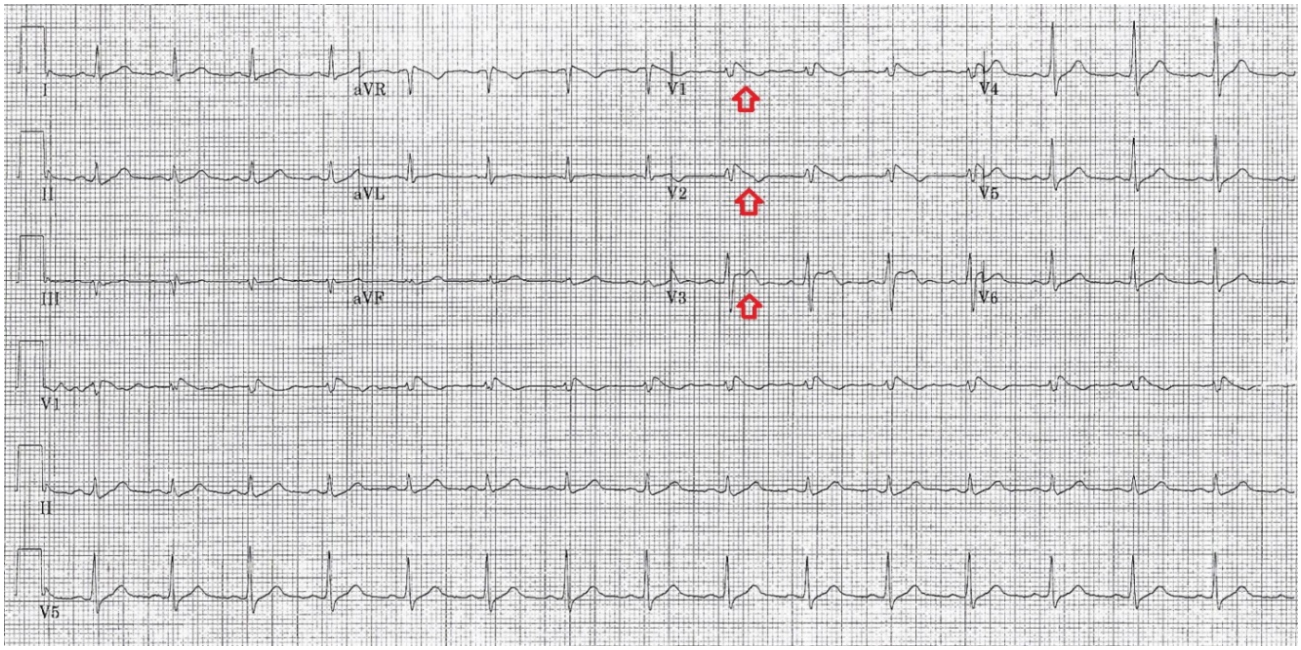

Figure 2. Hospital Day two showed normalization of ST depressions in II, III and AvF as well as improvement in the Brugada pattern seen in leads V1-V2 and ST elevation in V3

