The Journal of Emergency Medicine, Vol. ■, No. ■, pp. 1–4, 2016

0736-4679/$ - see front matter

http://dx.doi.org/10.1016/j.jemermed.2016.09.022

Clinical Communications: Adult

MOTOR VEHICLE COLLISION SECONDARY TO VENTRICULAR DYSRHYTHMIA: A CASE REPORT OF BRUGADA SYNDROME

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☐ **Abstract—Background: Brugada syndrome is a genetic disorder that increases an individual's risk for sudden cardiac death and ventricular dysrhythmias that was first described by the Brugada brothers in 1992. Brugada syndrome is characterized by an atypical electrocardiogram pattern that includes a bundle branch block and ST-segment elevation in the precordial leads. Case Report: A 74-year-old man had a cardiac arrest at the time of a low-speed motor vehicle collision. When emergency medical services arrived, the patient was in torsades de pointes. After resuscitation and return of spontaneous circulation, the patient was transferred to a Level I trauma center. He was ultimately diagnosed with Brugada syndrome after exclusion of traumatic injuries. Why Should an Emergency Physician Be Aware of This?: Brugada syndrome is still considered a "cannot miss" diagnosis in the emergency department, whether a patient presents with or without symptoms. In the mixed setting of trauma as a result of cardiac arrest, accurate diagnosis can be difficult due to the "chicken or the egg" dilemma.** © 2016 Elsevier Inc. All rights reserved.

☐ **Keywords—cardiac dysrhythmia; heart arrest; Brugada syndrome**

INTRODUCTION

Brugada syndrome (BS) is a relatively rare genetic disorder that increases an individual's risk for sudden cardiac death and ventricular dysrhythmias. The syndrome is characterized by an atypical electrocardiogram (ECG) pattern that includes a bundle branch block and ST-segment elevation in the precordial leads. Although this syndrome is rarely seen in the emergency department (ED), it remains an important differential consideration in otherwise healthy individuals who experience sudden cardiac death or syncope. We report the case of a 74-year-old man who remained asymptomatic throughout life until having an episode of torsades de pointes while driving, leading to a low-speed motor vehicle collision. The patient was subsequently found to have BS.

CASE REPORT

A 74-year-old man presented to a Level I trauma center ED as a transfer from a small local community hospital after a low-speed motor vehicle collision. The report from emergency medical services (EMS) personnel stated that the patient had initially been driving very slowly behind another vehicle. When the other vehicle stopped, our patient's vehicle collided with the stopped vehicle. Bystanders noted the patient to be slumped over and unresponsive in his vehicle, at which time EMS was called.

Upon arrival, EMS found the patient to be unresponsive, pulseless, with their initial ECG demonstrating torsades de pointes. Cardiopulmonary resuscitation was initiated. Paramedics administered epinephrine, magnesium, and

Received: 8 September 2016;
Accepted: 12 September 2016

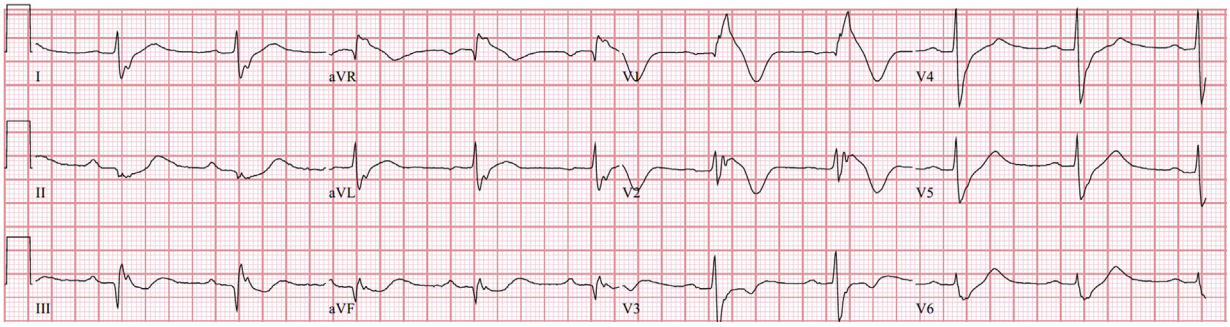

Figure 1. Electrocardiogram obtained after motor vehicle collision with widened QRS and coved ST elevations in leads V1 and V2.

amiodarone. The patient experienced return of spontaneous circulation and was transported to a local community hospital. However, due to the community hospital's computed tomography scanner being nonfunctional at that time, they stabilized the patient and transported him to our Level I trauma center to rule out traumatic injury and for higher level of care.

