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Case Report

An Unusual Case of Brugada Syndrome in an 82-Year Old Black Hypertensive Man

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ARTICLE INFO

Article history:
Received: 13 May, 2020
Accepted: 6 June, 2020
Published: 30 June, 2020

Keywords:
Brugada syndrome
elderly African
hypertensive heart disease

ABSTRACT

Rationale: Brugada syndrome (BrS) is a cardiac ion channel disease that is caused by an autosomal dominant genetic abnormality. It is frequently seen among young and middle-aged adults of Asian descent and rarely in blacks.

Patient Concerns: We report an extremely rare case of an 82-year-old male known hypertensive with poor drug compliance, who suffered recurrent palpitations and pre-syncope. His electrocardiogram showed an unusual right bundle branch block with coved ST segment elevation in leads V1-V3.

Diagnoses: The patient was eventually diagnosed with Brugada Syndrome with background hypertensive heart disease. He could not have genetic testing due to unavailability and cost.

Interventions: The patient was treated with antihypertensives but could not afford a device implant. He was counseled to avoid risk factors such as fever, extreme physical and emotional exertions etc.

Outcomes: At his last clinic visit two months post diagnosis he still had similar symptoms but no syncope nor sudden cardiac arrest.

Lessons: To the best of our knowledge, this is possibly the first reported case of BrS in an elderly African with hypertensive heart disease.

Introduction

Brugada syndrome (BrS) is a genetic arrhythmogenic disease that has an estimated incidence between 4% and 12% in patients succumbing to sudden cardiac death (SCD) [1]. Although, the incidence of BrS is more in Japan and Southeast Asia (nearly 1%), it may not be so rare in other parts of the world [2]. An electrocardiogram (ECG) is essential to diagnose BrS. The American College of Cardiology/American Heart Association criteria of 2012 recognize 2 ECG patterns: pattern 1 (the coved pattern) and pattern 2 (the saddle-back pattern) that includes ECG repolarization types 2 and 3 [3]. Presence of either pattern is an indication for SCD prevention using an implantable cardioverter defibrillator (ICD) or ablation therapy. Hypertensive heart disease (HHD) is a complication of chronic uncontrolled hypertension manifesting as abnormalities in myocardial structure and function such as left ventricular hypertrophy, ischemic heart disease and heart failure, in the absence of other primary cardiovascular abnormalities [4].

Case Report

An 82-year-old known hypertensive for twenty years not compliant with his medications resulting in poor blood pressure control. After a prolonged 8-year absence from the clinic, he re-attended with gradual onset progressively worsening palpitations of three years' duration which became unbearable few days prior to presentation. There was accompanying dizziness, excessive sweating and feeling of impending doom without chest pain. Symptoms were episodic in nature, worse while resting or sleeping but severe enough to wake him before resolving spontaneously. Our patient also observed that ingestion of (α-methyl dopa) tablets worsened the symptoms.

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http://dx.doi.org/10.31487/j.JICOA.2020.03.07

No chest pain or heaviness was noted. There was no known family history of heart attack, resuscitated cardiac arrest or sudden cardiac death. He never drank alcohol nor used tobacco in any form. Past medical history indicated hypoglycemic coma and herniorrhaphy occurring 8 and 11 years earlier. Examination revealed an elderly man, conscious and alert with a normal temperature of 37.2°C. He had no pallor, cyanosis, jaundice, edema or enlarged peripheral lymph nodes. There was an infra-auricular firm immobile parotid mass measuring 8cm x 6cm, with no tenderness or differential warmth.

Basic neurologic, chest and abdominal exams were all normal. Cardiovascular exam revealed a pulse rate (PR) of 84beats/min with occasional missed beats, blood pressure (BP) of 168/84mmHg (left arm, sitting) and 170/84mmHg (right arm, supine), normal jugular venous pulsation, a displaced heaving apex with fourth heart sound and loud second heart sound (pulmonary component). Electrocardiography showed coved ST segment elevation of >2mm, followed by a negative T-wave from chest leads V1 to V3, with a right bundle branch block (RBBB) pattern and mild left axis deviation (-5°) as shown in (Figure 1) below. Provocative test was not done for our patient because he had type 1 ECG pattern of BrS. Echocardiography showed concentric left ventricular hypertrophy, grade one left ventricular diastolic dysfunction and mild degenerative aortic valve disease.

After adequate counseling the patient declined to travel outside the country for implantation of a cardioverter-defibrillator citing financial constraints. At three months follow up, His blood pressure was well controlled on oral anti-hypertensives though he still complains of episodic palpitations and pre-syncope of lesser intensity. He has been counseled on risk factors to avoid.

