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RARE CASE OF SUDDEN CARDIAC ARREST SURVIVOR WITH BRUGADA SYNDROME IN A PERIPHERAL ARMY HOSPITAL - A CASE REPORT

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ABSTRACT

Brugada syndrome is a potentially life-threatening heart rhythm disorder. First described in 1992 by the Brugada brothers, the disease has since had an exponential rise in the numbers of cases reported, to such an extent that it is the most common cause of sudden cardiac arrest in males <40 .1 It's incidence seems to be particularly high in Southeast Asia.

We describe a case of Brugada syndrome in a young army soldier who presented in a peripheral hospital with complaints of episodic loss of consciousness. Patient later on had a sudden cardiac arrest and was revived after 15 minutes of Cardiopulmonary resuscitation and maintained on ventilator and ionotropic support for 3 days. Patient on becoming hemodynamically stable was airlifted to a tertiary care hospital where advanced cardiac workup revealed it as a case of Brugada syndrome. Pacemaker was inserted and patient has been stable since then.

KEYWORDS : Brugada syndrome, Cardiopulmonary Resuscitation

INTRODUCTION

Brugada syndrome is a potentially life-threatening heart rhythm disorder. Many people who have Brugada syndrome don't have any symptoms, and so they're unaware of their condition. First described in 1992 by the Brugada brothers, the disease has since had an exponential rise in the numbers of cases reported, to such an extent that it is the most common cause of sudden cardiac arrest in males <40 .[1] It's incidence seems to be particularly high in Southeast Asia where it had been previously described as Sudden Unexplained Nocturnal Death Syndrome (SUNDS), in Japan as pokkuri ("sudden and unexpectedly ceased phenomena") and in Thailand as Lai Tai ("death during sleep").[1]

CASE REPORT

26 year old serving soldier presented with 3 episodes of transient loss of consciousness in last 6 hours. Each such episode lasted for around 1-2 min followed by complete regaining of consciousness. History of fall and injury to lips and nostril were present. General and local examination were unremarkable. All routine haematological and biochemical investigations were within normal range .Family history revealed that father is a known case of Epilepsy on treatment for past 20 years.

Pt was admitted in Intensive Care Unit with provisional diagnosis of Syncope. Five hours after admission, patient had another episode of loss of consciousness associated with facial grimacing and abnormal movements of the body . Patient was sedated with intravenous diazepam. Patient was also dilantinised with 1000mg of phenytoin sodium given over 90 minutes. All vital parameters and ECG were normal. (Figure - 1)

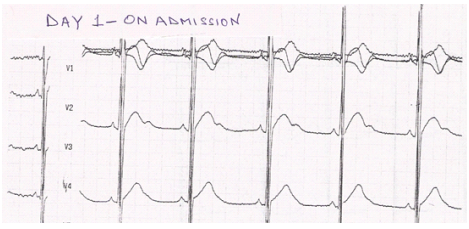

Figure - 1

Patient was placed on continous vital parameters monitoring in ICU and planned for evacuation to a higher center for neuroimaging and further management. Five hours later patient became unconscious. No pulse or Blood pressure were recordable. Cardiac monitor showed broad QRS complex Tachycardia followed by asystole. Cardiopulmonary Resuscitation (CPR) was started and Endotracheal intubation done. Inj Adrenaline (1:1000) given and CPR continued. After 15 min of CPR, Ventricular tachycardia (VT) (with broad QRS complex) rhythm was observed. Synchronized electrical cardioversion using 200 Joules was done for Pulseless VT as per existing CPR guidelines. After 2 attempts at Direct current (DC) Cardioversion, Normal sinus rhythm was established. Patient was placed on elective ventilation and required 3 drugs inotropic support to maintain Mean arterial pressure of more than 70 mm of Hg. Inj Amiodarone 300mg iv (loading dose) was given and Amiodarone infusion was started at 0.5 mg/min for 24 h. Central Venous catheterization was carried out and Central venous pressure measured. 24 hours post cardiac arrest patient improved and inotropes were tapered off. All necessary investigations were done which were normal. Patient was given weaning off trial to look for feasibility for extubation. Inotropes were stopped. Patient became fully conscious and alert and was extubated . He was put under close observation over next 24h in ICU. No abnormality in cardiac rhythm was present. Patient was transferred to a higher center.

