Indian Journal of Medical Case Reports ISSN: 2319–3832
Online, International Journal, Available at http://www.cibtech.org/jcr.htm
2021 Vol.10, pp. 13-16/Gayatri and Thabit
Case Report (Open Access)

A CASE REPORT ON ATYPICAL PRESENTATION OF BRUGADA SYNDROME

***Gayatri Suresh K P and Thabit Ahmed**
Unit of Cardiology, Bangalore Baptist Hospital, Bellary Road, Hebbal, Bangalore-560024
**Author for Correspondence: gayatrisuresh123@gmail.com*

ABSTRACT
A , 50 year old patient presented with the complaints of retrosternal chest pain with acute shortness of breath.ECG done showed ST elevation in inferior and RV leads, and was intubated. Post intubation, patient had multiple episodes of VT which was brought under control with defibrillation and amiodarone infusion. In view of ACS, the patient was taken for primary PCI. CAG was done and showed dimunitive RCA with 100% occlusion and hence stenting was not done and started on IV tirofiban infusion, patient was shifted to ICU. There was recovery with medical management and improvement in the LV systolic function and the patient was discharged in a stable state. Patient was on regular follow-ups.
Then the patient came with complaints of retrosternal chest discomfort with dizzy spells. ECG was done and showed coved ST elevation in V1-V3 with negative T waves which was diagnostic of Brugada syndrome. Similar ECG patterns were observed in case of his daughter and sister also. Therefore, with the diagnostic ECG changes and clinical criteria satisfied, the patients diagnosis was conformed as Brugada syndrome and then the patient was taken for AICD-automatic implantable cardioverter defibrillator insertion , the patient was stable and subsequently discharged.

***Keywords*:** *Brugada Syndrome, Electrocardiogram, Electro Cardiac Pattern, Ventricular Arrhythmias, Cardioversion, Percutaneous Coronary Intervention*

Abbreviations
ECG- Electro cardiogram, ACS- Acute coronary syndrome, PCI- Percutanoeus coronary intervention, RCA- Right coronary artery, CAG- Coronary angiogram, ICU- Intensive care unit, AICD- Automatic implantable cardioverter defibrillator, LV- Left ventricular

INTRODUCTION
The Brugada syndrome is an autosomal dominant disease that may cause syncope and sudden cardiac death in individuals (predominantly young) with a normal heart. Brugada syndrome characterized by an electrocardiographic pattern of complete or incomplete right bundle branch block and ST segment elevation in leads V1–V3.(Kurita T et.al,2002) The clinical presentation combines the typical electrocardiogram (ECG) findings of the Brugada ECG pattern with a presentation suggesting ventricular arrhythmias including, syncope, palpitations, nocturnal agonal respiration, sudden unexpected nocturnal death.

CASE
History of presentation
A 56 year old male patient, recently diagnosed with Type II Diabetes mellitus, presented to the emergency with the complaints of retrosternal chest discomfort radiating to the left arm associated with dizziness and excessive sweating for 2 hours prior to arrival. On general physical examination, the abdomen was soft, no abnormalities with CVS/RS and the vitals were RR-20/min, PR- 100/min, BP-

120/80 mm Hg, Temp- 98.4 F, SpO2-95% room air. ECG done showed ST elevation in inferior and RV leads.

