

Patent ductus arteriosus recanalization following its successful surgical closure (RCD code: IV-2B.4)

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Abstract

Patent ductus arteriosus (PDA) is rarely diagnosed late in adult life, and frequently in children as an isolated disorder or complex congenital heart defect. PDA represents an anomaly defined by the communication between descending thoracic aorta below the origin of left subclavian artery and proximal part of left pulmonary artery. The hemodynamic sequelae and resultant symptomatology of PDA depend on the diameter of communication and the difference between systemic and pulmonary vascular resistance. We present a case of an adult female patient after surgical repair of PDA with surgical patch leak. JRC D 2015; 2 (4): 115–118

Key words: PDA, congenital heart disease, treatment, patch leak

Case report

55-year-old woman who formerly underwent surgical closure of patent ductus arteriosus (PDA) was admitted to the department with the history of gradual impairment of exercise tolerance within 3 years before the index hospitalization. In direct pre-admission period the patient was characterized by dyspnea on exertion in New York Heart Association class III (NYHA).

Primary diagnosis and surgery

PDA was confirmed at the age of 40 with the concurrent functional capacity of NYHA class II. On physical examination prior to surgery, the patient exhibited a loud machine-like heart murmur over whole chest and irregular heart rhythm consistent with permanent atrial fibrillation in the electrocardiogram. Chest radiograph revealed enlarged cardiac silhouette along with prominent bronchovascular markings. Transthoracic echocardiography (TTE) denoted a turbulent continuous flow above pulmonary valve typical for PDA, and dilated left atrium (51 mm) and left ventricle (70/46), together with interventricular septum hypertrophy (13–14 mm), as well as moderate mitral and tricuspid valve

insufficiency. The patient was submitted to on-pump surgical closure of PDA in hypothermia (30°C) with the use of patch introduced via pulmonary artery. The direct postoperative period was uneventful. The patient was extubated the same day, while the drainage was ceased within 24 hours following the surgery. However, in the subsequent days hydrothorax and fever (body temperature of 38°C) were documented. The operative wound healed by primary intention and sutures were removed on the 8th postoperative day. The patient was discharged home for further ambulatory follow-up. Digoxin, spironolactone and furosemide were prescribed. Throughout further 15 years of follow-up the patient was not admitted to any hospital and refrained from regular follow-up visits. Still, the patient was characterized by permanently limited exercise tolerance, which aggravated within 3 years preceding current hospitalization.

Present hospital stay

During the index hospitalization several episodes of pulmonary congestion were observed, which required diuretic dose escalation. Yet, the patient was free from signs of right ventricular heart failure. Machine-like murmur was persistently present on chest auscultation. Rest electrocardiogram showed atrial fibrillation

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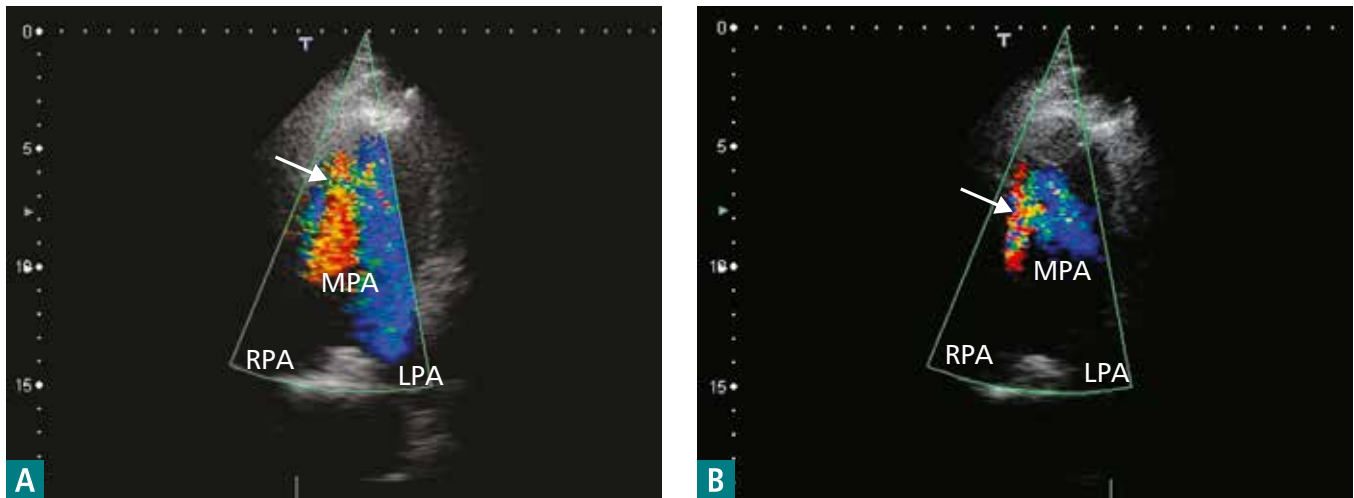