At the higher center Computerized Tomography (CT) scan of Brain was done which was normal. Holter monitoring and 2D Echocardiogram were also normal. ECG-for the first time revealed coving of ST segments in lead V1-V4 with T wave inversion (Figure- 2).

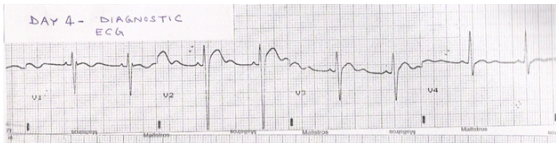

Figure-2

This alongwith recurrent episodes of syncope led to formulation of diagnosis of BRUGADA SYNDROME (Type 1). Single lead Automated Implantable Cardioverter-Defibrillator was inserted. Coronary Arteriography was also done which revealed a normal study. Patient has remained asymptomatic since then till date.

DISCUSSION

The most common underlying cause of sudden cardiac arrest is ventricular fibrillation (VF) or pulseless ventricular tachycardia[2].Survival rate with discharge out of hospital for SCA is <10% .[3]

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Identification of the underlying cause of syncope is difficult and might not be established. A diagnosis is important, however ,to determine the prognosis for each patient, which can vary from benign cases of vasovagal syncope to cardiac syncope, which is associated with a more than twofold increase in mortality rate.[4] Although cardiac-related causes only account of a relatively small proportion of cases (14%), they should always be excluded because affected individuals have a worse prognosis.[5] Although electrocardiography is an important part of the initial assessment of a patient with syncope, it is diagnostic in only 5% of cases and suggests a potential cause in a further 5% of patients. Any abnormalities in a baseline electro cardiogram, however, are predictive for an increased risk of sudden death, emphasizing the importance of investigating possible cardiac defects. However, in our patient the baseline ECG was normal. Conduction disturbances, pre-excited QRS complexes, a prolonged or shortened QT interval, epsilon waves, Q waves or a Brugada pattern are all indicative of arrhythmic syncope.[5]

Three types of repolarization patterns in the right precordial leads are recognized in Brugada Syndrome.[6,7] Type 1 ST-segment elevation is diagnostic of Brugada syndrome and is characterized by a coved ST-segment elevation ≥2 mm (0.2 mV) followed by a negative T wave. Type 2 ST-segment elevation has a saddleback appearance with a high take-off ST-segment elevation of ≥2 mm followed by a trough displaying ≥1 mm ST elevation followed by either a positive or biphasic T wave. Type 3 ST-segment elevation has either a saddleback or coved appearance with an ST-segment elevation of <1 mm. These three patterns may be observed sequentially in the same patient or following the introduction of specific drugs. Type 2 and 3 ST-segment elevation should not be considered diagnostic of the Brugada syndrome. Brugada syndrome is definitively diagnosed when a Type 1 ST-segment elevation (Brugada ECG) is observed in more than one right precordial lead (V1-V3), in the presence or absence of sodium channel blocking agent, and in conjunction with one or more of the following: documented VF, polymorphic ventricular tachycardia (VT); a family history of Sudden cardiac death (SCD) (<45 years old); coved-type ECGs in family members; inducibility of VT with programmed electrical stimulation (PES); syncope; or nocturnal agonal respiration.[6-9]

The presence of a syncope in a patient with Brugada syndrome is associated with an increased risk of sudden death or ventricular fibrillation, Since the patient described had a high risk of malignant arrhythmias, an implantable cardioverter-defibrillator (ICD) was indicated and implanted. This is the only effective treatment currently available for patients with Brugada syndrome.[10]

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