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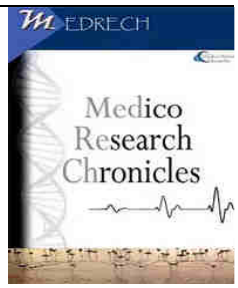

A CASE OF BRUGADA SYNDROME

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ARTICLE INFO | **ABSTRACT** | **CASE REPORT**

Article History
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A 25-year male came to OPD who had Upper Respiratory Tract Infection with fever for 3 days followed by sudden unconsciousness while on duty. He had spontaneous recovery from unconsciousness approximately after 1 hour. He was taken to a nearby hospital where he was evaluated initially. The patient was not a known case of Diabetes Mellitus (DM), Hypertension (HTN), Congenital, Rheumatic or Coronary heart disease. There was no past history of head trauma, epilepsy or similar occurrence. There is no family history of sudden death at a younger age. On examination he was found to have the following clinical features:

- Conscious & alert, Febrile (Temp 100°F)
- Pulse 98/Min, regular, all peripheral pulses well felt, no carotid bruit
- BP 110/80 mmHg
- Throat – mildly congested
- Lungs- bilateral normal vesicular breath sound with no added sound.
- Heart – S1, S2 normal no added sound.
- Other systemic examination revealed no abnormality.

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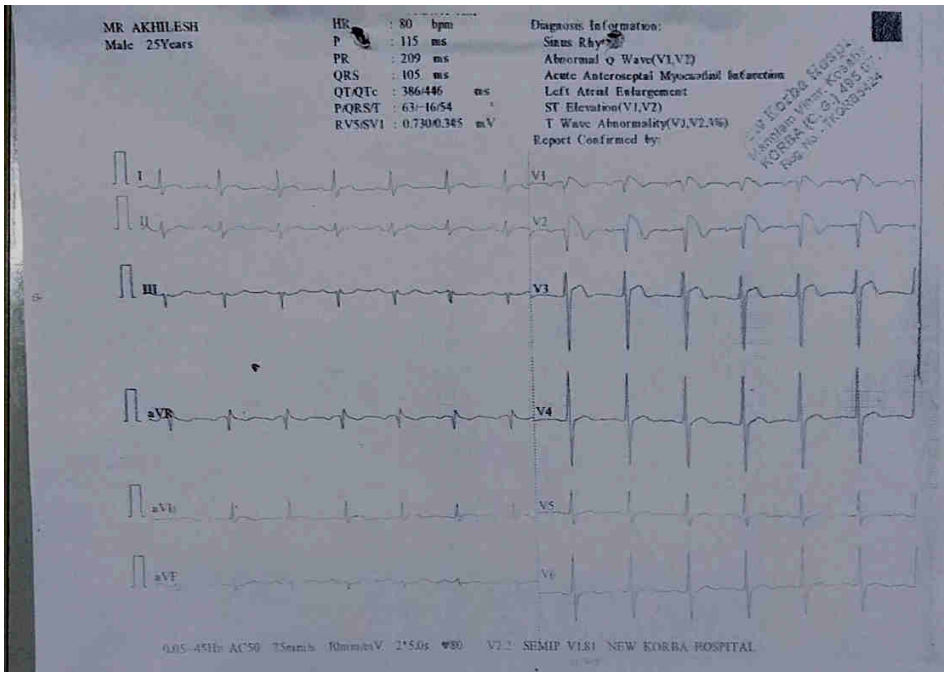
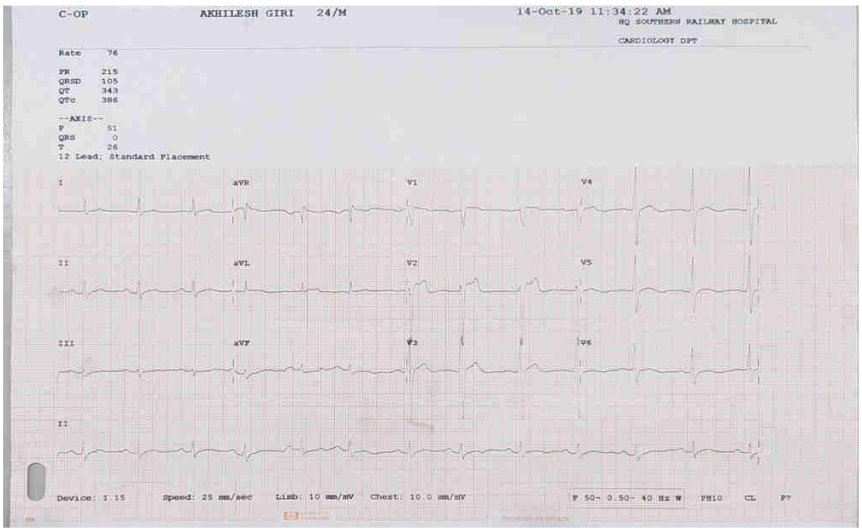
The preliminary laboratory blood tests were done, which revealed the following reports:

