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Hypertrophic cardiomyopathy or hypertensive heart disease?

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A 49-year-old male patient, with a history of typical angina, poorly controlled arterial hypertension, hypercholesterolemia and smoking, presented to a regional hospital because of chronic coronary syndrome exacerbation. Coronary angiography revealed a multivessel coronary artery disease with in-stent restenosis in the left circumflex artery and chronic total occlusion of the left anterior descending artery (panels B and C). Based on echocardiographic findings, hypertrophic cardiomyopathy (HCM) was suspected, and the patient was disqualified from coronary artery bypass grafting (CABG). The patient was referred to our tertiary health care center for further management, including high-risk percutaneous coronary interventions.

The stent in left circumflex artery was deployed in 2014. However, angina symptoms reappeared two years later, and the patient was treated pharmacologically for nearly four years in the outpatient setting without any further diagnostics.

On admission to our Department of Cardiology, the patient had no chest pain, and ECG recording showed negative T waves in I, aVL, and V3–V6, ST depression in I and aVL, and ST elevation with q waves in III, aVF, and V1–V2 leads (panel A). These changes were observed previously and initially interpreted as evidence of a previous myocardial infarction(s) without evidence of cardiac hypertrophy. The echocardiography performed at admission showed the maximum interventricular septum (IVS) thickness of 22 mm and the maximum left ventricle (LV) free wall thickness of 12 mm, without evidence of LV outflow tract obstruction,

with E/E' 13, with global longitudinal strain –9.2%, and without impairment of LV segmental contractility. However, the years-long history of poorly controlled arterial hypertension (on six antihypertensive drugs) raised doubts as to the suspicion of HCM. And the differential diagnostics of these two conditions based solely on echocardiographic imaging can be confusing [1]. Therefore, cardiac magnetic resonance (CMR) was performed to provide high-quality information that can reinforce HCM diagnosis [2]. CMR revealed an asymmetric LV hypertrophy with the maximum IVS thickness of 24 mm and the maximum LV free wall thickness of 16 mm. Additionally, we observed the presence of a mid-wall late gadolinium enhancement (LGE) in the hypertrophied mid and basal segments of the interventricular septum, i.e., a typical non-ischemic pattern of fibrosis in the course of HCM. CMR also showed an ischemic subendocardial LGE in the lateral and inferolateral walls, a characteristic for a past myocardial infarction (panels D and E).

The patient reported recurring chest pain at rest during hospitalization that required nitroglycerin administration (without any new ECG changes or troponin elevation). In consequence, we changed the initial diagnosis of chronic coronary syndrome to unstable angina. Considering the advancement of coronary lesions, the patient's case was again consulted on the Heart Team meeting, and cardiac surgeons qualified the patient for an urgent CABG.

An uneventful CABG was performed during the same hospital stay. The patient has successfully completed the rehabilitation and is now free of angina symptoms. Due to the low sudden cardiac death risk, the patient was not qualified for an implantable cardioverter-defibrillator [3, 4]. The patient is under constant care supervised by an HCM expert in the Outpatient Clinic at our Hospital.

The discharge diagnoses were unstable angina, previous myocardial infarction, low-risk HCM without left ventricular outflow tract obstruction, and poorly controlled hypertension.

