ABSTRACT

Brugada syndrome (BrS) is a rare genetic disease, of which its clinical manifestations include, but not limited to, syncope or sudden cardiac death. A 30-year-old Bangladeshi male patient with a past medical history of epilepsy was admitted following successful resuscitation from an out of hospital cardiac arrest secondary to ventricular fibrillation. Electrocardiogram (ECG) upon admission was suggestive of BrS type I. His old medical record showed similar ECG 2 months earlier when he had presented with syncope and was diagnosed with seizure. The correlation between BrS and epilepsy has been reported in the literature, discussing whether seizure is an uncommon presentation of BrS or whether epilepsy and BrS share similar genetic mutations that have the potential to cause both arrhythmia and seizures in some patients. Patients who present with seizure and ECG suggestive of Brugada pattern should be evaluated to rule out associated or underlying cardiac arrhythmia.

Key words: Ablation, Brugada syndrome, channelopathy, epilepsy, right ventricular outflow tract, seizure, sudden death, ventricular arrhythmia

INTRODUCTION

Brugada syndrome (BrS) is a genetic disorder inherited in an autosomal dominant fashion with a variable expression characterized by abnormal findings on electrocardiogram (ECG) in conjunction with an increased risk of ventricular tachyarrhythmia and sudden cardiac death.\(^1\)

Inherited idiopathic epilepsy and inherited cardiac arrhythmias both have been discovered to have mutations in ion channel subunit genes, and the association between these channelopathies has recently been reported in literature. Due to the similarities in presentation that may occur, a diagnosis of underlying BrS may be missed in some cases and labeled as epilepsy. We report a case of BrS, which was initially diagnosed and treated as epilepsy eventually successfully managed by an implantable cardioverter-defibrillator (ICD) implantation, then underwent epicardial right ventricular outflow tract (RVOT) ablation for recurrent ICD shocks for confirmed malignant arrhythmias.

CASE PRESENTATION

A 30-year-old Bangladeshi male patient was discovered to have experienced a sudden episode of loss of consciousness while talking with his friends. Emergency medical services arrived 20 min later and found the patient to be in cardiac arrest with ongoing bystander cardiopulmonary resuscitation (CPR). His initial cardiac rhythm was ventricular fibrillation (VF) as the initial rhythm following which CPR was started and delivery of direct current (DC) shock as per the advanced life support guidelines. Following the first DC shock, the cardiac rhythm changed to pulseless electrical activity arrest

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for which CPR was continued for 22 min until the return of spontaneous circulation. He was intubated during resuscitation and then transferred to the heart hospital emergency department. On physical examination, the patient had a blood pressure of 107/73 mmHg, heart rate of 120 bpm, regular rhythm, and was receiving mechanical ventilatory support. Notably, he had equal bilaterally reactive pupils with a normal respiratory and abdominal examination. Initial laboratory investigations were significant for white blood cells 18/μL, hemoglobin 16.6 g/dL, platelet count 282/μL, alanine transaminase 129 U/L, aspartate aminotransferase 102 U/L, troponin T 700 > 1100 > 500 ng/L, and a lactic acid of 3.9 mmol/L, all of which returned back to the normal limits within 2 days of receiving the supportive care. His renal function tests and electrolytes were within normal limits. A chest X-ray, computed tomography (CT) scan of his brain and CT pulmonary angiography were all unremarkable. His 12-lead ECG was significant for Brugada pattern type 1, with typical coved ST elevation of ≥2 mm in at least one right precordial lead [Figure 1]. The patient was admitted to the cardiology intensive care unit, and a hypothermia protocol was initiated following which the patient was extubated after 5 days without any permanent neurological

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**Figure 1:** Twelve lead electrocardiogram upon admission after the return of spontaneous circulation showing a pseudo right bundle branch block pattern with coved ST-segment elevation in the right precordial leads which is typical for Brugada type 1 pattern

**Figure 2:** Intracardiac electrogram showing one episode of ventricular fibrillation followed by appropriate implantable cardioverter-defibrillator shock
deficit. Upon reviewing the patient’s medical records, it was noted that he had initially presented to the Hamad General Hospital, Daha, Qatar, 2 months prior with a witnessed brief sudden episode of loss of consciousness and associated epileptiform seizure activity for almost 1 min, followed by the recovery and no postictal signs. He admitted having a history of similar episodes in the past and had been on sodium valproate in the past but had stopped taking it for more than 6 months. He had no family history of sudden cardiac death or epilepsy. There was no smoking history; he did not consume alcohol or any other elicit substances. His ECG at the time was documented to have Brugada type 1 pattern, and he was referred to both cardiology and neurology, after discussion between the teams, the working diagnosis was of epilepsy with an incidental finding of Brugada type 1 ECG. He was started on phenytoin with outpatient follow-up arranged with neurology and cardiology, before which he presented with his cardiac arrest. Before discharge, he was evaluated by an electrophysiologist and underwent implantation of a single-chamber ICD.