- Hb -12.7gm%
- TLC 6700, DC- Normal, Platelets- 1.7 L
- Random Blood Glucose-146 mg/dl,
- Liver Function Tests, Renal Function Tests, Lipid Profile and Electrolytes were normal.
- Malaria Parasite and Dengue (Ig G and Ig M) were Negative.

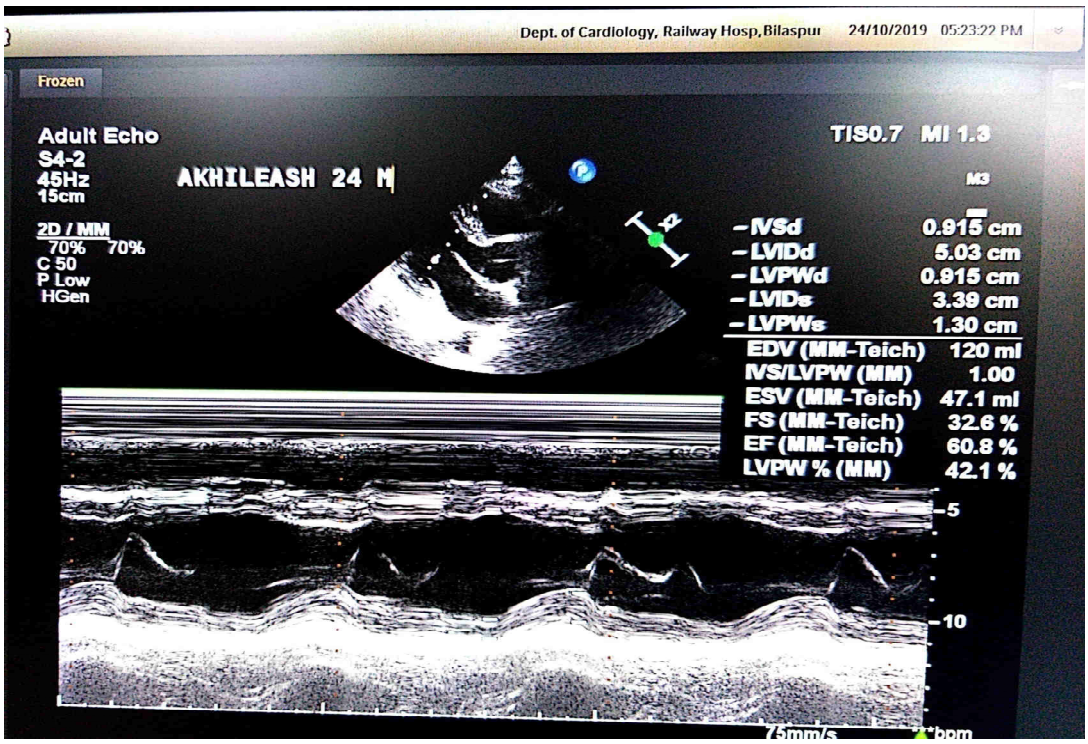
ECG taken within one hour of event in the initial treatment hospital revealed the following features.