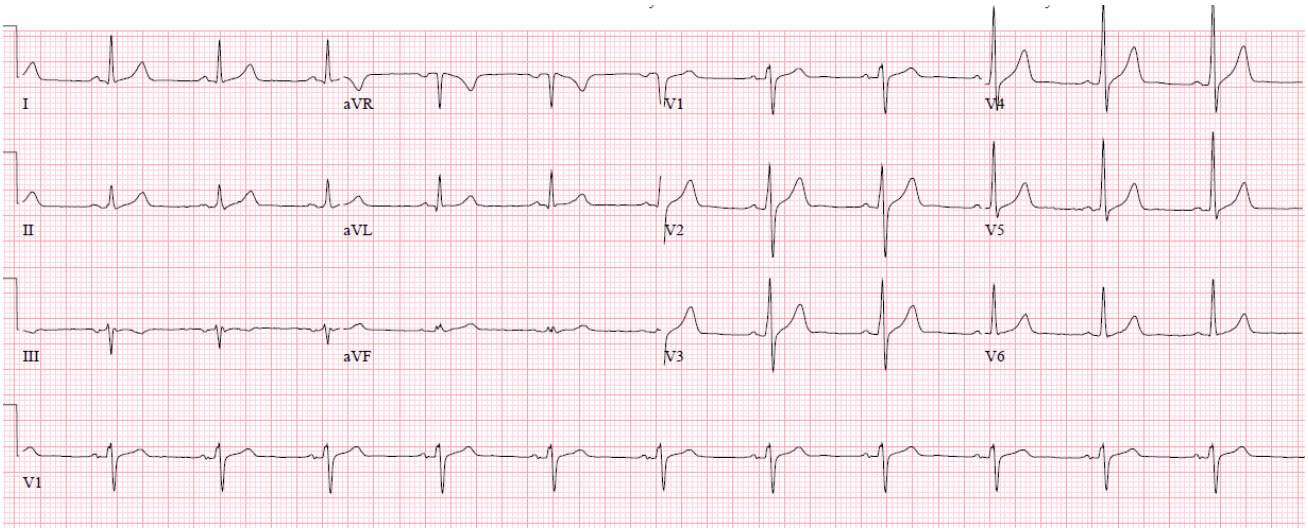

Figure 3. Follow up electrocardiogram on day of discharge revealed resolution of Brugada pattern

Echocardiogram showed no wall motion or valvular abnormalities with a normal left ventricular ejection fraction. Electrophysiology was consulted for concern for Brugada syndrome; however, no intervention was deemed necessary due to lack of prior history of syncope, and daily improvement of electrocardiographic findings. The patient refused coronary CT angiogram for ischemic evaluation but agreed to a non-contrast CT chest, which showed multifocal pneumonia and calculated calcium score of zero. On the day of discharge, the patient had no further symptoms of chest pain, remained afebrile and ECG showed normal sinus rhythm with full resolution of both Type 1 Brugada pattern in leads V1-V2 and ST elevation in V3 (Figure 3). Ultimately his Brugada was concluded to be multifactorial in the setting of fever and substance use, and his chest pain was attributed to vasospasm secondary to recent cocaine use. He was discharged with outpatient cardiology follow up and prescribed oral azithromycin and cefdinir to finish a course of five days of antibiotics for pneumonia.

3. Discussion

Fever is a known trigger for a transient Brugada pattern [2,3]. Hyperthermia is thought to cause a mutational change in temperature dependent sodium channels increasing the occurrence of arrhythmogenicity [4,5]. Additionally, medications have been well documented as a cause of reversible Brugada pattern. An international consortium, https://www.brugadadrugs.org/, provides an up to date list database for providers with medications causing transient Brugada such as Class I antiarrhythmic sodium channel blockers such as flecainide, lidocaine and procainamide [6]. Psychotropic medications such as lithium, oxcarbazepine, selective serotonin reuptake inhibitors, and cyclic antidepressants are also known to inhibit sodium channels. [7,8]. Intraoperatively certain anesthetics such as bupivacaine and high dose propofol infusions have also been documented to induce Brugada [9,10,11]. In addition, cocaine, alcohol and cannabis have been postulated to act like a Class I antiarrhythmic [12,13,14,15,16]. Another proposed mechanism for alcohol induced Brugada pattern is its activating properties on the parasympathetic nervous system triggering ventricular fibrillation and inhibition of sodium cardiac channels. There have been reports that vagal stimulation is a cause of sudden cardiac death in those diagnosed with Brugada syndrome [17,18]. Heroin and its opioid derivatives are likely mechanistically thought to behave as a sodium channel blocker as well, predisposing individuals to Brugada syndrome [19]. In all these cases, observation is the cornerstone of management due to the low risk of sudden cardiac death [20].

4. Conclusion

This case illustrates the importance of recognizing Brugada as part of the differential for ST elevation on ECG, in addition to other associated causes which may contribute to a transient Brugada pattern. In this particular case, cocaine, alcohol, heroin, fever and sepsis provided the perfect clinical scenario for an induced non-inherited Brugada electrocardiogram finding. Although Brugada syndrome increases the risk of sudden cardiac death, those with a transient Brugada pattern pose less of a risk for concern but still warrant cardiology follow up. Furthermore, these patients should be educated on the precipitating factors which may increase their relative risk of developing this electrophysiological phenomenon.

Ethics Approval and Consent to Participate

Not applicable.

Consent for Publication

Consent obtained at time of discharge.

Disclosure Statement

The authors have no conflicts of interest to declare.

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