CLINICAL VIGNETTE

Left ventricular aneurysm in hypertrophic cardiomyopathy with midventricular obstruction

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We report a case of a patient with left ventricular apical aneurysm (LVAA), caused by midventricular hypertrophic cardiomyopathy (HCM). A 64-year-old man was admitted to the hospital with a suspicion of postinfarction LVAA. The patient had transient ischemic attack 2 months before the admission. Cerebral magnetic resonance imaging revealed multiple cerebral infarctions, probably of embolic origin. During examination aimed to detect the cardiac source of embolism, LVAA was revealed. Electrocardiography demonstrated signs of left ventricular (LV) hypertrophy with strain. Echocardiography (Philips XI XP, Philips Healthcare, Eindhoven, The Netherlands) showed normal LV systolic function, hypertrophy of the midventricular segments, and near-complete obliteration of the LV chamber during systole (hourglass configuration; FIGURE 1A; Supplementary material, *Video S1*). Continuous-wave Doppler echocardiography showed an apical-to-basal gradient of 36 mm Hg in the LV cavity in systole and early diastole (FIGURE 1B). We found an apical outpouching in the LV, which was connected with the LV apical chamber through a narrow neck. This was consistent with the diagnosis of LVAA due to midventricular HCM (FIGURE 1C; Supplementary material, *Video S2* and *S3*). Coronary angiography did not show any lesions in the coronary arteries. Cardiac computed tomography (Toshiba Aquilion 128 TSX-101A, Toshiba Medical System, Ōtawara, Tochigi, Japan) demonstrated midventricular hypertrophy with obstruction of flow in systole and LVAA reminiscent of a 3-leaf clover in shape, without clots inside (FIGURE 1D and 1E). Aneurysmectomy was performed because of high risk of cerebral embolic stroke due to thrombus formation in the LVAA. Histological examination of the obtained specimens showed hypertrophied cardiomyocytes with signs of hibernation and necrosis in the aneurysmal wall, which confirmed the diagnosis of HCM. The patient was prescribed lifelong treatment with beta-blockers.

The midventricular obstructive form of HCM accounts for 9.4% of cases of hypertrophic cardiomyopathy.1,2 Midventricular obliteration in systole leads to 2-chamber (hourglass) appearance of the LV, which consists of basal and apical chambers.1 Continuous-wave Doppler echocardiography shows a paradoxical jet between 2 chambers: systolic flow from the apical chamber to the basal one interrupts in mid-systole, and then continues in late systole and early diastole due to relief of obstruction.3 LVAA is present in up to 28.3% of patients with midventricular obstruction.1 While midventricular obstruction is an independent risk factor for sudden death, apical aneurysm poses the risk of arrhythmia and embolic stroke.2

Left ventricular apical aneurysm in HCM should be differentiated from postinfarction aneurysm, LV diverticulum, cardiac sarcoidosis, myocarditis, Behçet disease, and Chagas disease. It is a common complication after myocardial infarction, associated with a particularly difficult decision-making process regarding treatment.4 Presence of midventricular obstruction helps to differentiate between midventricular HCM and other causes of aneurysm formation.1 Coronary angiography is needed to rule out ischemic heart disease. Cardiac computed tomography has the advantage of precise assessment of LV morphology and wall thickness, as well as simultaneous evaluation of the coronary arteries. Contrast-enhanced

