Giant apical pseudoaneurysm in the left ventricle as a late complication of takotsubo syndrome: Not a benign course of the disease

Mislav Lozo[1, 2*], Lovel Giunio[1, 2*], Dino Miric[1, 2], Darija Bakovic[1, 2], Cristijan Bulat[3], Tonci Batinic[4], Anteo Bradaric[1, 2], Jaksa Zanchi[1, 2], Josip A Borovac[1, 2]

[1]Department of Cardiovascular Diseases, University Hospital of Split, Split, Croatia
[2]University of Split School of Medicine, Split, Croatia
[3]Department of Cardiac Surgery, University Hospital of Split, Split, Croatia
[4]Department of Radiology, University Hospital of Split, Split, Croatia
[*]Both authors equally contributed to the study

Correspondence to:
Josip A Borovac, MD, PhD, FHFA, Department of Cardiovascular Diseases, University Hospital of Split (KBC Split), Soltanska 1, 21 000 Split, Croatia, phone: +385 92 172 13 14, e-mail: jborovac@mefst.hr

DOI: 10.33963/KP.a2023.0136

Received:
April 18, 2023

Accepted:
June 10, 2023

Early publication date:
June 18, 2023

Left ventricular pseudoaneurysm (LVP) is a rare and life-threatening complication that is most often reported after myocardial infarction or cardiac surgery but can also occur after bacterial endocarditis, chest trauma, or myocardial tumor invasion [1, 2]. LVP develops when a zone of free-wall cardiac rupture is contained by the pericardium or scar tissue, without myocardial tissue involvement. Fatal rupture can be prevented by urgent surgical aneurysmectomy [3]. LVP patients treated with surgery have a mortality rate of 23%, while those treated medically die in 48% of cases [4]. To the best of our knowledge, LVP as a late complication of takotsubo syndrome (TS) has not been described in the literature while Jaguszewski et al. reported on ventricular rupture as an early complication of TS thus confirming that this entity might not always have a benign course [5].

A 77-year-old female with a medical history of arterial hypertension, non-insulin-dependent type 2 diabetes mellitus, dyslipidemia, hypothyroidism, and rheumatoid arthritis presented with crushing substernal chest pain that started 5 hours earlier. Her ECG showed sinus tachycardia (104 bpm) and diffuse ST-segment elevations in the inferior and anteroseptal leads. She reported that the symptoms had started following an emotionally intense event (a large family reunion dinner). Cardioselective biomarkers were markedly high (high-sensitivity troponin I level of 4894 ng/l and N-terminal prohormone of brain natriuretic peptide (NT-proBNP) level of 4221 pg/ml). A diagnosis of acute coronary syndrome was made, and an urgent invasive work-up was undertaken. We performed coronary angiography with left ventriculography that revealed the akinetic/dyskinetic midsegment and apical parts of the left ventricle (LV) accompanied by apical ballooning and hyperkinesis of the basal LV segments, consistent with TS diagnosis (Figure 1, Supplementary material, *Video S1*), with no obstructive coronary artery disease (Figure 1B). Furthermore, a transthoracic echocardiographic examination (TTE) showed the formation of an inferoapical mural thrombus (Figure 1C, far left) and reduced left ventricular ejection fraction (LVEF) of 42%. Thus, the patient was discharged with an oral anticoagulant in full therapeutic dose along with optimal medical therapy. Eight weeks later, the patient received a follow-up TTE that showed an increase in systolic function (LVEF, 53%) with complete resolution of the thrombus while the presence of a small inferoapical LV aneurysm was noted (Figure 1C, middle image).

Eight months after this first hospitalization, she presented again to the Emergency Department with worsening dyspnea upon minimal exertion, dry cough, and generalized weakness. Her NT-proBNP level was was 3041 pg/ml, and urgent TTE was performed. It showed a gigantic oval non-contractile structure connected to the LV via a narrow neck (Figure 1C, far right). LVEF was preserved

