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STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Dynamic left ventricular outflow tract obstruction

Dynamiczne zawężanie drogi odpływu lewej komory

Maciej Wójcik, Robert Błaszczyk, Łukasz Konarski, Andrzej Tomaszewski, Andrzej Wysokiński

Department of Cardiology, Medical University of Lublin, Lublin, Poland

A 64-year-old Caucasian woman with stable vital signs, preserved systolic ejection fraction (EF) of 66%, three-hour-lasting typical chest pain, and elevated cardiac troponin levels was diagnosed with acute coronary syndrome (NSTEMI). She was secured with a bare metal stent in the left anterior descending artery in the early afternoon and received standard medication. Overnight, she progressively deteriorated with marked hypotensia, dyspnoea, tachycardia, and worsened peripheral perfusion. Dobutamine infusion was started. By early morning, she was already on high doses of dobutamine. However, she further deteriorated, with systolic blood pressure (BP) between 75 and 85 mm Hg. No signs of ischaemia were visible on 12-lead ECG but a new 4/6 (Levine's scale) systolic ejection murmur in the left third intercostal space was heard. Transthoracic echocardiography (TTE) revealed: impaired EF of 48%, apical ballooning (Fig. 1A, arrows) that further increased during systole (Fig. 1B, arrows), hyper-contractility of left ventricular outflow tract (LVOT) with systolic anterior motion of anterior mitral leaflet (SAM, Fig. 1B) that resulted in a significant LVOT-gradient of 128 mm Hg (Fig. 1C, D), and mitral regurgitation (MR, Fig. 1C). Dobutamine was discontinued and, under close supervision, intravenous metoprolol and fluids were administered. With a reduction of her heart rate and LVOT contractility, as well as increased left ventricle volume (LVV), the murmur disappeared and her BP improved. A repeated TTE several hours later and also the next day showed only mild MR but neither SAM nor dynamic LVOT obstruction (DLVOTO). The LVOT-gradient was 10 mm Hg (Fig. 1E). The actual incidence of DLVOTO is unclear and can be under-estimated. Inotropes are known to induce transient SAM and DLVOTO in otherwise healthy people. SAM is a consequence of dragging Venturi-effect of anterior mitral leaflet by accelerated blood flow in hypercontractile LVOT. SAM further increases the severity of DLVOTO and results in (significant) MR, which clinically presents as 'new' or 'augmented previously heard' systolic murmur. Moreover, transient apical ballooning results not only from local ischaemia and stunning, but also from severity of DLVOTO. Management must be individualised on the basis of the patient's status. Generally, that DLVOTO gradient can be lowered with supervised beta-blockers (or, if contraindicated, nondihydropyradine calcium channel blockers) and fluid infusion that leads to lower basal contractility, increased LVV, disappearance of SAM, and improvement of MR. All this plus subsequent coronary revascularisation reduces apical ballooning by improving apical segments movement and reducing basal hypercontractility. Inotropic agents should be avoided. A standard clinical approach to a patient in currently procedurally-oriented medicine is still important. Any ejection murmur accompanied by haemodynamic compromise detected in acute coronary settings should be screened with echocardiography to exclude DLVOTO.

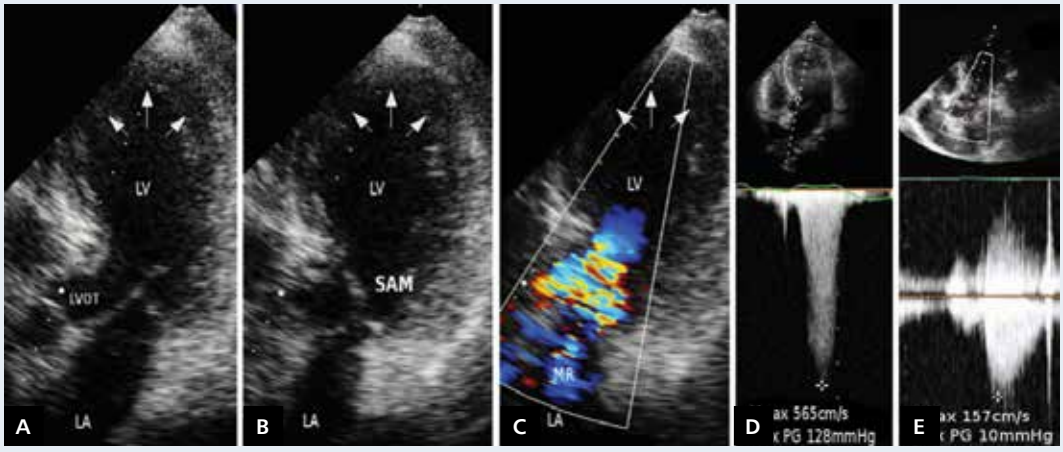

Figure 1. Transthoracic echocardiography. Apical five-chamber view showing: apical ballooning (**A**–**C**), hyper-contractility of left ventricular outflow track (LVOT) with systolic anterior motion (SAM) of anterior mitral leaflet (**B**) that resulted in mitral regurgitation (MR) (**C**) and significant gradient in LVOT of 128 mm Hg (**D**). The control examination (**E**) showing only mild MR but neither SAM nor dynamic LVOT obstruction (with LVOT-gradient of 10 mm Hg); LA — left atrium; LV — left ventricle

Address for correspondence:
Maciej Wójcik, MD, Department of Cardiology, Medical University of Lublin, SPSK Nr 4, ul. Jaczewskiego 8, 20–097 Lublin, Poland, tel/fax: +48 81 724 41 51, e-mail: m.wojcik@am.lublin.pl

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