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Electrocardiogram quiz

Two 12-lead electrocardiograms (ECG) were recorded on an asymptomatic 66-year-old white male bicyclist with a history of paroxysmal nocturnal atrial fibrillation treated with flecainide. The second ECG (Fig. 1B) was performed with precordial leads placed one intercostal space above the standard ECG (Fig. 1A). What is your diagnosis?

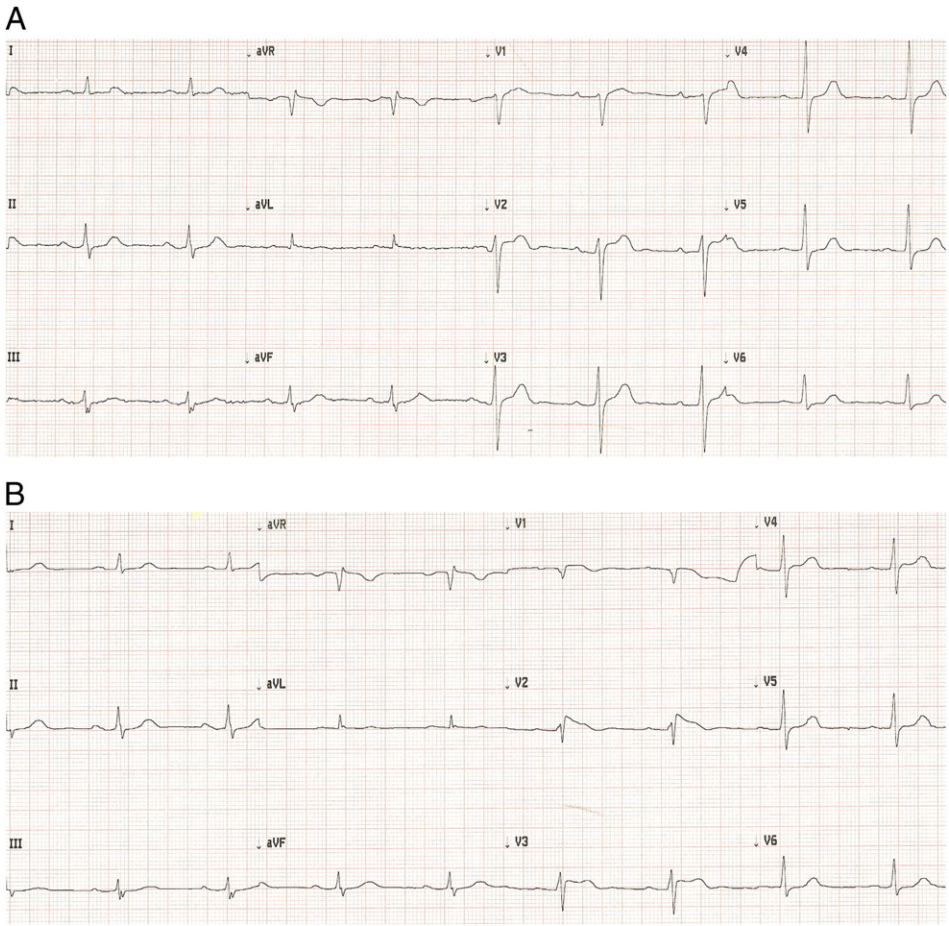

Fig. 1. A, 12-Lead ECG with standard lead placement. B, 12-Lead ECG performed with precordial leads one intercostal space above compared to the standard lead placement.

The ECG suggests

1. Acute pulmonary embolism (PE) with right ventricular (RV) strain
2. Right ventricular infarction
3. Arrhythmogenic RV dysplasia/cardiomyopathy (ARVD/C)
4. A channelopathy

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Electrocardiogram quiz

Answers:

1. Acute PE with RV strain

The presence of right bundle-branch block, T-wave inversions in lead V_1-V_3, and a history of atrial fibrillation could suggest a diagnosis of acute PE. However, there is absence of sinus tachycardia, S1Q3T3 pattern, or right axis deviation. Although T-wave inversions have the greatest accuracy for RV dysfunction and adverse outcomes in PE, in our patient, they appear only with non-standard lead placement. Presence of QR in V1 is an ECG sign associated with RV strain, but our patient has a QS pattern, which makes PE an unlikely diagnosis.

2. Right ventricular infarction

Right ventricular infarction is usually suspected in the setting of inferior wall myocardial infarction. Our patient did not have any evidence of ST elevation in the inferior leads. Presence of ST-segment elevation in the right-sided chest leads (V_1 in our patient) is a relatively sensitive and specific sign of right ventricular infarction; however, he is asymptomatic. Occasional appearance of ST elevation in V_2-V_3 may also occur, especially when the injury to the inferior wall is minimal. However, presence of ≥1-mm ST-segment elevation in one or more of the right sided leads (V_4R, V_5R, V_6R) in the setting of acute myocardial infarction is required for a diagnosis of acute RV myocardial infarction that is absent in our patient. In addition, our patient is an athlete, and the minimal ST segment elevation was presumed to represent early repolarization.