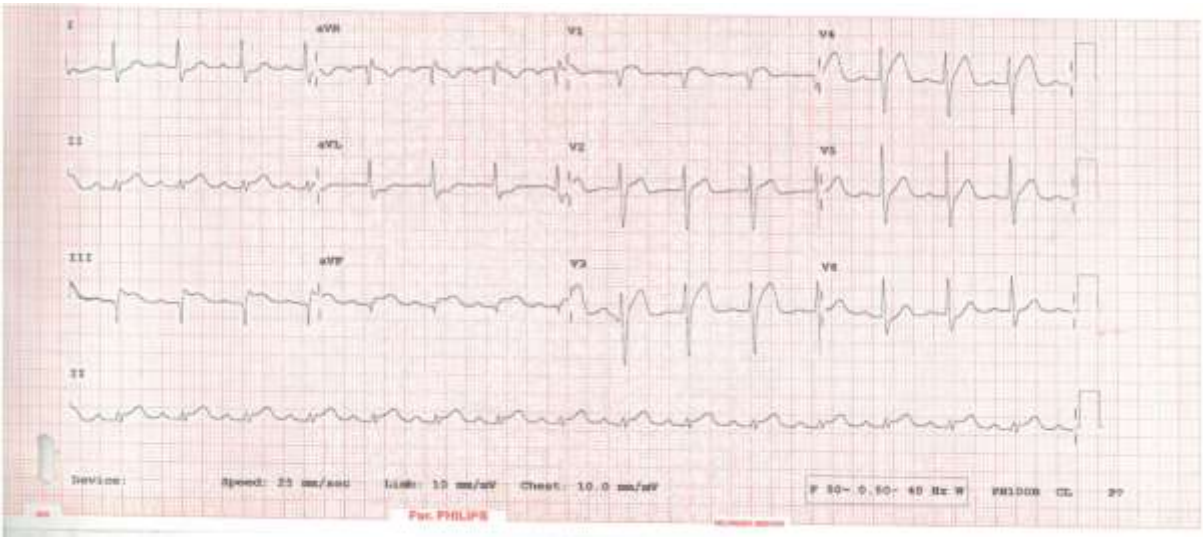

Figure 1: ECG changes presented initially

The patient was diagnosed with ACS – STEMI – IWMI, loading doses of dual antiplatelets, statins and initial anticoagulation dose were given and emergency cardiology consult was given. Activation of primary PCI pathway was initiated and the rhythm strip showed,

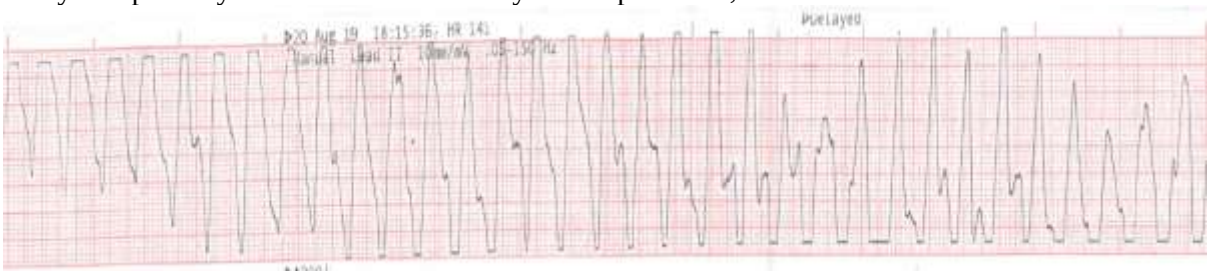

Figure 2: Rhythm strip changes

The patient was in cardiogenic shock with a GCS 3/15.
Resuscitation was initiated as per ACLS protocol and DC cardioversion done, he was shifted to cath lab for revascularization (Primary PCI) with amiodarone infusion. (Post intubation, patient had multiple episodes of VT which was brought under control with defibrillation and amiodarone infusion)