Initial ECG in the ED showed a prolonged QT interval, widened QRS, and coved ST elevations in leads V1 and V2 (Figure 1). Review of previous medical records revealed a prior ECG from 2008 that also demonstrated coved ST segments in leads V1 and V2, consistent with BS (Figure 2). Our trauma evaluation, consisting of a whole-body computed tomography scan, did not reveal any evidence of injury. With the findings on ECG, a cardiac event as the underlying cause of the motor vehicle collision was presumed.

Cardiology was consulted, and after their evaluation the patient was promptly transferred to the cardiac catheterization laboratory. Catheterization did not show any evidence of coronary artery disease, but did demonstrate evidence of possible Takotsubo cardiomyopathy.

The patient did not experience any recurrent episodes of ventricular dysrhythmia during the hospital admission. He recovered well and subsequently had an internal cardiac defibrillator placed. A thorough history failed to reveal either a previous history of syncope or sudden cardiac death, nor any family history of sudden cardiac death or cardiac dysrhythmias.

DISCUSSION

Originally described in 1992 by the Brugada brothers, BS is a genetically inherited channelopathy and is estimated to be the cause of up to 12% of sudden cardiac death cases, especially in those with structurally normal hearts (1,2). Currently, estimates put the prevalence of the disease between 1 and 5 persons per 10,000. BS is far more common in men than women, with more than 80% of cases in Western countries and > 90% of cases in Southeast Asian countries being men (3). The peak incidence for first cardiac event is 41 years old ± 15 years, with cases being reported from infants to 84-year-old patients (4).

There have been over 35 genetic loci associated with BS, with the most common mutation found in about 20–30% of patients. This common mutation leads to accelerated inactivation of the sodium channel (5). Although the sodium channel is the classic channel that malfunctions, both the cardiac potassium and calcium channels have also been implicated (6). In addition, recent literature has also pointed toward the right ventricular outflow tract as a possible physical source of the dysrhythmias in BS patients (7).

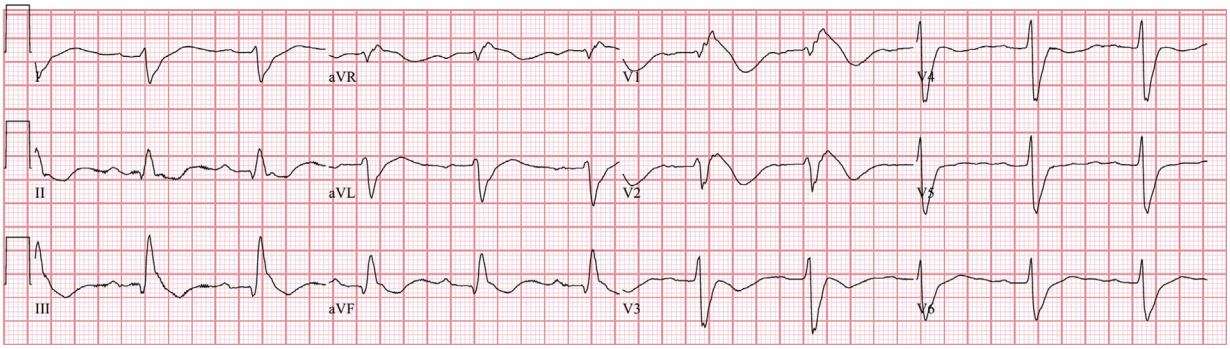

Figure 2. Prior electrocardiogram from 2008 that also demonstrated coved ST segments in leads V1 and V2.