Circulation

ECG CHALLENGE

Ventricular Tachycardia and Electrocardiographic ST-Segment–Elevation Myocardial Infarction Without Coronary Artery Disease

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ECG CHALLENGE

A 66-year-old man with a medical history of hypertension and hyperlipidemia presented to an urgent care center with sudden-onset severe midsternal chest tightness and associated palpitation, diaphoresis, and shortness of breath. The patient had similar episodes in the past that resolved spontaneously. The ECG showed wide-complex tachycardia with a rate in the low 200s as shown in Figure 1A. Given the patient's stable blood pressure, he was started on procainamide therapy with an initial 100 mg intravenous bolus followed by an infusion. Within minutes, the patient converted to sinus rhythm with resolution of his symptoms. Repeat ECG, however, was now concerning for lateral ST-segment–elevation myocardial infarction (Figure 1B).

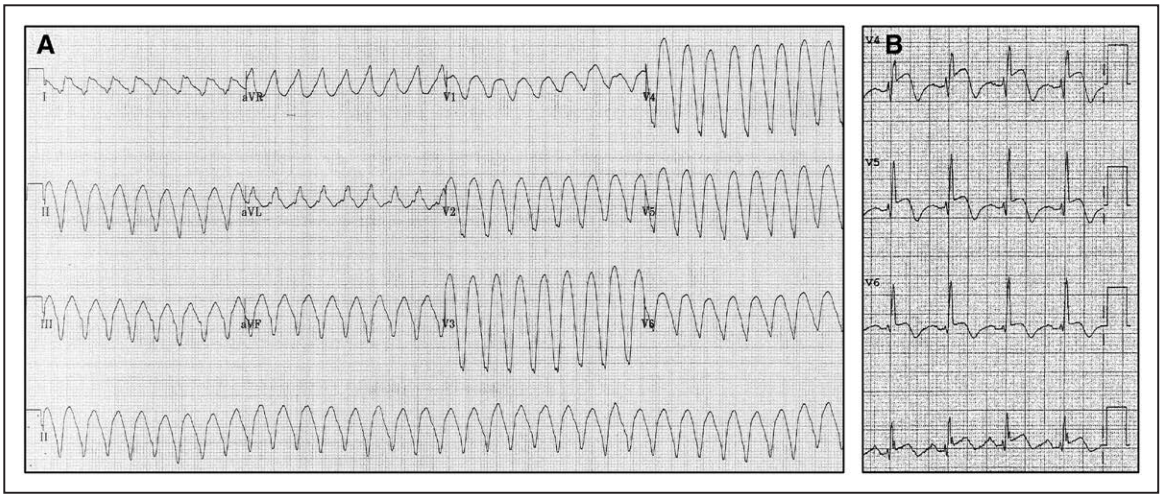

Figure 1. ECGs of a patient who presented with chest pain, palpitation, and lightheadedness.
A, Wide-complex tachycardia on presentation at a rate of 202/min. **B,** Immediately after termination of the tachycardia, there is prominent ST-segment elevation in the lateral chest leads.

