Left ventricular aneurysm in hypertrophic cardiomyopathy with mid-ventricular obstruction

Authors: Nelya Oryshchyn, Yuriy Ivaniv

Article type: Clinical vignette

Received: February 29, 2020.

Accepted: March 18, 2020.

Published online: March 19, 2020.

ISSN: 0022-9032

e-ISSN: 1897-4279

This is an Open Access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives 4.0 International License (CC BY-NC-ND 4.0), allowing third parties to download articles and share them with others, provided the original work is properly cited, not changed in any way, distributed under the same license, and used for noncommercial purposes only. For commercial use, please contact the journal office at kardiologiapolska@ptkardio.pl.
Left ventricular aneurysm in hypertrophic cardiomyopathy with mid-ventricular obstruction

Nelya Oryshchyn¹², MD, PhD, Yuriy Ivaniv¹, MD, PhD

¹Danylo Halytsky Lviv National Medical University, Ukraine
²Lviv Regional Centre of Cardiology, Ukraine

A short title:

Left ventricular aneurysm in hypertrophic cardiomyopathy

Corresponding author: Nelya Oryshchyn, MD, PhD, Diagnostic Radiology Department, Danylo Halytsky Lviv National Medical University, 69 Pekarska str. Lviv, 79010, Ukraine, ph.: +38 (032) 275-76-32, oryshchyn_n@yahoo.com

I have nothing to declare (there is no conflict of interests)
We report a case of a left ventricular apical aneurysm (LVA), caused by mid-ventricular hypertrophic cardiomyopathy (HCMP). A 64-year-old male patient was admitted to the hospital with suspicion of a postinfarction LVA. The patient suffered from transitory ischemic attack 2 months before the admission. Cerebral magnetic resonance imaging (MRI) revealed multiply cerebral infarctions of probably embolic origin. During the search for the cardiac source of embolism LVA was revealed. Electrocardiography demonstrated signs of left ventricular (LV) hypertrophy with strain. Echocardiography (EchoCG) (Philips XI XP, Philips Healthcare) showed normal LV systolic function, hypertrophy of mid-ventricular LV segments, with near-complete obliteration of LV camera during systole (hourglass configuration – Fig. 1A, Suppl. Video 1). Continuous Doppler showed an apical to basal LV cavity gradient of 36 mm Hg in systole and early diastole (Fig. 1B). We revealed apical LV outpouching, connecting with the LV apical chamber through the narrow neck, consistent with the diagnosis of the LVA due to mid-ventricular HCMP (Fig. 1C, Suppl. Video 2,3). Coronary angiography didn’t show any lesion in coronary arteries. Cardiac computed tomography (CT) (Toshiba Aquilion 128 TSX-101A, Toshiba Medical System) demonstrated midventricular LV hypertrophy with obstruction of flow in systole, LVA of three-leaf form, without clots inside it (Fig. 1D,E). Given the high risk of cerebral embolic stroke due to clot formation in the LVA surgical aneurysmectomy was performed. Obtained specimens were explored by morphologist, hypertrophied cardiomyocytes with signs of hibernation and necrosis were revealed in the aneurysmal wall, confirming the diagnosis of HCMP. Beta-blockers were prescribed to the patient as life-long medication.

Midventricular obstructive form of HCMP accounts for 9.4% of cases of hypertrophic cardiomyopathy [1, 2]. Midventricular obliteration in systole leads to two-chamber (“hourglass”) appearance of the LV cavity, which consists of basal and apical chambers [1].
Continuous Doppler shows paradoxical jet between two chambers: systolic flow from apical chamber to basal one interrupts during mid-systole, then continues in late systole and early diastole due to relief of obstruction [3]. LVA is present in up to 28.3% of patients with mid-ventricular obstruction [1]. While midventricular obstruction is an independent risk factor of sudden death, apical aneurysm poses the risk of arrhythmic disorders and embolic stroke [2].

Apical LVA in HCMP should be differentiated from postinfarction aneurysm, LV diverticulum, cardiac sarcoidosis, myocarditis, Behcet and Chagas disease. LVA is a common complication after myocardial infarction, posing especially difficult decision making in the treatment of a patient [4]. Presence of mid-ventricular obstruction helps to differentiate between mid-ventricular HCMP and other causes of aneurysms [1]. Coronary angiography is needed to rule out ischemic heart disease. Cardiac CT has the advantage of precise assessment of LV morphology, wall thickness and simultaneous assessment of coronary arteries. Cardiac MRI with contrast allows to evaluate LV geometry and the extent of myocardial fibrosis [5]. Treatment recommendations are scarce in HCMP with an apical aneurysm. ICD implantation, life-long anticoagulation are under consideration. The decision about surgical aneurysmectomy depends on the risk of systemic embolism and arrhythmic disorders [1, 4].
References:


Figure 1.

A. Echocardiography. Apical two-chamber view. Mid-ventricular hypertrophy with obstruction of the left ventricular cavity in systole (arrow);

B. Doppler-echocardiography shows mid-ventricular gradient between basal and apical left ventricular chambers: arrow – systolic mid-ventricular gradient, arrowhead – diastolic gradient;

C. Echocardiography, apical long-axis view. Arrow – apical aneurysm;

D. Cardiac computed tomography with contrast. Arrow – apical aneurysm, asterisks – hypertrophied mid-ventricular segments;

E. 3D reconstruction in cardiac computed tomography. Arrow - apical aneurysm.