Persistent ductus arteriosus in old patient with atrial fibrillation

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Persistent ductus arteriosus (PDA) is a congenital cardiovascular malformation connecting the pulmonary trunk of the proximal left pulmonary artery and the descending aorta distal to the left subclavian artery. Normally, the duct closes after birth as a result of a sudden increase in arterial oxygen saturation and a decrease in the level of vasoactive prostaglandins. The incidence of persistent ductus arteriosus accounts for approximately 10% of all congenital heart diseases.[1] PDA is usually diagnosed in early childhood by echocardiography and cardiac catheterization. Moderate patent ductus arteriosus may lead to the increase in pulmonary flow and pulmonary hypertension and cause the pressure overload of the right ventricle. In large PDA, the Eisenmenger syndrome develops, which may be associated with other congenital malformations such as coarctation of the aorta, aortic stenosis, ventricular septal defect (VSD), patent foramen ovale (PFO) or pulmonic stenosis. In adulthood, PDA is usually an isolated finding, related to a higher prevalence of heart failure or endarteritis. Computed tomography (CT) and cardiac magnetic resonance (CMR) as noninvasive methods are indicated when additional quantifications of right ventricle (RV), left ventricle (LV), shunt and pulmonary artery hypertension (PAH) are required.

The 86-year-old woman presented with the symptoms of heart failure New York Heart A II (NYHA II) (after several months of increased exertional dyspnea and paroxysmal irregular palpitations). The medical history disclosed arterial hypertension treated with quinalapril, amiloride, hydrochlorothiazide and metoprolol, and hyperthyroidism successfully treated with thiamazolum [euthyroid with thyroid-stimulating hormone (TSH) 3.09 pg/mL, and free thyroxine (TT4) 1.61 µIU/mL at present]. The physical examination revealed irregular heart rate of about 90 bpm, a continuous murmur in the second left clavicular space, edema and reddening of the right lower limb. The ECG showed atrial fibrillation (AF) with the average ventricular rhythm of 81 bpm (Figure 1). No sign of right ventricular hypertrophy was found on ECG. The 24-hour Holter monitoring of the ECG showed AF with the mean heart rate 69 bpm and 88 asymptomatic pauses exceeding two seconds during the sleep hours (maximum 2663 ms) (Figure 2). Ultrasound imaging of the lower extremities excluded the signs of deep venous thrombosis.

Transthoracic echocardiography demonstrated signs of pulmonary hypertension and a moderate regurgitation of the tricuspid valve. Right ventricular systolic pressure (RVSP) was elevated (65 mmHg), tricuspid annular plane systolic excursion (TAPSE) 16, right atrium (RA) 22 cm², RV 19 cm², interior vena cava (IVC) 15 mm, pulmonary artery (PA) 25 mm, RV/LV eccentricity index 0.9. The global systolic RV function was normal, while pulmonary artery was dilated (28 mm). Doppler echocardiography excluded the presence of an intracardiac shunt. Computed tomography was performed to exclude pulmonary embolism. It showed dilatation of the pulmonary trunk (30 mm) and the right pulmonary artery (29 mm). CT showed signs of a small shunt between the aorta and the pulmonary artery with calcification in the aortic wall (Figure 3).

CMR was ordered for further evaluation and to confirm the diagnosis of the aortopulmonary shunt. It showed a marked dilatation of the right ventricle with a normal thickness of the free wall (2–3 mm), normal systolic function (ejection fraction 62%), paradoxical movement of the interventricular septum, dilated pulmonary trunk (34 mm) and pulmonary arteries (right 30 mm and left 27 mm). The pulmonary valve demonstrated no insufficiency. Delayed-enhancement images subsequently obtained demonstrated a small focus of hyperenhancement in the septal insertion points. The magnetic resonance angiography (angio-MR) revealed a shunt between the aorta beyond the level of the left subclavian artery and the pulmonary trunk (shunt diameter 3 mm) (Figure 4 & 5). Pulmonary angiographic im-

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Figure 1. Atrial fibrillation on electrocardiogram on admission.

Figure 2. The 24-hour Holter monitoring of electrocardiogram: bradycardia and asymptomatic pauses during night.
Rajewska-Tabor J, et al. PDA in old patient with AF

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Figure 3. Aorto-pulmonary shunt in computed tomography angiography of pulmonary arteries. Axial (A) and sagittal (B) images depict a jet of non-enhanced aortic blood into the contrast enhanced pulmonary artery (arrows).

Figure 4. Aorto-pulmonary shunt in the cardiac magnetic resonance (arrow). Ao: aorta; MPA: main pulmonary artery; PDA: persistent ductus arteriosus.

