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Case Report

BRUGADA SYNDROME – A CASE REPORT

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ABSTRACT
Brugada syndrome is an autosomal dominant channelopathy caused by "loss of function", mutation in the sodium channel gene SCN 5A. It occurs mainly in the Asian males usually presenting with life threatening ventricular arrhythmias or sudden cardiac death. It is characterised by ST segment elevation in the right precordial leads and right bundle branch block. Here, we report a case of 50 years old male presenting with dizziness and syncope.

Keywords: *Brugada Syndrome; Autosomal Dominant; Genetic Testing*

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 death in young people (Brugada and Brugada, 1992). Its prevalence is 0.10% in the world (Fowler SJ and Priori, 2009). Being autosomal dominant channelopathy inheritance, it is caused by mutation in the gene SCN 5A in most of the cases (Chen *et al.,* 1998).

CASES
A 50 years old male presented with dizziness and syncope while he was standing. After 5 minutes of adopting the supine position, he recovered. There was no history of chest pain, dyspnoea and palpitation. He was non smoker, non alcoholic, non hypertensive and non diabetic. But his family members revealed that his father died at the age of 45 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. A 12 leads standard electrocardiogram (ECG) revealed ST segment elevation and T wave inversion in leads V1 and V2, consistent with the classical coved "Brugada Type I pattern". 2D Echocardiography and X-ray chest revealed no abnormality.
The diagnosis of Brugada syndrome was made on the history and ECG changes. Patient was referred to higher centre for automated implantable cardioverter-defibrillator implantation.

DISCUSSION
Brugada syndrome is an autosomal dominant inheritance of mutations in the cardiac sodium channel gene with the result that mutations in the gene SCN 5A are responsible for most of the cases (Chen *et al.,* 1998). The diagnosis of Brugada syndrome is made on the clinical and typical ECG findings. Type I is a coved ST segment elevation of atleast 2mm followed by a negative T wave with little or no isoelectric separation and present in more than one right precordial leads (from V1 to V3) and one of the following: documented ventricular fibrillation, polymorphic ventricular tachycardia, and a family history of sudden cardiac death at 45 years of age or younger, Type I pattern on ECG in family members, ventricular tachycardia which can be induced with programmed electrical stimulation, syncope, nocturnal agonal respiration, while Type II and III patterns on ECG showed the same 2mm or greater J point elevation but a positive T wave – "the saddle back appearance" to the ST-T portion with conversion to Type I following challenge with sodium channel blocker and of the clinical features is described above (Antzelevitch *et al.,* 2005). Brugada syndrome is accountable for life threatening ventricular arrhythmias and sudden cardiac death in majority of patients. Automated cardioverter-defibrillator implantation is the only treatment in such patients (Epstein *et al.,* 2008; Priori *et al.,* 2002; Aguilar – Shea and Gallardo Mayo, 2015). 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 referred to higher cardiac center for implantation of 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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