Figure 1. Transthoracic echocardiography. Parasternal short-axis view. Color Doppler ultrasound reveals the presence of turbulent shunt between aorta and pulmonary artery trunk in the systole (Panel A) and diastole (Panel B) (arrows). MPA – main pulmonary artery; LPA – left pulmonary artery, RPA – right pulmonary artery

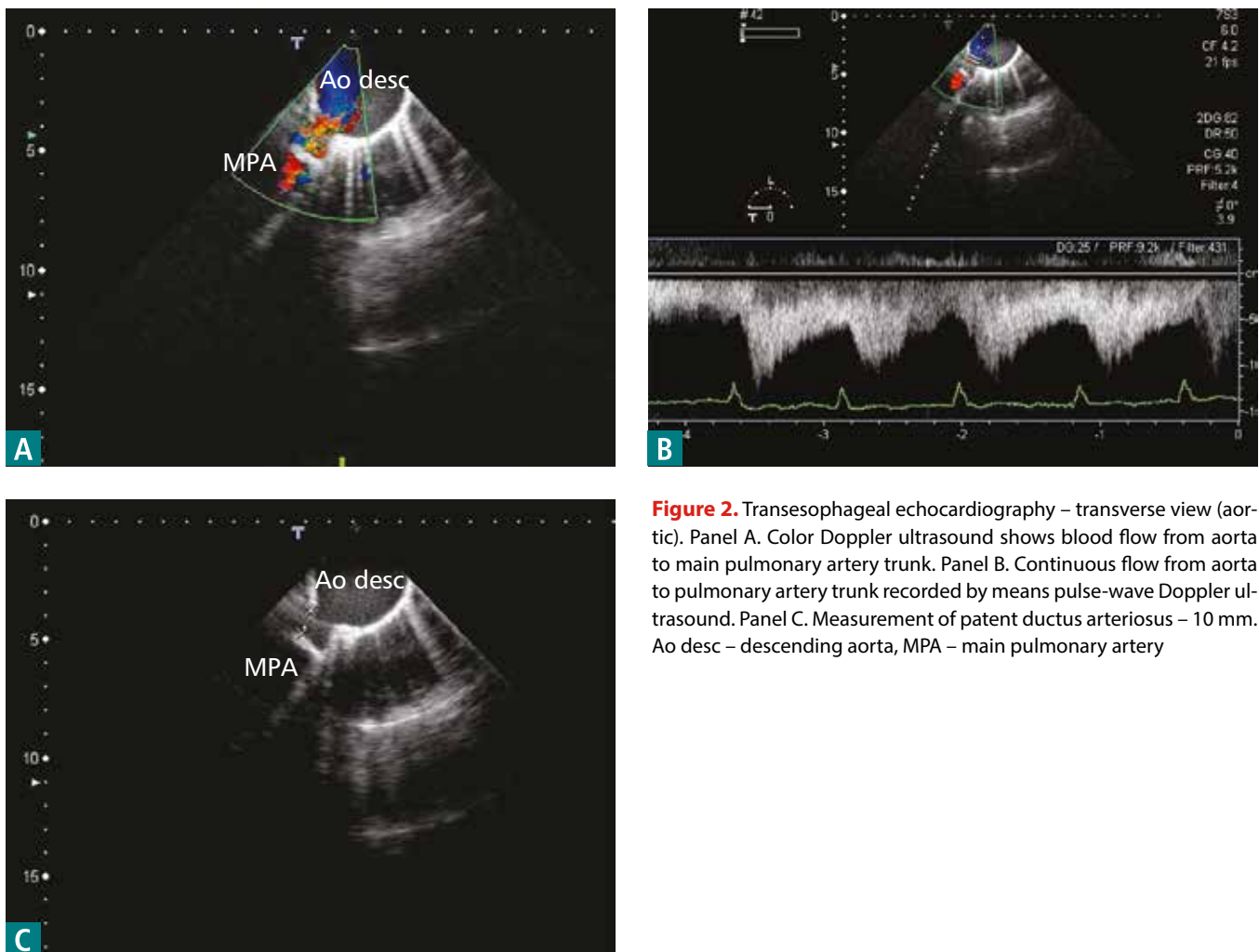


Figure 2. Transesophageal echocardiography – transverse view (aortic). Panel A. Color Doppler