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Focus on ECGs: Case #24

Routine Outpatient Electrocardiogram: What Is the Diagnosis?

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A 52-year-old woman with a 5-year history of hypertrophic cardiomyopathy (HCM) underwent routine follow-up evaluation, during which a 12-lead electrocardiogram (ECG) was obtained (Fig. 1).

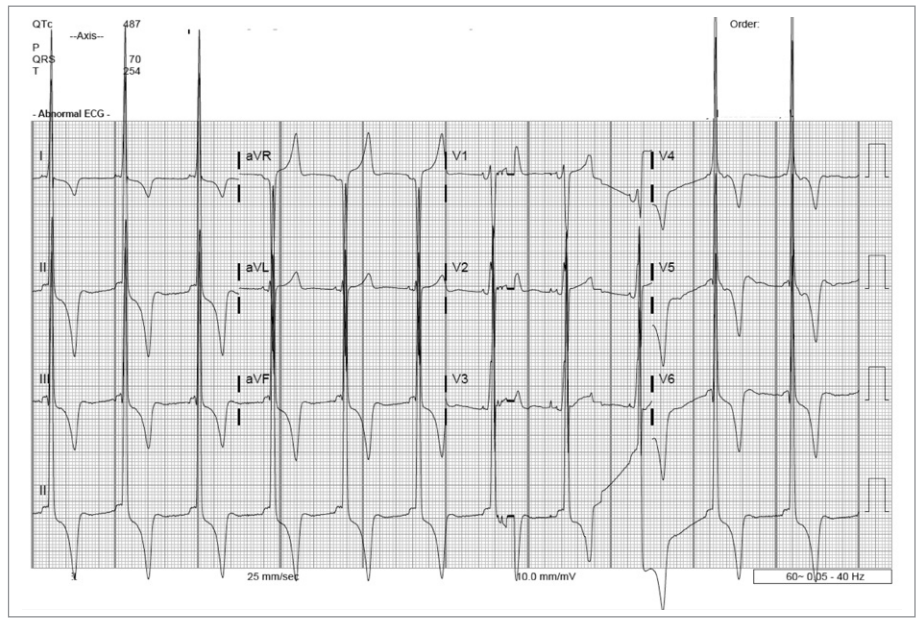

Fig. 1

What is the diagnosis based on the ECG?

A) Wolff-Parkinson-White syndrome
B) Hypertrophic cardiomyopathy
C) Left bundle branch block
D) A and B

See next page for the answer.

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***D)* A and B**

The patient's 12-lead ECG clearly shows sinus rhythm with a short PR interval and prominent delta waves, consistent with a Wolff-Parkinson-White (WPW) pattern (Fig. 1). The tall QRS complexes meet the diagnostic criteria for left ventricular hypertrophy (LVH); the ST-segment depression with deep T-wave inversion in the inferolateral leads suggests LVH with repolarization changes.

Repolarization changes secondary to a WPW pattern can cause ST-T changes. ST depression with giant T-wave inversion in the inferolateral leads is thought to be typical of the apical variant of HCM[1]; however, this pattern is not specific to apical HCM. Many patients with ST depression and giant T-wave inversion in the inferolateral leads do not meet cardiac magnetic resonance–based criteria for having apical HCM.[1] Indeed, transthoracic echocardiograms (apical 4-chamber view) obtained at end-diastole (Fig. 2A) and end-systole (Fig. 2B) during the patient's visit showed severe concentric LVH with an LV ejection fraction >70% and no signs of systolic anterior motion of the mitral valve or LV outflow tract obstruction. No apical hypertrophy was noted.

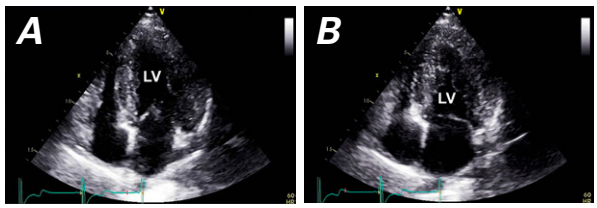

Fig. 2

Few case studies have focused on patients with HCM and WPW syndrome,[2] and the relationship between the 2 disorders remains poorly understood. Some researchers have proposed a genetic association.[3-6] Danon disease, a cardioskeletal myopathy syndrome that sometimes presents with HCM, may be associated with WPW syndrome. Other inherited conditions involving mitochondrial gene mutations, protein kinase gene mutations, and glycogen storage diseases, such as those caused by *PRKAG2* gene mutations, have also been investigated for their potential to link HCM and WPW.

On the basis of her ECG and 2011 American College of Cardiology Foundation/American Heart Association guidelines for patients with HCM,[7] this patient is not eligible for implantable cardioverter-defibrillator (ICD) therapy to prevent sudden cardiac death. Atrial fibrillation (AF) is one of the most common arrhythmias in patients with WPW syndrome.[8] In the presence of an accessory pathway, AF increases the risk of death because it can induce an extremely fast ventricular response that deteriorates to ventricular fibrillation.[2] Atrial fibrillation is also the most common arrhythmia in patients with HCM, and it is poorly tolerated. Therefore, patients with both HCM and WPW are at increased risk of morbidities associated with AF and a rapid ventricular response.[2] The effectiveness of prophylactic ablation for asymptomatic patients with WPW is not clear.[9] In one retrospective study of 40 patients with WPW and HCM, eliminating the accessory pathway was associated with a decrease in the AF recurrence rate, but not with changes in rates of death, ICD intervention, or cardiac transplant.[2] Further testing and risk stratification—by means of either electrophysiologic studies or long-term rhythm analysis (with an event recorder or implantable loop recorder)—may be warranted in applicable patients who have a low threshold for ablation of the accessory pathway or AF.

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