(Pic. No.1) This ECG shows "coved" ST-segment elevation followed by a negative T wave in V1 & V2. The patient was treated with conventional treatment with oral Tab. Paracetamol 500mg 1 tab. thrice daily, Tab. cetirizine 10 mg twice daily and Tab. Cefuroxime 500mg twice daily in the initial evaluation center and was referred to our center. At our center the patient was asymptomatic. His clinical examination did not reveal any abnormalities. The ECG taken at our center on 3rd day was like this: (Pic. No.2)



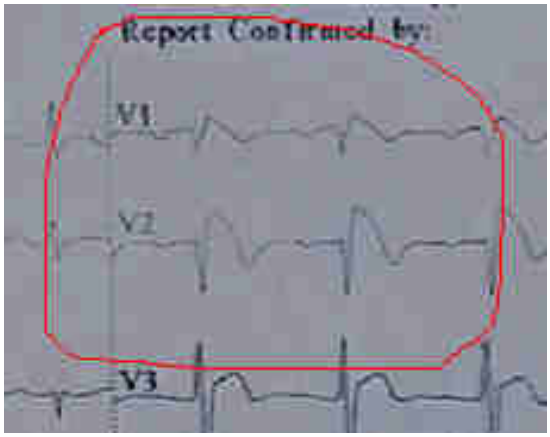
The Echocardiography done at our center revealed no structural abnormality.



(Pic. No.3) Other relevant investigations like EEG and MRI were found to be normal.

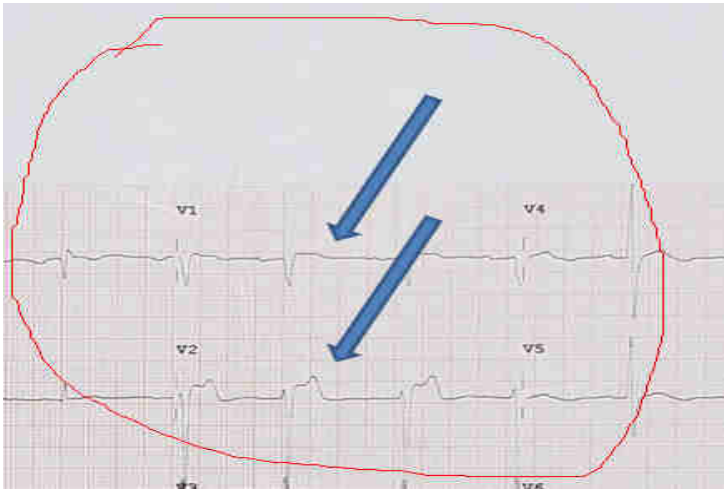
CLINICAL SUMMARY:

A young man of 24 Year had one episode of unconsciousness on 3rd day of febrile illness without a past history of epilepsy, head trauma.



(Pic. No.4)

ECG during episode was found to have a "coved" ST segment elevation followed by a negative T wave in V1 & V2



After clinical stabilization the subsequent ECG was having-a "saddleback" appearance of the ST segment in lead V1 with ST segment elevation

(Pic. No.5)

Echo was noncontributory, MRI head and EEG –were normal. Other relevant investigations in view of fever and unconsciousness were also noncontributory. With the above clinical history, features, and investigations it was diagnosed as *BRUGADA SYNDROME*.

DISCUSSIONS

Brugada syndrome is an arrhythmogenic disease responsible for sudden cardiac death in individuals with structurally normal hearts.[1] Three ECG patterns of Brugada syndrome has been described. Type 1 features ≥2 mm of downsloping or *coved shaped* ST-segment elevation followed by a negative T wave. Type 2 has ≥2 mm of ST elevation at the terminal portion of the ST segment, with a positive T wave that creates a *saddleback* appearance. Type 3 may have either coved shaped or saddleback ST-segment elevation with <1 mm magnitude of elevation at the terminal portion (see below Pic. No.6)[2] The penetrance and expressivity are highly variable, like some cases may be in the lifelong asymptomatic state and some may succumb to Sudden Cardiac Death (SCD) in the first year. It is inherited as an autosomal dominant trait (although more than half are sporadic). Basically, this is a sort of cardiac action potential disorder where the loss of function mutations are seen in Na^+ and Ca^+ ion entry by gene SCN5A and CACNA1C respectively. The gain of function mutation occurs in K^+ exit by KCNE.[3]