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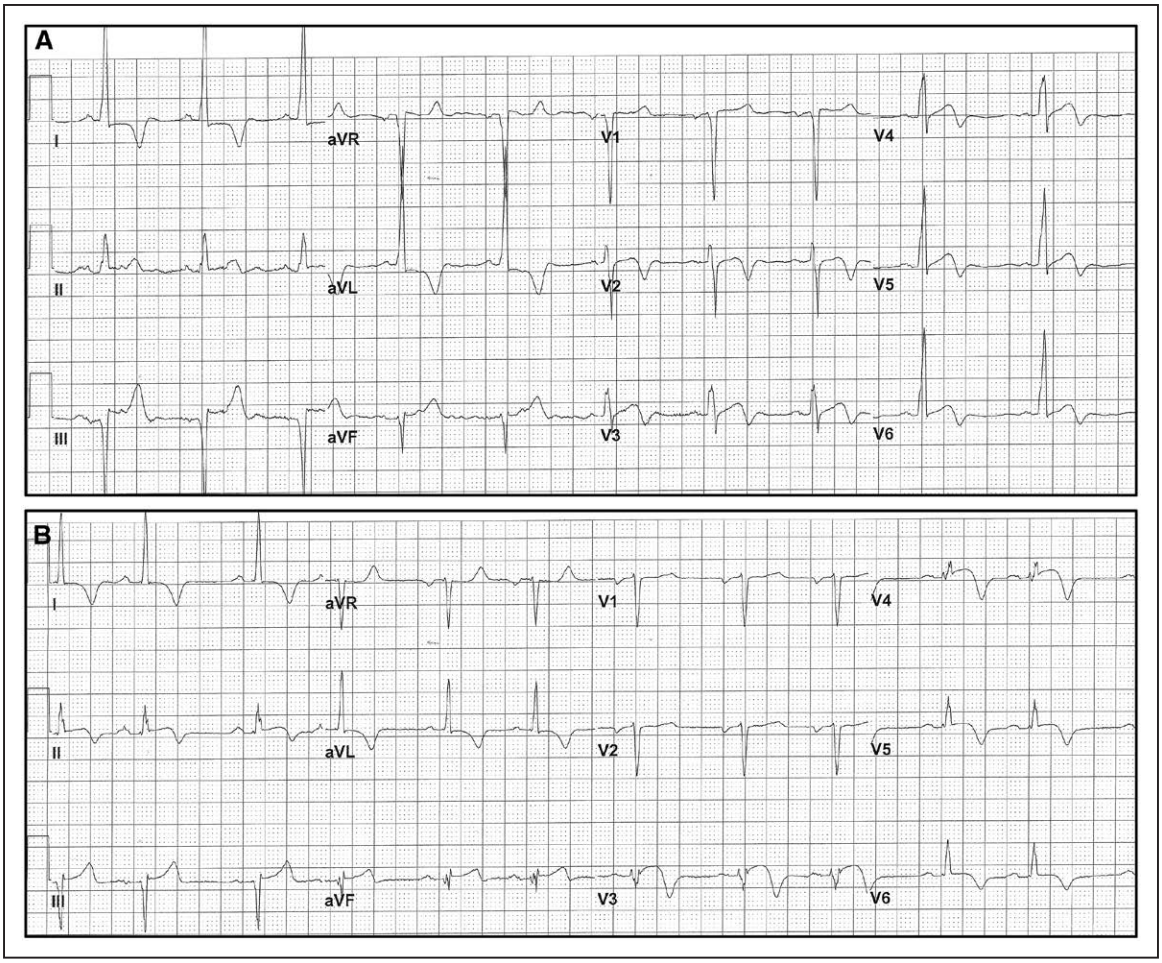

Figure 2. Twelve-lead ECGs of the patient 7 years apart.
A and **B,** Both ECGs show left ventricular hypertrophy and ST-segment elevation in the lateral chest leads.

Note the 2- to 5-mm upward convex ST elevation followed by negative T waves in leads V4, V5, and V6. Aspirin, heparin, and atorvastatin were initiated, and the patient was transferred to a higher level of care for emergent cardiac catheterization. There the troponin level was negative, and cardiac catheterization did not show any coronary artery disease. Subsequently, it turned out that this was the patient's third negative cardiac catheterization in 7 years, with each ECG presentation concerning for ST-segment–elevation myocardial infarction, as shown in Figure 2A and 2B. Unfortunately, this information was not available before proceeding with the cardiac catheterization. What other diagnosis would explain the patient's presentation and the ECG changes?