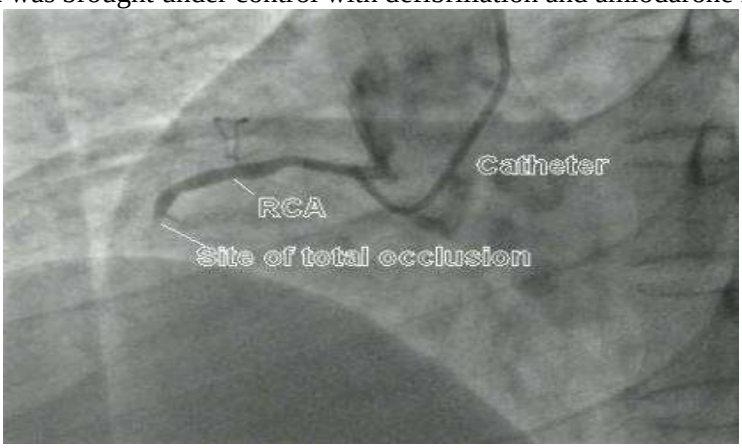

Figure 3: Angiogram findings

Investigations and Management

CAG was done, which showed Single vessel disease – Mid RCA 100% Thrombotic occlusion with diminutive RCA < 2mm causing significant dampening pressures on catheter insertion and hence stenting was deferred.

Intracoronary GP IIB /IIIA antagonist was given followed by IV infusion. The investigations showed Hb- 15.6 gm%, WBC- 24600 (N -95%/L-5%), Platelets- 3.01 lakhs, Creatinine- 1.24 mg%, Na- 138 mmol/L, K- 3.72 mmol/L, there was an increase in Troponin T from 367 to 671 and the TRANSTHORACIC ECHO showed Inferior wall regional motion abnormality with LVEF – 55%, Grade I LVDD.

Differential diagnosis

In the differential diagnosis the patient was presented with of polymorphic VT and IWMI in the background.

The patient was then shifted to ICU, where he had 1 run of sustained hemodynamically stable VT which terminated after a bolus of amiodarone. Patient had no further episodes of VT and was discharged in a stable state. He was on regular follow-ups.

Diagnosis as Brugada syndrome

The patient then came with complaints of retrosternal chest discomfort with dizzy spells. ECG was done and showed coved ST elevation in V1-V3 with negative T waves which was diagnostic of Brugada syndrome.

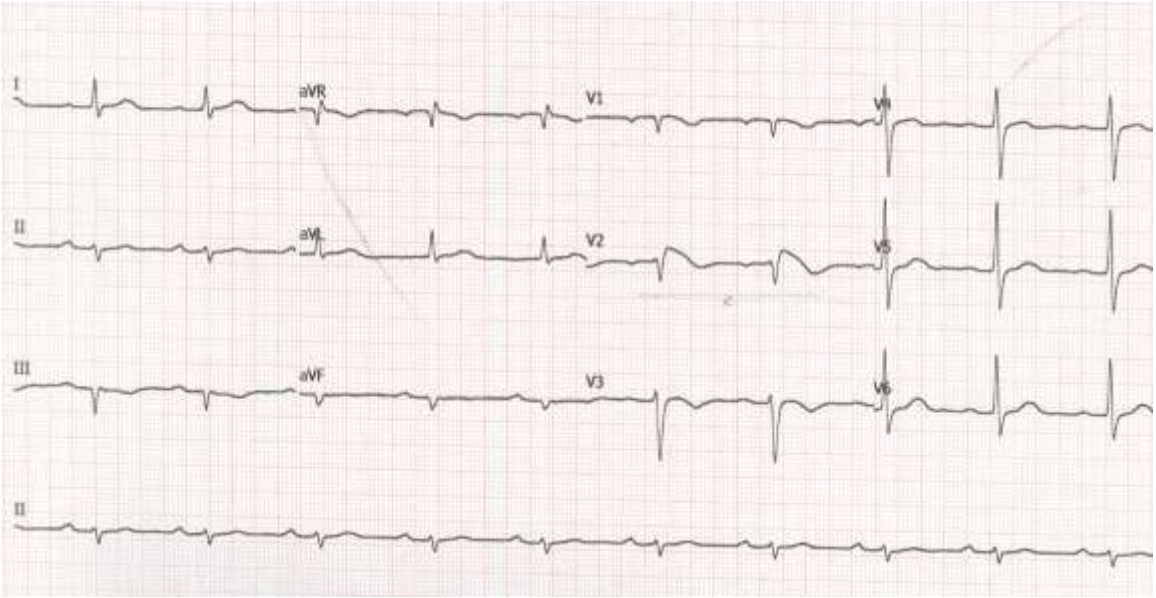

Figure 4: ECG showing coved ST elevation

The retrospective data showed that the patient's sister had the same complaints with a history of ICD insertion. Similar ECG patterns were observed in case of his daughter as well. With the diagnostic ECG changes and clinical criteria satisfied, for Brugada syndrome, patient was electively admitted for ICD insertion with BIOTRONIK PLEXA pro MRI SDX 65/15.

