Chest Discomfort, Abnormal Electrocardiogram, Clean Coronaries

IMAGE OF THE MONTH

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CASE PRESENTATION

A 49-year-old Caucasian male presented with exertional chest discomfort radiating to his left arm (relieved by nitroglycerin), accompanied by shortness of breath and dizziness. He denied palpitations, nausea, diaphoresis, or syncope. Past medical history was remarkable for untreated hypertension. He was on no medications. Surgical history was unremarkable. His father had his first myocardial infarction (MI) before age 60. There was no further family history of heart disease or sudden cardiac death. Social history revealed a nonsmoker, with rare alcohol and no illegal drug use.

PHYSICAL ASSESSMENT

The patient's vital signs were as follows: blood pressure 116/53 mm Hg; pulse 64 beats/min; respiration 16 breaths/min; oxygen saturation 98% on room air; temperature 36.6°C; body mass index 33.1. He was not in acute distress. Head, neck, and neurologic exams were unremarkable. Lungs were clear to auscultation bilaterally with nonlabored respirations. Cardiac exam revealed normal rate and rhythm, no murmur or rub, mild S4, and no jugular venous distention. His abdomen was nontender, without masses, bruits, or hepatosplenomegaly. His extremities revealed normal pulses and no peripheral edema or varicosities.

DIAGNOSTICS

Labs and Radiology

Comprehensive metabolic panel, complete blood count, thyroid-stimulating hormone, erythrocyte sedimentation rate, C-reactive protein, D-dimer, and chest X-ray were normal. Troponins were indeterminate. Brain natriuretic peptide was mildly elevated. Triglycerides, total cholesterol, and low-density lipoprotein were elevated. High-density lipoprotein was low.

Electrocardiography

An electrocardiogram (ECG) was ordered to evaluate for myocardial infarction or ischemia. The ECG revealed a sinus rhythm rate of 79 beats/min, marked anterolateral ST- and T-wave changes, possible ischemia or subendocardial injury, and left ventricular (LV) hypertrophy.

Cardiac Catheterization

Cardiac catheterization was done because of acute ECG changes, indeterminate troponins, chest discomfort, and family history. The coronary arteries revealed no hemodynamically significant disease. LV end-diastolic pressure was severely elevated and the apex revealed severe hypertrophy with normal anterior and inferior wall motion.

Echocardiography

A transthoracic echocardiogram was completed to evaluate cardiac anatomic structure and function. It revealed a hyperdynamic LV with an ejection fraction of 75%, normal LV diastolic function, mild basilar and midconcentric LV

hypertrophy, LV apical hypertrophy with decreased apical cavity size, and no significant valvular disease.

ETIOLOGY OF APICAL HYPERTROPHIC CARDIOMYOPATHY

Apical hypertrophic cardiomyopathy (AHCM) is a form of hypertrophic cardiomyopathy (HCM) primarily affecting the LV apex (pure type), but it may also affect the interventricular septum (mixed type). Unlike other variants of HCM, the LV outflow tract is unobstructed. In approximately half of HCM patients, the etiology is an autosomal protein mutation of the sarcomere. This results in myocardial fiber disarray and interstitial fibrosis leading to apical hypertrophy in the pure form, and apical and septal hypertrophy in the mixed form.[1-3] Pathophysiology is thought to be due to "a mismatch of supply and demand secondary to small vessel coronary artery disease, delayed relaxation of the myocardium, decreased capillary-to-myocardial-fiber ratio, and decreased coronary perfusion pressure."[4] It affects 18% of Japanese patients with hypertrophic cardiomyopathy and only 3%-10% in the rest of the world, and it is less common in North America.[5]

DIAGNOSIS

Clinical Presentation

Common presentations include exertional chest discomfort, dyspnea, palpitations, atrial arrhythmias, syncope, and an audible S4. Sudden cardiac death and MI are found less often in this variant. Differential diagnoses include coronary artery disease, LV tumors or thrombus, endomyocardial fibrosis,[6] and noncompaction of the LV myocardium. Family history should be reviewed for sudden cardiac death, MI, stroke, and atrial or ventricular arrhythmias.

The ECG often resembles anterolateral ischemia due to giant negative T waves (>10 mm) in V3-V4, and ST-segment depression (≥ 1 mm) in V3-V6.[7] LV hypertrophy

Figure 1. Top arrow points to very tall R wave, indicative of left ventricular hypertrophy. Bottom arrow points to giant negative T waves > 10 mm in size.