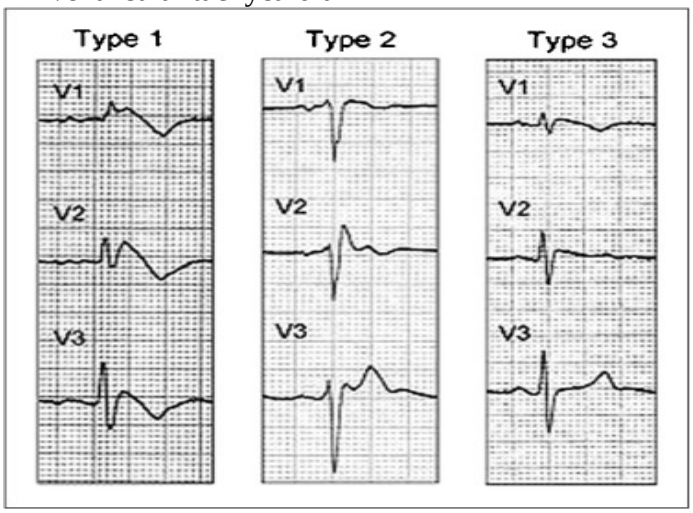
Molecular background of Brugada Syndrome: 18–30% of cases of Brugada syndrome result from loss of function mutations in the SCN5A gene, which encodes a subunit of a cardiac sodium channel. Temperature-dependent gating changes in these sodium channels have been identified, suggesting a possible molecular mechanism for the appearance of the Brugada ECG patterns in febrile patients. Previous reports have documented Brugada ECG patterns arising in the context of a febrile illness such as pneumonia, bacteremia, viral and gastrointestinal infections. Judicious use of paracetamol to prevent fever may help such cases to prevent fatal arrhythmia.[4]

Definite diagnostic criteria for Brugada Syndrome:

It requires the presence of a Type 1 ECG pattern, or conversion of Type 2 or Type 3 pattern to Type 1 following provocation testing with a sodium channel blocker, plus one the following:

- Documented ventricular fibrillation
- Self-terminating polymorphic ventricular tachycardia
- Family history of sudden cardiac death at age <45
- Type 1 ECG pattern in a family member

- Electrophysiological induction of ventricular tachycardia
- Unexplained syncope felt consistent with a tachyarrhythmia**.**



(Pic. No.6)

The ECG changes can be provoked in the electrophysiology lab by infusing ajmaline or procainamide

In our case, the young man of 25 years has no definite family history of sudden cardiac death. A strong possibility of unexplained syncope consistent with a tachyarrhythmia cannot be ruled out. The typical ECG pattern "coved" ST-segment elevation followed by a negative T wave in V1 & V2 during or just after recovery from unconsciousness and subsequent change in its pattern to a "saddleback" appearance of the ST segment in lead V1 with ST-segment elevation strongly favored the diagnosis of Brugada syndrome.

MANAGEMENT

Non-pharmacological

In an acute emergency- (e.g., Electrical storm)- Defibrillation /Other resuscitation remains the mainstay of treatment.

Pharmacological

IV Isoproterenol/ Isoprenaline (Enhancement of the ICa channels) OR IV Quinidine (INa)

Placement of ICDs is known to be the only and most powerful treatment modality for the prevention of sudden cardiac death in patients with a history of VF and/or aborted sudden cardiac death, i.e., for the so-called secondary prevention of sudden cardiac death. In this case implantation of an AICD stands to be a Class II A indication.

Drugs to be avoided in Brugada syndrome

Cardiovascular	Antiarrhythmic agents/Sodium channel blocker
	Class IA: ajmaline, cibenzoline, *disopyramide*, procainamide
	Class IC: *flecainide*, pilscainide, *propafenone*
	Beta-blocker: propranolol
	Calcium channel blocker: *diltiazem, nifedipine*
	Potassium channel opener: *nicorandil*
	Nitrate: *isosorbide dinitrate, nitroglycerine*

Psychiatric	**Phenothiazine**: cyamemazine, perphenazine
	Selective serotonin reuptake inhibitor: **fluoxetine**
	Tricyclic antidepressant: amitryptaline, nortriptyline, desipramine, clomipramine
	Tetracyclic antidepressant: maprotiline
Other	Dimenhydrinate, alcohol intoxication, cocaine intoxication

CONCLUSION

The Brugada syndrome can be revealed on ECG during a febrile illness. Patients with an induced Brugada pattern on ECG may be at particularly high risk of malignant arrhythmias and sudden cardiac death. These patients should receive antipyretic medications and have their acute illness managed aggressively. Follow-up with a specialist in cardiac electrophysiology and additional diagnostic testing after the acute illness has resolved is desired for any patient with an electrocardiographic Brugada pattern.

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