3. Arrhythmogenic RV dysplasia/cardiomyopathy

Electrocardiographic diagnosis of ARVD/C relies on activation delay (ε waves, localized prolongation of QRS greater than 110 milliseconds in V_1-V_3, and prolonged terminal activation duration ≥55 milliseconds in V_1-V_3) and repolarization abnormalities (T-wave inversions in V_1-V_3) during sinus rhythm. Although our patient has T-wave inversions in V_1-V_3, other electrocardiographic findings consistent with a diagnosis of ARVD/C are absent. Also a definitive diagnosis of ARVD requires set task force criteria obtained from imaging, ECG, histologic evaluation, and positive family history and history of arrhythmias.

4. Channelopathy

Brugada syndrome is a genetic channelopathy that is linked to mutations in *SCN5A*, the gene that encodes the α-subunit of the sodium channel. This gives rise to characteristic ECG manifestations that are often dynamic or concealed. The ECG findings of Brugada may be unmasked by several factors including administration of sodium channel blockers. Placement of the right precordial leads in a superior position (up to the second intercostal space above normal) can increase the sensitivity of the ECG for detecting the Brugada phenotype in some patients, both in the presence or absence of a drug challenge [1,2].

Electrocardiogram with standard lead placement in our patient revealed J-point elevation in V_1 and horizontal ST elevation in V_2. An ECG repeated with leads placed one intercostal space above revealed coved ST elevation in V_2 with ST-segment elevation in V_1 and V_3 also noted. This is consistent with type 1 Brugada syndrome. Flecainide was stopped. A repeat ECG, performed one week later with conventional (Fig. 2) and unconventional lead placement, showed resolution of the ST elevation. Thus, unmasking of the Brugada-type ECG was most likely secondary to sodium channel blockade due to flecainide and by the nonstandard precordial lead placement.

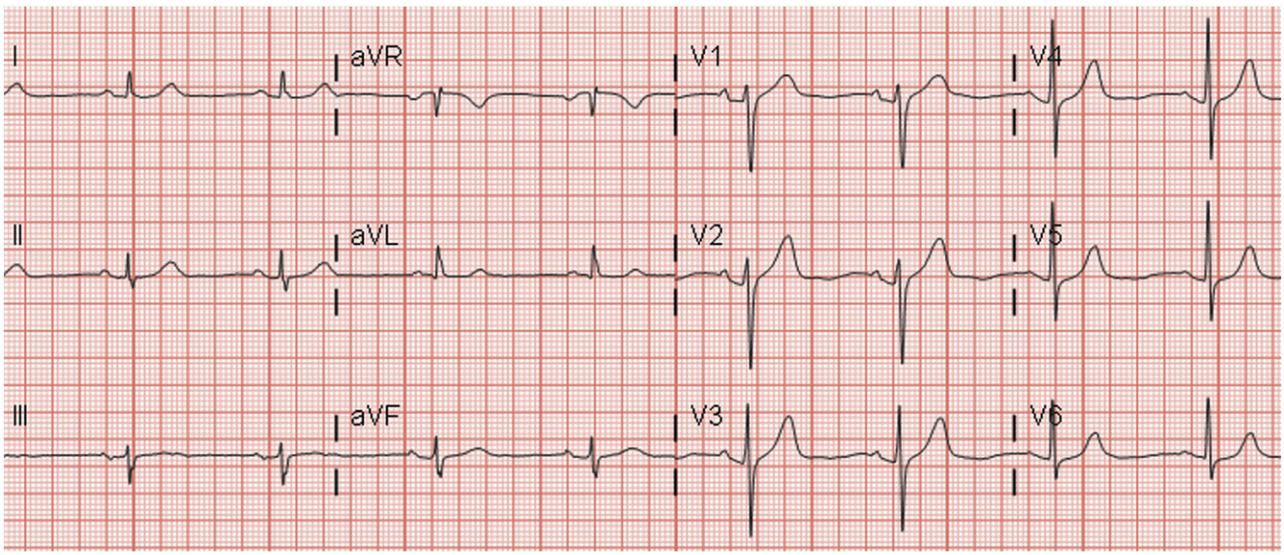

Fig. 2. 12-Lead ECG performed with standard lead placement one week after flecainide was discontinued.

References

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2. Miyamoto K, et al. Diagnostic and prognostic value of a type 1 Brugada electrocardiogram at higher (third or second) V_1 to V_2 recording in men with Brugada syndrome. Am J Cardiol 2007;99:53.