RESPONSE TO ECG CHALLENGE

The presented ECGs provided a strong clue toward a likely clinical diagnosis. Persistent ST elevation in the lateral chest leads spanning several years suggested the presence of an apical aneurysm.[1,2] Apical aneurysm in the absence of a history of myocardial infarction or abnormal Q waves is a finding typical of hypertrophic cardiomyopathy (HCM) with midventricular obstruction.[1,2] In HCM, patients exhibiting ST-segment elevation in V4 to V6 have been found to be significantly more likely to have midventricular cavity obliteration with apical aneurysm compared with those without ST-segment elevation (93% versus 7%, $P<0.001$).[1,2] The morphology of the wide-complex tachycardia on presentation was consistent with ventricular tachycardia (VT) arising from the left ventricular apex: the very fast rate was probably related to the short reentrant circuit around the apical aneurysm, and the negative QRS complexes in all leads except for the relatively high leads of aVL and aVR indicated an apex-to-base ventricular activation sequence (Figure 1A). Echocardiogram revealed severe concentric left ventricular hypertrophy with midventricular predominance and apical akinesis. Cardiovascular magnetic resonance imaging confirmed the diagnosis of HCM with midventricular cavity obliteration and apical aneurysm (Figure 3A and 3B).

Patients with HCM with apical aneurysms represent an uncommon (2.2%; 95% confidence interval,

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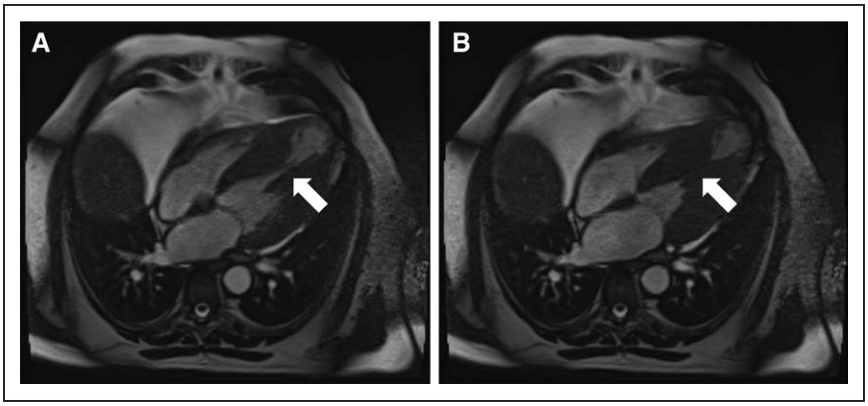

Figure 3. Cardiac magnetic resonance imaging of the heart. **A,** Diastole. **B,** Systole. Arrows point to midventricular cavity obliteration.

1.4–3.1) but important subgroup, with ≤68% of the aneurysms caused by a midventricular obstruction.[1] Cardiovascular magnetic resonance imaging can best identify the presence of an apical aneurysm as well as scarring. These patients are at high risk for VT, apical thrombus formation, thromboembolic events, and sudden cardiac death, with a yearly adverse event rate of 10.5%.[1] In contrast, the prognosis of patients with apical HCM is much more benign. Apical aneurysm is usually associated with transmural scarring, an arrhythmogenic substrate for malignant VT. In 1 study, as many as 42% of patients with apical aneurysms demonstrated episodes of nonsustained VT, a known determinant of increased risk for sudden cardiac death in HCM.[1]

Calcium channel blockers and β-blockers may be effective in the reduction of symptoms; however, case reports have exhibited recurrent VT resistant to medical therapy.[3] In refractory cases, ablative therapy of the scar tissue or apical aneurysmectomy may be indicated.[3] Our patient underwent dual-chamber implantable cardioverter-defibrillator placement for secondary prevention and was discharged on metoprolol and amiodarone. Because of the severe midventricular cavity obliteration, catheter ablation was planned only if he had breakthrough arrhythmias. He has done well on medical therapy; to date, no additional episodes of VT or implantable cardioverter-defibrillator discharges have occurred.

In summary, ST-elevation is not always indicative of acute myocardial infarction. Although it is the first diagnosis to be considered, after multiple negative catheterizations, alternative diagnoses must be entertained. Echocardiogram and cardiovascular magnetic resonance imaging can help identify other important causes such as midventricular HCM with apical aneurysm. Once the correct diagnosis has been established, these patients need to be educated about their disease, and a laminated copy of their ECGs should be provided. Such simple interventions can help avoid unnecessary subsequent cardiac catheterizations.

ARTICLE INFORMATION

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Disclosures

None.

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