CASE REPORT

Mid-Ventricular Obstructive Hypertrophic Cardiomyopathy and Apical Aneurysm Mimicking Acute ST-Elevation Myocardial Infarction

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Nonischemic ST-segment elevation may be confused as acute ST-elevation myocardial infarction (STEMI), especially in patients with atypical presenting symptoms. Among the possible differential diagnosis, hypertrophic cardiomyopathy (HCM) should be considered. Mid-ventricular obstructive hypertrophic cardiomyopathy (MVOHCM) is a rare type of cardiomyopathy, accounting for approximately 5% of all HCM cases. ST-segment elevation on electrocardiogram (ECG) in patients with MVOHCM is a rare clinical presentation. We present a case of MVOHCM and apical aneurysm mimicking acute STEMI

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ST-segment elevation on electrocardiogram (ECG) is considered to reflect acute transmural ischemia due to acute epicardial coronary artery occlusion by thrombosis formation and commonly used to diagnose acute ST-elevation myocardial infarction (STEMI). Nonischemic ST-segment elevation on ECG may be confused as STEMI, especially in patients with atypical presenting symptoms. Among the possible differential diagnosis, hypertrophic cardiomyopathy (HCM) should be considered.¹ Mid-ventricular obstructive hypertrophic cardiomyopathy (MVOHCM) is a rare type of cardiomyopathy, accounting for approximately 5% of all HCM cases.² ST-segment elevation on ECG in patients with MVOHCM is a rare clinical presentation. We present a case of MVOHCM and apical aneurysm mimicking acute STEMI.

A 66-year-old female was admitted with suspected acute coronary syndrome, primarily because of pronounced ST-segment elevations in the inferior and precordial leads. She has experienced shortness of breath and chest distress for several

years, the day before admission she felt aggravated chest discomfort. She had a history of cerebral infarction for 20 years. She had no history of diabetes, hypertension, or family members with sudden cardiac death and denied alcoholism or drug abuse.

The patient's initial vital signs included heart rate 74 bpm, blood pressure 140/80 mmHg, temperature 97.7 °F (36.5 °C), and oxygen saturation 99% on room air. Cardiac auscultation revealed a normal S1 and S2 and a grade 2/6 systolic murmur at the apex. The patient's lung and abdominal examination was unremarkable. Electrocardiogram at admission revealed normal sinus rhythm and left ventricular hypertrophy by voltage criteria. There was convex ST-segment elevation in I, II, III, aVF and V₂–V₆ leads followed by T-wave inversion (dome shaped), and abnormal Q waves were not observed (Fig. 1). No previous ECGs were available for comparison. Emergent transthoracic echocardiogram showed a significant asymmetric hypertrophy of mid-left ventricle. The maximal

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Figure 1. The presenting ECG showed sinus rhythm of 74 bpm, QRS amplitude criteria for hypertrophy of the left ventricle, ST segment elevated in leads II,III, aVF, V₃–V₆ (≥ 1 mm) and ST depression in leads I, aVL. (This ECG was recorded on paper at 25 mm/s with a standardization of 10 mm = 1 mV).

wall thickness of left ventricle was 22.6 mm and the hypertrophy was localized to the left anterior-medial septum, there was a high early systolic peak flow velocity greater than 3.77 m/s in mid-left ventricle with a high intraventricular pressure gradient 57 mm Hg across the obstruction. There were normal pressure gradient and flow velocity across the left ventricular outflow tract. Although the global left ventricular ejection fraction was not markedly impaired (left ventricular ejection fraction = 60%), aneurysm formation was observed at the left ventricular apex (26.4 × 27.2 mm). (Fig. 2, Video 1). Laboratory tests yielded a normal platelet count and coagulation profile. The cardiac troponin I was 0.048 ng/mL at admission and

0.047 ng/mL 12 hours later (normal <0.02 ng/mL) and B-type natriuretic peptide was 508 pg/mL (normal <200 pg/mL).

Based on the patient's ECG and echocardiogram findings, the ST-segment elevation was considered related to MVOHCM-associated apical aneurysm. The patient was not transferred directly to the cardiac catheterization and instead was treated with optimal medical therapy (aspirin, β -blocker). She was followed as an outpatient for 6 months and remained clinically stable. Her follow-up ECG at 6-month is similar to the presenting ECG.

HCM is a genetic cardiac disease characterized by asymmetrical septal hypertrophy. Approximately 5% of HCM patients present

Figure 2. Transthoracic echocardiography. (A) Parasternal long-axis view showing asymmetric septal hypertrophic cardiomyopathy in end-diastole (mid-wall thickness at end-diastolic phase 22.6 mm). (B) The four-chamber view showing a combination of basal and mid-ventricular hypertrophic cardiomyopathy (white arrow) and an apical aneurysm (*). (C) Continuous-wave Doppler echocardiogram showed early systolic peak flow velocity to be greater than 3.77 m/s. LA = left atrium; RA = right atrium; RV = right ventricle; LV = left ventricle, Ao = aorta.

with mid-ventricular obstruction. Several studies have shown the potential association between mid-ventricular HCM and apical aneurysm formation.^{3–5} Maron et al. showed that the prevalence of left ventricular apical aneurysm (LVAA) in all non-Japanese HCM case was 2.2%.⁶ MVOHCM accompanied by apical aneurysm may increase the risk of lethal arrhythmia, thromboembolic stroke, progressive heart failure, even sudden cardiac death.^{7–10} However, de Gregorio et al. recognized it was a benign remodeling trend because average progression rate of the apical pouch was 3.9% per year during 14-year echocardiogram follow-up.¹¹

Echocardiogram is an important noninvasive tool to identify and perform risk stratification of the patients with HCM. In the present case, the echocardiogram reveals the hypertrophy in the mid portion of the left ventricle and formation of apical aneurysm. Continuous-wave Doppler shows high flow velocity with high pressure gradient cross the mid-left ventricle (Video 1). Although the pathogenesis of apical aneurysm in the setting of MVOHCM is not fully understood, the pressure gradient between the apex and the mid-ventricular may be one important reason. In the present case, the pressure gradient was close to 60 mmHg. The pressure overload on the apical wall, relatively narrowing of the small intramyocardial coronary arteries, decreased coronary perfusion pressure, oxygen supply/demand mismatch, and decreased capillary myocardial fiber ratio due to mid-ventricular obstruction might eventually lead to myocardial dysfunction and apical aneurysm formation.^{12, 13}

Most commonly ECG abnormalities of MVOHCM included abnormal Q waves, ST-segment depressions with T-wave inversions and left ventricular hypertrophy by voltage criteria. In contrast, ST-segment elevation is a rare finding in patients with MVOHCM. Ichida et al.¹⁴ showed that the ST-segment elevation in V₃–V₅ on ECG is a useful marker for the early detection of LVAA formation in hypertrophic cardiomyopathy patients, the sensitivity was 66.7% (14/21), and the specificity was 98.7% (223/226).

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