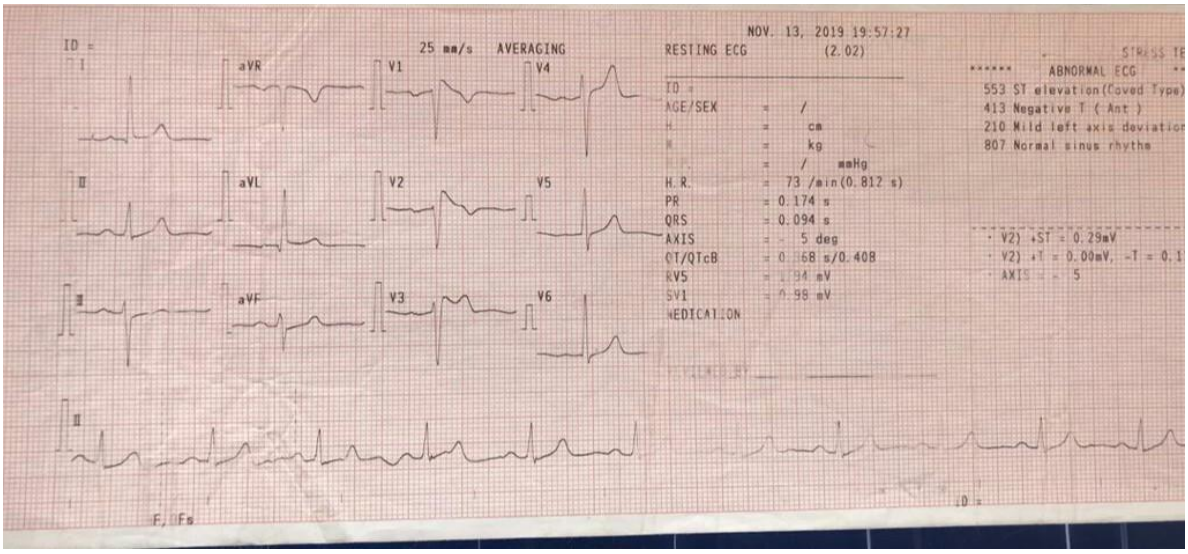

Figure 1: ECG of an 82-year-old African Hypertensive with coved ST-Segment elevation in Leads V1-3.

Discussion

We report this rare case of Brugada syndrome in an elderly African with background hypertensive heart disease to show that BrS might not be uncommon in Africans and in people of advanced age who are likely to have background heart conditions. Brugada syndrome is an arrhythmogenic disease characterized by ST-segment elevation and RBBB pattern in the right precordial leads [5]. Estimated world prevalence is 0.03% with higher frequency in males and Asians [6].

Symptom onset is usually between the 3rd and 6th decade with about 25% having a positive family history [6]. Our index case occurred in an elderly Nigerian man with typical ECG changes as in (Figure 1) but without a known family history suggestive of BrS or other causes of sudden cardiac death. Bonny *et al*. identified 5 black Africans (resident in France) with BrS with an average age of 50.6 years, 80% male preponderance and positive family history of SCD in only one patient [7]. The apparently low family history seen among Africans with BrS may be due to poor recall, unavailable medical records or inadequate investigation of sudden death.

This syndrome manifests as autosomal dominant mutations in the cardiac sodium channel gene SCN5A– a key pathogenetic mechanism that gives rise to most BrS cases though some may be due to a new genetic mutation or certain medications [8]. The importance of this syndrome is the association with sudden cardiac death in adults [5]. Genetic testing, although ideal was not done in our patient due to unavailability and high cost. Interestingly, no SCN5A mutation was documented among the 5 black Africans with BrS suggesting a possible genetic variation with race [7].

The abnormal heart rhythms seen in those with BrS often occur during period of high vagal tone such as following a heavy meal, at rest or even during sleep, as reported by our index patient. They may also be triggered by a fever or excessive alcohol intake [9]. According to a recent consensus document, Type 1 ST segment elevation, either spontaneously present or induced with the sodium channel-blocker challenge test, is considered diagnostic. Type 2 and 3 may lead to suspicion, but provocation testing is required for diagnosis [10].

Apart from idiopathic cases, RBBB can have several causes including, acute MI, RVH, congenital heart disease, myocarditis, pulmonary embolism and rarely Wolff-Parkinson White syndrome. ST elevation can occur in acute MI, coronary vasospasm, pericarditis, LVH, BrS and fascicular bundle branch blocks [11]. Acute myocardial infarction can be readily diagnosed on ECG and so is ventricular hypertrophy using appropriate voltage criteria. For pericarditis, ST elevation occurs in widespread leads with concave morphology and unaltered R wave

amplitude [12]. Coronary vasospasm should be suspected with transient ECG changes resembling infarction that quickly normalizes in response to nitroglycerin [13]. Pulmonary embolism frequently presents with sinus tachycardia but in few cases the characteristic $S_IQ_{III}T_{III}$ classic pattern may be seen.

Anan Milmat *et al.* reported from the Survey on Arrhythmic Events in Brugada Syndrome (SABRUS) that a first arrhythmic event occurred 6.7 years later in patients with prophylactic ICD implants compared to those with aborted cardiac arrest. The former group also had a higher positive family history of SCN5A mutations and sudden cardiac death [13, 14]. Although our patient had an ECG diagnosis of BrS, he was unable to get a cardioverter-defibrillator inserted due to unavailability and cost. Bonny *et al.* have clearly highlighted the peculiar challenges of arrhythmia management in the African setting and recommend improved funding, personnel training and preventive care as possible solutions [15].

Conclusion

Brugada syndrome, although relatively rare, can occur in elderly Africans. Further research and community ECG screening will help identify at-risk individuals requiring prompt intervention.

Abbreviations

BrS: Brugada Syndrome
ECG: Electrocardiogram
ICD: Implantable Cardioverter Defibrillator
SCD: Sudden Cardiac Death

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