Open Journal of
Clinical & Medical
Case Reports

Volume 4 (2018)
Issue 5

ISSN 2379-1039

Brugada syndrome– A case report

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Abstract

Brugada syndrome (BrS) is a genetic disorder found in 1.34 per 1,00,000 persons characterized by sudden cardiac death (SCD) [3] associated with one of several ECG patterns characterized by incomplete right bundle-branch block (RBBB) and ST-segment elevations in the anterior precordial leads. Brugada syndrome is an autosomal dominant channelopathy caused by mutation in the sodium channel gene SCN 5A, which encodes the cardiac voltage-gated sodium channel NaV1.5. These mutations reduce the sodium current (INa) available during the phases 0 and 1 of the cardiac action potential. It occurs mainly in the Asian males usually presenting with life threatening ventricular arrhythmias or sudden cardiac death [7]. Here, we report a case of 38 years old male presenting with dizziness and syncope.

Keywords

brugada syndrome; ICD implantation

Introduction

Brugada syndrome is arrythmogenic disease characterised by ST segment elevation in the right precordial leads, right bundle branch block and without a structural cardiomyopathy. It is responsible for the sudden cardiac death in young people [2]. Being autosomal dominant channelopathy inheritance, it is caused by mutation in the gene SCN 5A [4] leading to a loss of function of the cardiac sodium channel by different mechanisms. The most common symptoms are dizziness and syncope.

Case Presentation

A 38 year old man admitted to hospital with complaints of syncope and sudden onset of chest discomfort with palipitaion. He was an ex-smoker, non-alcoholic, non-hypertensive and non-diabetic. But his family members revealed that his father died at the age of 50 years. Physical examination was normal. Vitals BP 130/80 mmHg, Pulse rate 80 beats/min. Cardiovascular, nervous system, respiratory and gastrointestinal system examination was unremarkable. Laboratory investigations including hemogram, lipids, electrolytes, liver and renal profile as well as cardiac biomarkers were normal. On 12 leads standard electrocardiogram there is normal sinus rhythm, incomplete RBBB, ST segment elevation with T wave inversion in v1 and v2, consistent with the classical coved “Brugada Type I pattern”. On ECHO evaluation all chambers are normal sized. The diagnosis of Brugada syndrome was made on the history

and ECG changes. Patient underwent ICD Implantation and hospitalized for 7 days. Patient was stable, feels better hence discharged.

Discussion

Brugada syndrome is a potentially life-threatening heart rhythm disorder that is inherited. People with Brugada syndrome have an increased risk of abnormal heart rhythms from the lower chambers of the heart (ventricular arrhythmias). Brugada syndrome is an autosomal dominant inheritance of mutations in the gene SCN 5A which encodes for the α-subunit of the cardiac sodium channel [4]. The reported mutations include missense mutation, nonsense mutation, nucleotide insertion/deletion (which may alter mRNA splicing or create a stop codon by shifting the open reading frame), and splice site mutation.

The diagnosis of Brugada syndrome is made on the clinical and typical ECG findings [1]. It is characterized by a coved ST segment elevation of at least 2mm followed by a negative T wave with little or no is electric separation and present in more than one right precordial leads (from V1 to V3) [2]. Repeated ECG recordings or signal-averaged ECG may be useful in the risk stratification. Brugada syndrome is accountable for life threatening ventricular arrhythmias and sudden cardiac death in majority of patients [5]. Arrhythmic events are observed at rest or while asleep, most frequently from 12 AM to 6 AM, less frequently in the evening, and the least during the daytime. Sudden cardiac arrest in patients with Brugada Syndrome is usually not associated with exercise [8].

There is no effective pharmacologic treatment for sudden cardiac death due to Brugada syndrome, although isoproterenol and quinidine is found to be beneficial for treatment of electrical storm in Brugada syndrome. Automated cardioverter-defibrillator implantation is the only treatment for the patients [6]. Genetic testing can detect the high risk relatives. As our patient was having typical changes of Type I Brugada syndrome on ECG along with family history of sudden death of his father, he was considered as a high risk case following which he was advised for implantation of implantable cardioverter-defibrillator.

Conclusion

Brugada syndrome should be suspected in patients showing characteristic ECG changes in the form of ST segment elevation followed by inverted T wave in V1 and V2 leads along with clinical history of the patient as well as of his family members. Early recognition and intervention can save the life of the patient.

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Manuscript Information: Received: December 04, 2017; Accepted: March 05, 2018; Published: March 15, 2018

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Citation: Joseph S, Nair SK. Brugada syndrome– A case report. Open J Clin Med Case Rep. 2018; 1385.

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