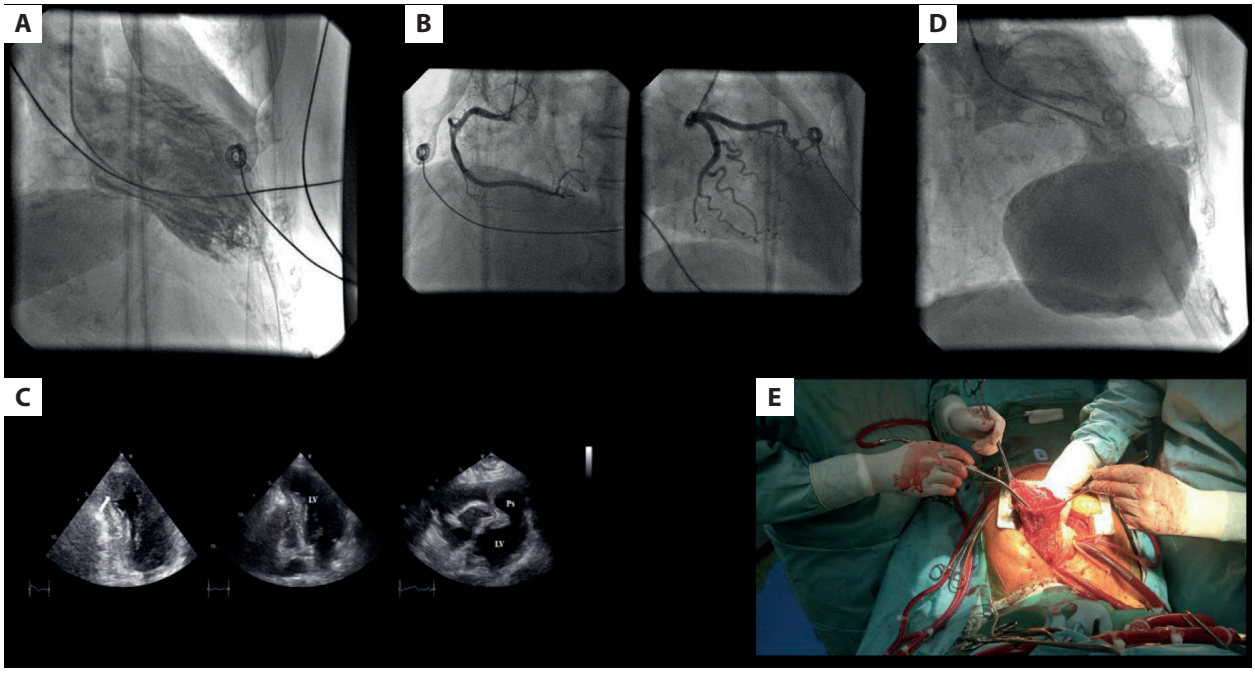

Figure 1. A. Left ventriculography performed during the first hospital admission showed apical ballooning with basal hypercontractility consistent with the diagnosis of takotsubo syndrome as well as the presence of contrast filling defect in the inferoapical region indicating possible thrombus formation. **B.** Coronary angiography performed during the first hospital admission showed normal coronary anatomy without obstructive atherosclerotic disease. **C.** Images from transthoracic echocardiographic examination (TTE) performed during the first hospital admission showing a left ventricular (LV) thrombus (22 × 28 mm, white arrow) (the far left image) followed by complete dissolution of the thrombus 8 weeks later on follow-up TTE (middle image) and a huge oval non-contractile structure connected to LV via a narrow neck surrounded by the isoechogenic wall up to 17 mm of thickness visualized during the second hospitalization. **D.** Left ventriculography performed during the second hospitalization revealing an inferoapical pseudoaneurysm structure 72 × 65 mm in size, with a neck diameter measuring approximately 21 mm. **E.** Surgical resection of the pseudoaneurysm (aneurysmectomy) and closure of the LV by a double-layered pericardial patch performed by cardiac surgeons

(55%). Coronary angiography and left ventriculography were performed again — the coronary arteries were patent while ventriculography showed again the massive oval structure connected to LV via a narrow neck (Figure 1D, Supplementary material, *Video S2*). Cardiac magnetic resonance showed that the pseudoaneurysm wall consisted predominantly of a "sickle-like" mural thrombus up to 15 mm in size surrounded by a thin layer of visceral pericardium up to 2 mm, without perfusion, thus a diagnosis of giant LVP was made (Supplementary material, *Video S3*). Due to the high risk of spontaneous rupture, the patient was referred for urgent surgical aneurysmectomy that was performed by using a double-layer heterologous pericardial patch (Figure 1E). The patient was discharged six days after surgery and was followed up for one year.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/kardiologia_polska.

Article information

Conflict of interest: None declared.

Funding: None.

REFERENCES

1. Caldeira A, Albuquerque D, Coelho M, et al. Left ventricular pseudoaneurysm: imagiologic and intraoperative images. Circ Cardiovasc Imaging. 2019; 12(12): e009500, doi: 10.1161/CIRCIMAGING.119.009500, indexed in Pubmed: 31766861.
2. Moreno R, Gordillo E, Zamorano J, et al. Long term outcome of patients with postinfarction left ventricular pseudoaneurysm. Heart. 2003; 89(10): 1144–1146, doi: 10.1136/heart.89.10.1144, indexed in Pubmed: 12975400.
3. Garcia-Guimaraes M, Velasco-Garcia-de-Sierra C, Estevez-Cid F, et al. Current role of cardiac imaging to guide surgical correction of a giant left ventricular pseudoaneurysm. Int J Cardiol. 2015; 198: 152–153, doi: 10.1016/j.ijcard.2015.06.129, indexed in Pubmed: 26186572.
4. Frances C, Romero A, Grady D. Left ventricular pseudoaneurysm. J Am Coll Cardiol. 1998; 32(3): 557–561, doi: 10.1016/s0735-1097(98)00290-3, indexed in Pubmed: 9741493.
5. Jaguszewski M, Fijalkowski M, Nowak R, et al. Ventricular rupture in Takotsubo cardiomyopathy. Eur Heart J. 2012; 33(8): 1027, doi: 10.1093/eurheartj/ehs054, indexed in Pubmed: 22416075.