DISCUSSION

One of the genes linked to this syndrome is SCN5A, the gene encoding for the cardiac sodium channel. Mutations in SCN5A cause a functional reduction in the availability of cardiac sodium current in Brugada syndrome.(Smits *et al.,* 2002) The disease could manifest at first time as cardiac arrest without any previous symptom, and the electrocardiographic pattern could be intermittent, requiring a pharmacological challenge with Class I antiarrhythmic drugs to unmask ST elevation. Several conditions producing Brugada-like electrocardiographic patterns should be borne in mind and excluded while making a diagnosis of the Brugada syndrome. (Nelson *et al.,* 2019)

Our case report is about an atypical , rare presentation of polymorphic VT and IWMI in the background of Brugada syndrome, which was not suspected initially. The commonest presentation of Brugada is asymptomatic. Here, in this case, the retrospective data showed that the patient's sister had the same complaints with a history of ICD insertion. Similar ECG patterns were observed in case of his daughter as well.

Indication For AICD

The indications for ICD's have evolved from secondary prevention of sudden cardiac death to primary prevention in vulnerable patients. The most prominent international guidelines that has established indications for ICDs are the 2006 ACC/AHA/ESC guidelines on ventricular arrhythmias and sudden death, the 2012 update of the 2008 ACC/AHA/HRS device guidelines, and the 2012 ESC guidelines on heart failure (Tracy *et al.*,2012; Antzelevitch *et al.*, 2002)

There are no specific guidelines evolved so far in the treatment of Brugada syndrome, there is a need for consensus statement and guidelines for the same.

Learning objectives

- High index of suspension to diagnose Brugada syndrome in patients presenting with polymorphic VT.
- Possibility of Brugada syndrome is masked by ECG features of IWMI with poly morphic VT.
- Pharmaco invasive strategy in ACS especially if the culprit vessel is not amenable for intervention.
- Use of a DX lead AICD with RA sensing in place of dual chamber AICD.

Disclosures*:* The authors have nothing to disclose.
Funding**:** There are no funds involved with the work.

REFERNCES

Antzelevitch CH, Brugada P, Brugada J, Brugada R, Shimizu W, Gussak I, Perez Riera AR (2002). Brugada syndrome: a decade of progress. *Circulation Research*, **91**(12) 1114-8.

Kurita T, Shimizu W, Inagaki M, Suyama K, Taguchi A, Satomi K, Aihara N, Kamakura S, Kobayashi J, Kosakai Y (2002). The electrophysiologic mechanism of ST-segment elevation in Brugada syndrome. *Journal of the American College of Cardiology*, **40**(2) 330-4.

Nelson MR, Assar MD, Htaik O, Schussler JM. Brugada ST (2019). elevation masquerading as ST elevation myocardial infarction. In: *Baylor University Medical Center Proceedings*, **32**(1), 107-109. Taylor & Francis.

Smits JP, Eckardt L, Probst V, Bezzina CR, Schott JJ, Remme CA, Haverkamp W, Breithardt G, Escande D, Schulze-Bahr E, LeMarec H (2002). Genotype-phenotype relationship in Brugada syndrome: electrocardiographic features differentiate SCN5A-related patients from non–SCN5A-related patients. *Journal of the American College of Cardiology*. **40**(2) 350-6.

Tracy CM, Epstein AE, Darbar D, DiMarco JP, Dunbar SB, Estes III NM, Ferguson Jr TB, Hammill SC, Karasik PE, Link MS, Marine JE (2012). ACCF/AHA/HRS focused update of the 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. *Circulation*, **126**(14) 1784-800.