Exercise-induced syncope and Brugada syndrome

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ABSTRACT

Brugada syndrome (BrS) is a hereditary condition that is characterized by ST elevation, ventricular tachycardia or fibrillation, and sudden cardiac death in otherwise healthy patients. Life-threatening arrhythmias generally occur, while at rest, with fever or during vagotonic states. Exercise is generally not considered a trigger for ventricular arrhythmias or syncope in patients with BrS. We describe a patient who presented with exercise-induced syncope, ventricular tachycardia during an exercise test, and was found to be both genotypically and phenotypically positive for BrS. This case highlights a potentially important role of exercise testing in diagnosing and risk stratifying certain patients with BrS.

Keywords: Brugada syndrome, exercise, treadmill stress testing, ventricular fibrillation

INTRODUCTION

Brugada syndrome (BrS) is an inherited arrhythmogenic condition characterized by marked ST elevation in the precordial electrocardiographic (ECG) leads and an increased risk of sudden cardiac arrest due to ventricular tachycardia or fibrillation in young and otherwise healthy patients. BrS is thought to be responsible for more than 20% of sudden cardiac death in patients with structurally normal hearts.[1] Genetic basis for the syndrome has been attributed to mutations in *SCN5A*, the gene encoding for the α subunit of the sodium channel in cardiac muscle tissue.

Symptoms of BrS include ventricular arrhythmias and syncope, which usually occur at rest or while asleep.[2,3] Exercise is generally not considered a trigger for ventricular tachycardia or syncope in patient with BrS. We describe a patient who presented with exercise-induced syncope, ventricular tachycardia during an exercise test, and was found to be both genotypically and phenotypically positive for BrS.

CASE REPORT

An 18-year-old previously healthy male presented to cardiology clinic after experiencing syncope while running laps. He experienced loss of consciousness for a few minutes but was successfully resuscitated with cardiopulmonary resuscitation. Initial workup including electrolytes and toxicology screen was normal. However, his ECG showed the typical coved-type ST-segment changes in V1 and V2 consistent with BrS [Figure 1]. Since the syncope was triggered by exercise, he underwent a treadmill stress test, during which he became near syncopal and his rhythm degenerated to a wide complex tachycardia consistent with ventricular tachycardia [Figure 2]. His symptoms self-resolved shortly after exercise was stopped. A cardiac magnetic resonance imaging did not demonstrate any pathologic findings.

Genetic testing was done and the patient was found to have a pathogenic disease-causing mutation in the SCN5A gene consistent with BrS. The c. 4140_4142delCAA variant that was identified is located in coding exon 22 of the SCN5A gene and has been associated with varying degrees of cardiac conduction disorders, as well as a family history of sudden death in several studies.[4-6] Additional testing including ECGs and

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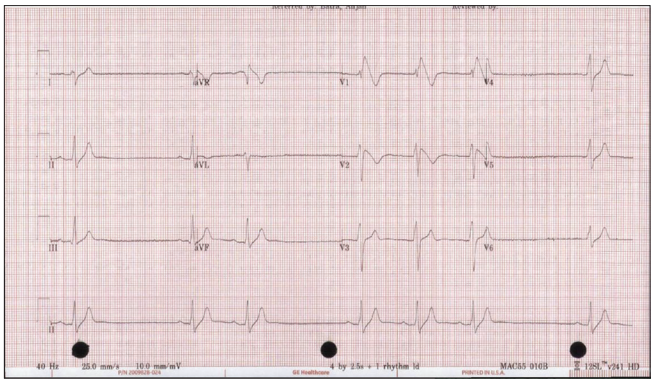

Figure 1: 12-lead ECG tracing showing the typical coved-type ST-segment elevation in the right precordial ECG leads, especially apparent in leads V1 and V2. This ECG is consistent with Type 1 Brugada ECG tracing, which is diagnostic for the disease. ECG: Electrocardiographic

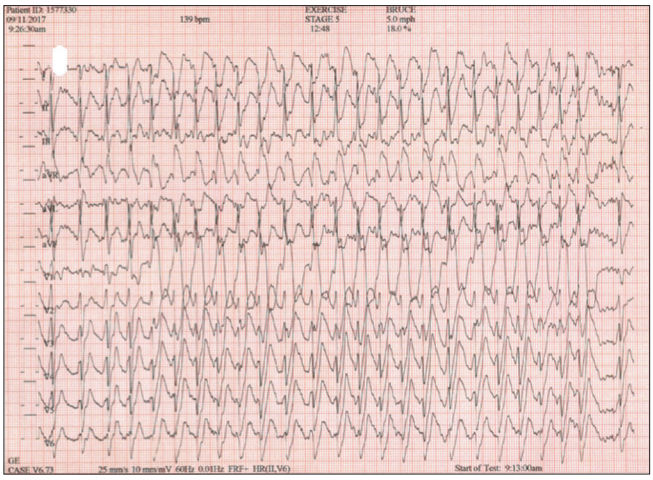

Figure 2: Wide complex tachycardia during Bruce protocol Stage 3 exercise testing

genetic testing for the same mutation in his brother and parents came back negative. The patient underwent an electrophysiology study, which showed easily inducible, hemodynamically unstable polymorphic ventricular tachycardia at 260 bpm, requiring DC cardioversion for conversion to sinus rhythm. The patient was started on nadolol 40 mg daily and a transvenous dual-chamber implantable cardiac defibrillator was implanted. The patient underwent a subsequent exercise test that showed no ventricular tachycardia on beta-blockers.

DISCUSSION

There are few data on the risk of exercise and the role of exercise stress testing in BrS. The symptoms of BrS traditionally occur at rest, often while the patient is asleep. It is hypothesized that this occurs as a consequence of increased vagal activity and withdrawal of sympathetic activity. It is atypical for patients with BrS to experience symptoms while exercising, and therefore, it is generally recommended that patients with BrS are not restricted from playing sports.[7,8] Exercise testing is also not generally a part of the diagnostic workup for BrS.

Our patient experienced significant cardiac symptomatology with exertion. Furthermore, treadmill stress testing was helpful in risk stratifying the patient. Although the literature on exercise testing in patients with BrS is limited, there are a few studies that have demonstrated a potential role for exercise testing in these patients. A review of the literature by Masrur *et al.* identified 166 BrS patients who underwent exercise testing and found that a majority (57%) of BrS patients developed ST-segment elevation while undergoing stress testing.[9] They found no reports of exercise-related sudden death, but there were four cases of syncope after exercise. They also found two reports of ventricular tachycardia and one report of multiple ventricular extrasystoles during exercise testing. Exercise unmasked a BrS ECG pattern in five patients. They concluded that exercise is associated with syncope and ST augmentation after exercise and may be helpful in unmasking BrS.[9] Makimoto *et al.* reported augmentation of ST-segment elevation during recovery from exercise testing was specific in patients with BrS and can be an independent predictor for cardiac events and poor prognosis, especially for patients with syncope alone and for asymptomatic patients.[10] Our case adds to the existing literature that an exercise stress test can be helpful in detecting ECG changes and life-threatening arrhythmias in a controlled environment. Furthermore, the observation that exercise can worsen the ST abnormalities in BrS and produce ventricular arrhythmias suggest that certain patients with BrS may need to be restricted from vigorous exercise. Beta-blockers may be beneficial in this subset of patients.

CONCLUSIONS

Exercise stress testing may have a potential role in the diagnosis and management of a patient with suspected BrS, particularly in patients who are symptomatic with physical activity. Although it is generally recommended that patients with BrS are not restricted from playing competitive sports, our case report suggests that it may be beneficial to risk stratify patients based on the results of exercise stress testing.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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