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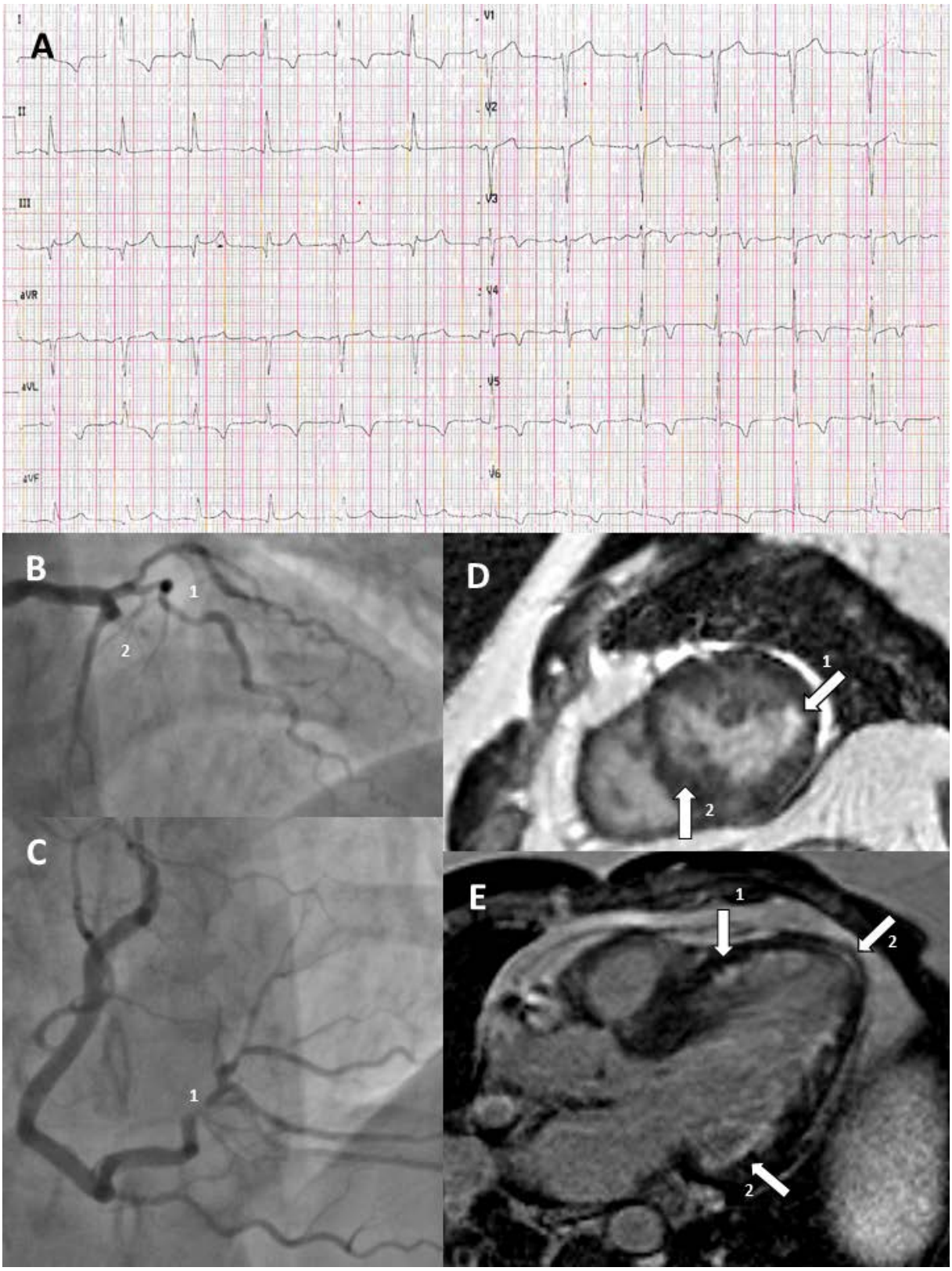

Figure 1. A. ECG reveals likely evidence of previous myocardial infarctions of the lateral and inferior walls without evidence of cardiac hypertrophy. **B.** Coronary angiography shows multiple atherosclerotic lesions with proximal occlusions of the left anterior descending artery (1) and the left circumflex artery (2, in-stent restenosis). **C.** Coronary angiography shows non-

obstructive lesions in the right coronary artery with a critical stenosis in the posterior descending artery (1). **D.** Cardiac magnetic resonance late gadolinium enhancement image, short-axis view, shows a subendocardial enhancement of the inferolateral wall characteristic for a previous myocardial infarction (1) and a non-ischemic mid-wall late gadolinium enhancement typical of fibrosis in the course of hypertrophic cardiomyopathy (2). **E.** Cardiac magnetic resonance late gadolinium enhancement image, three-chamber view, shows a non-ischemic mid-wall late gadolinium enhancement typical of fibrosis in the course of hypertrophic cardiomyopathy (1) and ischemic subendocardial late gadolinium enhancement typical of a previous myocardial infarction (2); asymmetric mid septal hypertrophy with a maximum wall thickness of 24 mm can be observed