Figure 5. Volume-rendered 3D reconstruction shows PDA (arrow) connecting Ao and MPA. Ao: aorta; MPA: main pulmonary artery; PDA: persistent ductus arteriosus.

ages also showed dilatation of the main pulmonary artery (diameter of the pulmonary trunk was 34 mm).

The magnitude and velocity of the encoded images demonstrated an anomalous turbulent flow (signal void) originating from the aorta and continuing towards the pulmonary artery (Figure 6). The shunt velocity was calculated as 91 cm/s and volume 15 mL/beat. Measurements of the aortic and pulmonary flows with phase-contrast imaging revealed a shunt: pulmonary-to-systemic flow ratio (Qp/Qs) = 1.3, which was considered insignificant. No other abnormalities were observed. Pulmonary perfusion was considered normal.

According to recommendations of European Society of Cardiology (ESC), noninvasive treatment was considered.[2] Due to the diagnosis of the atrial fibrillation of unknown duration, a treatment with oral anticoagulant (rivaroxaban) and β-blocker (metoprolol), beyond previous treatment of arterial hypertension (quinapril, amiloride, hydrochlorothiazide), was applied.

After one year of follow up the status of the patient was stable: there were no significant changes in the ECG monitoring (persistent atrial fibrillation with 50 episodes of as-
ymptomatic above 2 seconds, max. 2 742 ms) and the echocardiographic parameters did not change either. The patient did not report any signs of dizziness or syncope.

Transthoracic echocardiography is the key diagnostic modality in diagnosing the cause of pulmonary hypertension. However, the sonographic access sites are sometimes not adequate for all regions of interests. The choice of a further imaging technique depends on the clinical problem. Pulmonary hypertension is a progressive and frequently fatal disease of different etiology and different treatment options.[3] Therefore, explanation of its cause is crucial. Small intra- or extracardiac shunts can be omitted by transthoracic echocardiography and should always be excluded by further imaging techniques. In the case presented, the angio-CT revealed the equivocal diagnosis due to nondynamic feature of standard imaging and concomitant aortic calcification close to shunt. CMR as a noninvasive technique is nowadays widely used and is becoming the gold standard for visualizing the heart and large arteries. CMR has also become today one of the most versatile of all cardiovascular imaging techniques. It allows imaging of cardiac anatomy, cardiac function, blood flow measurement of the aorta and pulmonary arteries, pulmonary arterial distensibility, perfusion of the lungs. The assessment of the anatomy and function of the right ventricle is very precise and essential in monitoring patients with systemic-pulmonary shunts. It can help to clarify the nature of the lesion, the amount of shunting and can detect associated anomalies.[4,5] Magnetic resonance velocity mapping allows the measurement of flow through planes transecting the great vessels for the calculation of shunt flow volumes.[6]

According to the current recommendations, PDA should be closed in patients with LV volume overload (recommendation class I C), should be considered in patients with PAH and pulmonary artery pressure (PAP) > 2/3 of systemic pressure with Qp/Qs > 1.5 and in patients with small PDA with continuous murmur and normal LV and PAP (recommendation class II C) preferably with percutaneous procedures.[2] When the shunt is small and hemodynamically insignificant (Qp/Qs < 1.5), the recommendations are not so clear. Device closure must be avoided in patients with the Eisenmenger syndrome and a small silent duct. Surgery is reserved for unsuitable anatomy for device closure. Concomitant diseases, age, risk of possible complications after the procedure and patient's preferences must also be considered. Moreover, the elderly patients do not always understand the necessity of treatment, and the life expectancy also seems essential. Individual approach in these patients should be implemented. Patients with LV dysfunction and with PAH should be followed every 1–3 years in specialized centers for Grown Up with Congenital Heart defects (GUCH).

The patient presented here also suffers from persistent atrial fibrillation. The treatment with oral anticoagulants was induced due to Score for Atrial Fibrillation Strok Risk (CHA2DS2-VASCs scale) of 4 points. Following the latest recommendations for stroke prevention in patients with atrial fibrillation, the novel oral anticoagulant (NOAC) (rivaroxaban) treatment was chosen.[7] The advantage of NOACs [no need to control parameters of anticoagulation in serum – international standardized ratio (INR), more reliable anticoagulant treatment in comparison to old oral anticoagulants (OACs)] have been the reasons of the chosen therapy.

The diagnosis and treatment of congenital heart disease in elderly patients is challenging. Cardiac magnetic resonance imaging is non-invasive and quantifies accurately the cause of pulmonary hypertension in elderly patients with an undiagnosed congenital heart disease. Furthermore, it can be safely performed to monitor pulmonary hypertension without the risk of ionizing radiation.

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