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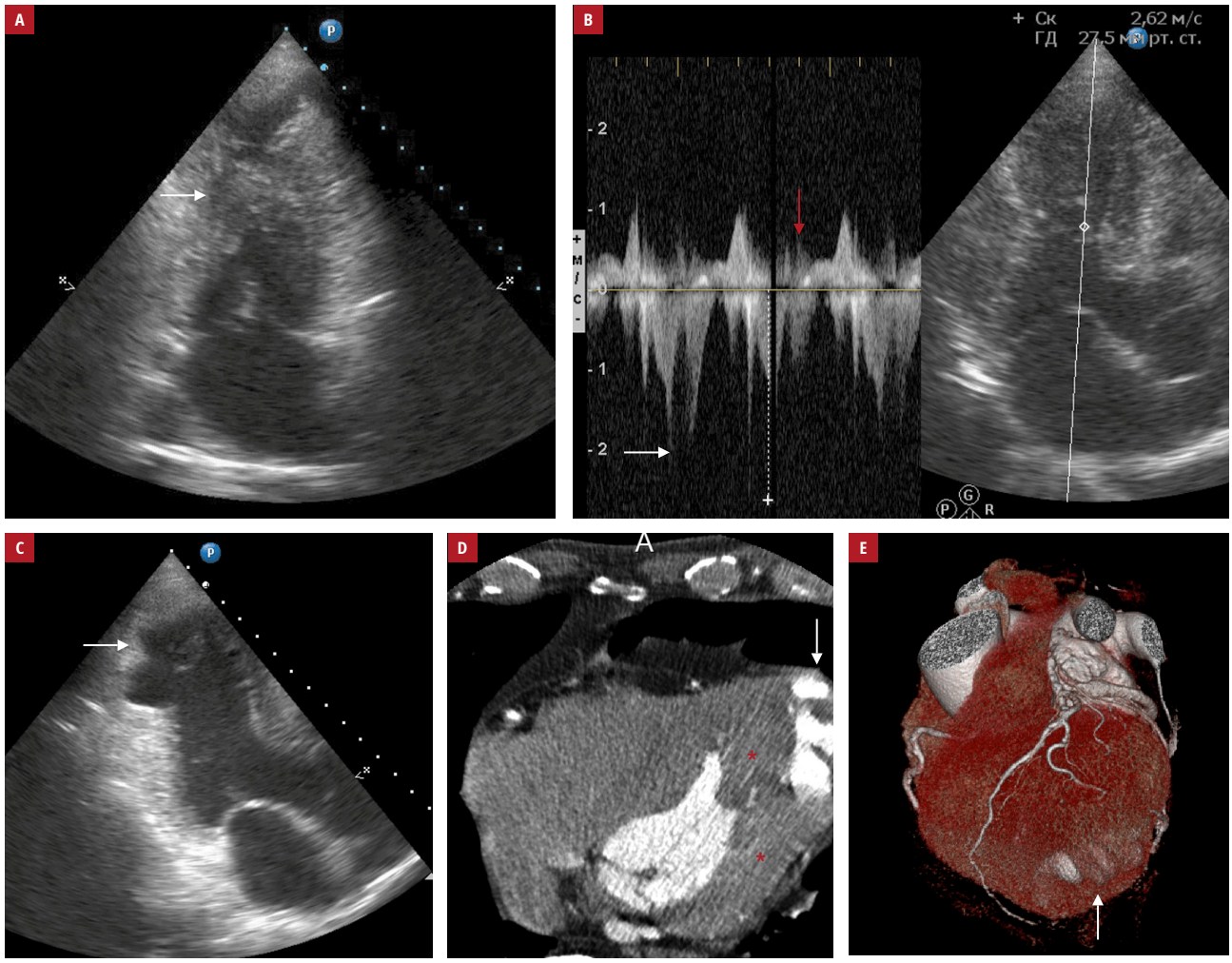

FIGURE 1 **A** – echocardiography, apical 2-chamber view: midventricular hypertrophy with obstruction of the left ventricular cavity in systole (arrow); **B** – Doppler echocardiography showing the systolic midventricular gradient (white arrow) and the diastolic gradient (red arrow) between the basal and apical left ventricular chambers; **C** – echocardiography, apical long-axis view: apical aneurysm (arrow); **D** – contrast-enhanced cardiac computed tomography: apical aneurysm (arrow) and hypertrophic midventricular segments (asterisks); **E** – three-dimensional reconstruction of apical aneurysm (arrow) on cardiac computed tomography

cardiac magnetic resonance imaging allows clinicians to evaluate LV geometry and the extent of myocardial fibrosis.5 Treatment recommendations for the management of HCM with apical aneurysm are scarce. Implantable cardioverter-defibrillator insertion and lifelong anticoagulation should be considered. The decision to perform aneurysmectomy depends on the risk of developing systemic embolism and arrhythmia.1,4

SUPPLEMENTARY MATERIAL

Supplementary material is available at www.mp.pl/kardiologiapolska.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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