ECG in an Athlete With Syncope

Innocuous Incomplete Right Bundle Branch Block or Brugada Pattern?

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ABSTRACT

Abnormalities of an athlete’s electrocardiogram may be the result of an underlying heart disease and may carry a risk of sudden death. It is important that electrocardiographic abnormalities are correctly distinguished. We present the case of a young marathon athlete presenting with syncope and incomplete right bundle block pattern suggestive of a type 2 Brugada pattern. (Level of Difficulty: Advanced.) (J Am Coll Cardiol Case Rep 2021;3:1760–1763) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENT ILLNESS

A 22-year-old man was admitted for evaluation of 2 episodes of syncope. The patient experienced the first syncope episode during a casual walk on a footpath when he fell down and sustained an injury. The second episode of syncope occurred while he was standing and talking with his friends. There was an absence of febrile spike during both episodes. The patient denied a history of any drug consumption. The initial diagnosis was neurocardiogenic syncope on the basis of his history and symptoms. After this episode, he was thoroughly evaluated by a neurologist and underwent neuroimaging (magnetic resonance imaging [MRI] of the brain), the findings of which were normal. He gave a history of active participation in college sports and was running 20 to 25 km every day. He did not give any family history of sudden cardiac death. Clinical examination was unremarkable. His ECG on presentation and an ECG taken 1 intercostal space higher are shown in Figures 1 and 2, respectively.

LEARNING OBJECTIVES

- To recognize abnormalities in an athlete’s ECG that may carry a significant risk of sudden cardiac death and differentiate these findings from those of benign conditions.
- To use the Corrado index and Chevallier’s beta angle to differentiate between a Brugada pattern and a right bundle branch pattern.
- To use the flecainide challenge test to provoke a Brugada type 1 pattern to confirm or exclude the diagnosis.

PAST HISTORY

There was no significant history of past medical illness.
ECG FINDINGS AND DIFFERENTIAL DIAGNOSIS ON ECG

The ECG on presentation showed sinus bradycardia with heart rate of 50 beats/min. He had incomplete right bundle branch block (RBBB) in leads V1 to V2 with 1-mm (V2) to 2-mm (V1) J point-elevation and a down-sloping ST-segment depression. He had a saddle-shaped ST-segment coving pattern with 1-mm ST-segment elevation in lead V3. These changes were highly suggestive of a Brugada type 2 pattern. An ECG taken 1 intercostal space higher did not show significant changes. However, these changes can also be seen in athlete’s heart. To differentiate between Brugada syndrome (BrS) and athlete’s heart, the Corrado index and Chevallier’s beta (β) angle were calculated, and a flecainide challenge test with 200 mg was performed. There are specific criteria to differentiate between the ECG changes in RBBB seen in athlete’s heart and those found in BrS (1,2).

OTHER INVESTIGATIONS

The 2-dimensional echocardiogram was normal, without a gradient across the aortic valve or left ventricular outflow tract, and the MRI scan of the brain was also normal. Holter monitoring was performed to elicit evidence of any kind of arrhythmia or transient pattern conversion to type 1 BrS. The Holter monitoring results were within normal limits. An ECG was taken with chest leads positioned 1 to 2 places higher than usual, and no difference was noted.

MANAGEMENT

The patient was admitted and thoroughly evaluated, as stated earlier. Given the unavailability of intravenous ajmaline, procainamide, and flecainide, an oral flecainide challenge was performed. There was no transition of Brugada pattern from type 2 to type 1 (Figure 3), thus almost ruling out a Brugada origin (3). On the basis of the ECG criteria and the negative flecainide challenge test result, we performed an electrophysiological study. The corrected sinus node recovery time was within normal limits. Antegrade conduction was through the atrioventricular (AV) node and showed no dual AV node physiology. Retrograde conduction was concentric and decremental. No tachycardia was induced with right atrial pacing and ventricular pacing with atropine and isoproterenol. Long-short cycle length pacing did not induce any tachycardia, hence we concluded that it was hypervagotonia. There was no repeat similar episode during hospitalization or on follow-up, so no active treatment was given. The patient was advised to discontinue strenuous exercise and follow up regularly.

DISCUSSION

1. Corrado index: The ratio between the peak height of QRS-ST/peak of the ST-segment after 80 ms is >1 in BrS and <1 in athletes. This ratio was <1 in our patient. The ECG of healthy athletes depicts a fast r'-wave. The ST-segment is usually not elevated in lead V1 with a Corrado index that is ≤1 (1).
2. Chevallier’s beta (β) angle: This angle is formed by the ascending S and descending r' in RBBB seen in lead V1. It was <58° in our patient, thus suggesting athlete’s heart. It is normally >58° in patients with BrS. The criteria had a sensitivity of 79% and specificity of 84% (2).

3. The duration of the base triangle of r' at 5 mm from the high take-off is >3.5 mm in a type 2 Brugada pattern. This is much shorter in athletes with RBBB, as seen in our patient. The criteria has been found to have a sensitivity of 81% and a specificity of 82%.

Complete and incomplete RBBB occurs more commonly in endurance sports athletes without underlying structural heart disease. The reason is exercise-induced right ventricular remodeling causing increased cavity size and resultant increased conduction time.

Provocative testing with various agents has been suggested whenever there is suspicion of a Brugada pattern in the ECG (3). The sensitivity and specificity of the flecainide test in SCN5A mutation-positive probands and their families have been reported as 77% and 80%, respectively. Pharmacologic provocation should be performed only when the baseline ECG is not diagnostic of BrS, as in our patient.

Drug challenge should be performed under strict monitoring of blood pressure and a 12-lead ECG, and facilities for cardioversion and resuscitation should be available. Drug administration should be stopped if a type 1 pattern becomes apparent on the ECG, if the ventricular arrhythmias develop, or if the QRS complex widens to ≥130% of the baseline (3). The RBBB pattern and the bradycardia regressed once there was a pause in his athletic activity, as shown in Figure 4.

This benign diagnosis, in contrast to BrS, would alter
his prognosis considerably and enable him to continue his sports activities.

FOLLOW-UP

After regular follow-up, there was no similar episode, and an ECG showed regression of the RBBB pattern.

CONCLUSIONS

On the basis of the ECG criteria and the negative result of the flecainide challenge test, it was determined that the patient’s ECG changes were the result of athlete’s heart, and his syncope was probably caused by hypervagotonia.

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KEY WORDS awareness, electrocardiogram, electrophysiology, exercise