Six weeks later, the patient attended the device clinic for routine follow-up and mentioned that he had experienced four recent episodes of abnormal movements and brief loss of consciousness. ICD interrogation revealed four episodes of VF, which had been appropriately shocked by his ICD. The patient was admitted and underwent epicardial radiofrequency catheter ablation as a treatment for his recurrent VF and ICD therapy [Figures 2 and 3]. He was discharged without anticonvulsant medication and has been followed up for 14 months with no further episodes of arrhythmia or loss of consciousness until the present time.

DISCUSSION

BrS is a well-recognized cause of ventricular tachyarrhythmias and sudden cardiac death. The proposed mechanism consists of underlying inherited channel disorders associated with paroxysmal dysfunction of excitable tissues and may present with extracardiac features.[1]

Inherited idiopathic epilepsy as well as inherited cardiac arrhythmias is both attributed mainly to mutations in ion channels subunit genes. A variety of mutations have been identified in genetic studies, mainly the SCN5A and
SCN10A, which encode for subunits of a cardiac sodium channel causing syndromes such as BrS.\(^2\)

The coexistence of epilepsy and a cardiac channelopathy has previously been reported by L. Fauchier et al. in their case report of a male patient with a long history of epilepsy incidentally discovered to have BrS.\(^3\) Although there is no definitive evidence that any cardiac channelopathy may cause true epilepsy, there have been suggestions recently pointing toward the possibility of a mutation of an ion channel gene being coexpressed in the heart and brain, leading to dysfunction in both.\(^4\) This has been further substantiated in an animal study which demonstrated that the BrS-linked SCN5A gene mutation was selectively expressed in the limbic regions of the rat brain, providing a mechanism which may regulate excitability and rhythmic firing within these circuits.\(^5\) Furthermore, the presence of both epilepsy and BrS in a family confirmed to have the SCN5A mutation is also suggestive that the same mutation leading to cardiac arrhythmias may result in increased susceptibility for recurrent seizure activity.\(^6\)

Owing to the similarities in the clinical presentation of both conditions such as syncopal attacks, patients may either be mislabeled as epilepsy alone, or the simultaneous presence of both seizure disorder and BrS may be missed. Although the presence of a seizure does suggest a primary neurological problem, it may simply be a manifestation of cerebral hypoperfusion, also referred to as syncopal seizures.\(^6\)

We report a middle-aged male patient who was diagnosed with epilepsy due to his history of loss of consciousness. The importance of his BrS ECG pattern was not appreciated at the first hospital visit; he subsequently presented with VF cardiac arrest. Similar cases have been reported as BrS presenting with seizure as a result of ventricular arrhythmia-induced cerebral hypoperfusion.\(^7,8\) Furthermore, there are reported BrS cases associated with epilepsy where the patient was documented to have seizure with epileptiform activity on EEG in the absence of arrhythmia on the simultaneous ECG recording.\(^9\)

Catheter ablation for the management of recurrent ventricular arrhythmias is an emerging treatment option for BrS patients but is largely limited to tertiary or quaternary Electrophysiology (EP) Centers due to its technical complexity. Although long considered a “normal heart” ventricular tachycardia (VT) condition, there is increasing evidence of myocardial substrate changes, including inflammation and fibrosis in the RVOT and these regions of RVOT substrate are targeted during ablation [Figure 4]. This approach has a high success rate for controlling malignant arrhythmias, typically >90% freedom from VT/VF at 12 months, which was also born out in our case.\(^2-11\)

**CONCLUSION**

BrS is associated with seizure either by being one of the uncommon clinical manifestations or a presentation of associated epilepsy due to a channelopathy affecting both cardiac and brain excitable tissues. Patients who present with seizure and ECG suggestive of Brugada pattern should be carefully evaluated by a cardiologist to rule out associated cardiac inherited arrhythmia. Catheter ablation in BrS patients who are experiencing recurrent ICD therapy appears to be a highly effective strategy for the control of malignant arrhythmias.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

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