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Case Report

Brugada Syndrome Unmasked by Urinary Tract Infection

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Abstract
Brugada syndrome is an characteristic electrocardiogram finding which indicates risk of arrhythmia and sudden cardiac arrest leading to death. A characteristic downsloping st elevation with coved morphology and t wave inversion in V1-V3 is indicative of brugada syndrome and is often unmasked by fever and certain drugs. Here we present a case of 56 year old male in without any cardiac complaints in whom brugada syndrome was unmasked by fever due to urinary tract infection and treating the underlying infection and fever caused the electrocardiogram changes to subside.
Keywords: *brugada syndrome, brugada sign, fever, arrhythmia, sudden cardiac death.*

Introduction

Brugada syndrome was recognized and described as a new clinical entity by Spanish cardiologists paedro brugada and joseph brugada and is caused mostly due to underlying genetic disorder and is diagnosed by characteristic electrocardiography finding of a persistent ST elevation greater than 2mm in the electrocardiographic leads v1-v3 with a right bundle branch block and is often asymptomatic and unmasked by fever and certain drugs and carries the risk of malignant arrythmia and sudden cardiac death.[1]

Case Report

56 year old male presented with complaints of fever which is of high grade for 3 days along with complaints of burning micturition for 5 days and patient denied any history of chest pain, dyspnea, palpitations and syncope. There is no remarkable family history and no history of sudden death in relatives. On examination, he was febrile with a temperature of 39.7°c and heart rate of and remaining vital signs were within normal limits. Urine routine examination was showing plenty of pus cells and chest x ray was normal. Electrocardiography showed an downsloping ST elevation and right bundle branch block in V1, V2, with a coved morphology followed by t wave inversion in v1, v2. Patients electrolytes were normal and cardiac enzymes were negative and echocardiography ruled out structural heart disease. Antipyretic was given and antibiotic was started and on day 2 patient was afebrile and electrocardiography done showed only right bundle branch block changes in leads V1,V2.

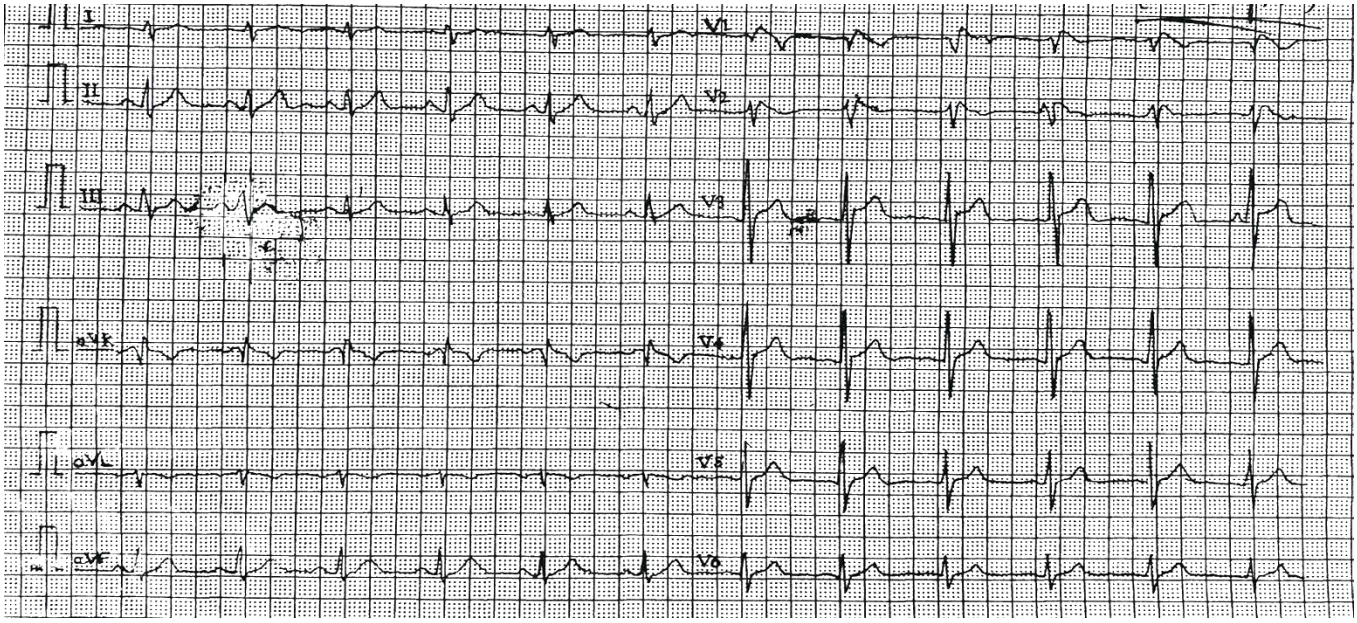

Figure 1 Electrocardiography showed an downsloping ST elevation and right bundle branch block in V1,V2, with a coved morphology followed by t wave inversion in v1,v2.

Discussion

The brugada syndrome is commonly diagnosed by ECG during sinus rhythm showed right bundle branch block, normal QT interval and persistent ST elevation in precordial leads V1,V2,V3 and is not explained by electrolyte disturbances, ischaemia or any structural heart disease.[1] There are three types of brugada syndrome as following Type 1 is described as a prominent coved ST-segment elevation displaying j wave amplitude or ST-segment elevation >2mm at its peak, followed by a negative T-wave, with little or no isoelectric separation in right precordial leads Type 2 was defined as high take off of ST-elevation, but j wave amplitude gives rise to gradually descending ST-segment elevation remaining 1mm above the baseline followed by a positive or biphasic T wave that results in a saddle back configuration. Type 3 is a right precordial ST-segment elevation of <1mm of saddle back type, coved type, or both.[2] Only type 1 pattern is diagnostic either spontaneously or after provocative drug test, whereas type 2 pattern may raise suspicion of brugada syndrome but diagnosis can be made only when type 1 pattern appears or is induced by sodium channel blockers. Type 3 brugada pattern is not considered nowadays.[3] Most common mutation associated with brugada syndrome is mutation in the cardiac sodium channel gene SCN5A, the gene encoding for the α-subunit of the sodium channel.[4] Febrile illness due to various causes is known to induce premature closing of the sodium channels leading to unmasking of the brugada syndrome[5] Drugs like sodium channel blockers, anaesthetics, cocaine, methadone, antihistaminics, electrolyte imbalance.[5] Recent studies show that up to 11% of patients with brugada syndrome have spontaneous early repolarization pattern in the inferior and lateral leads and these group of patients seems to have more severe phenotype.[6] It is often associated with malignant arrhythmia and may lead to sudden death.[7] Follow up of patients with brugada syndrome showed low incidence of severe arrhythmia after the first clinical event was diagnosed and previous history of aborted sudden cardiac death and syncope predicted adverse outcome.[8] Application of implantable cardioverter-defibrillator in persons who are at high risk is life saving treatment modality.[9]

Conclusions

Brugada syndrome is a rare disorder of arrhythmia and may be fatal at times and hence needs proper attention and diagnosis. Rapid diagnosis and treatment of underlying aggravating factors and

application of intra cardiac defibrillator will be life saving for the patient.

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