ultrasound shows blood flow from aorta to main pulmonary artery trunk. Panel B. Continuous flow from aorta to pulmonary artery trunk recorded by means pulse-wave Doppler ultrasound. Panel C. Measurement of patent ductus arteriosus – 10 mm. Ao desc – descending aorta, MPA – main pulmonary artery

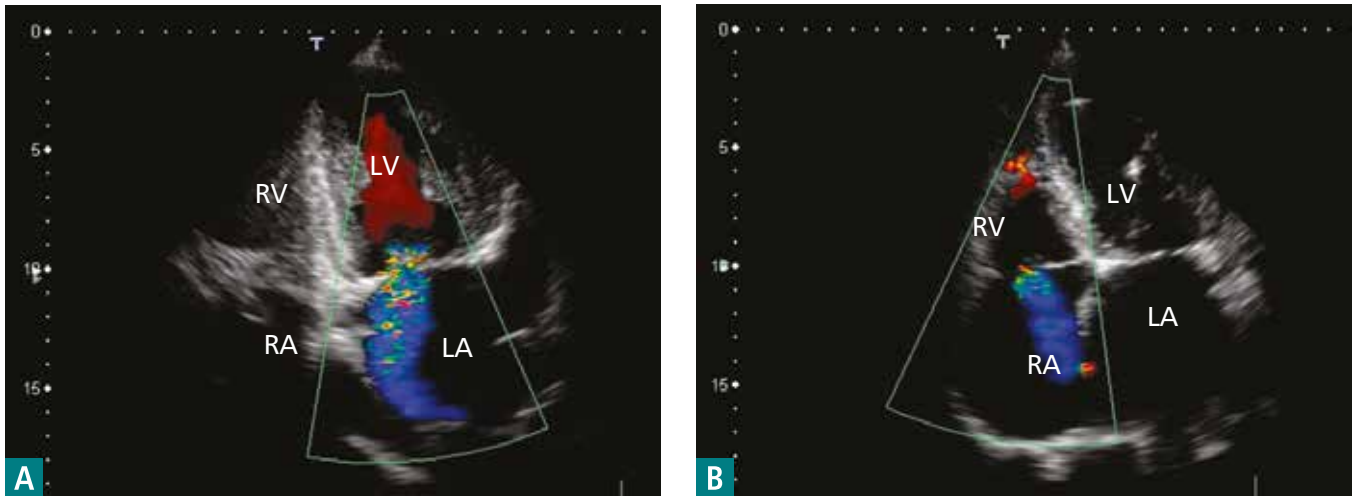


Figure 3. Transthoracic echocardiography. 4-chamber apical view. Panel A. Severe mitral valve insufficiency. Panel B. Tricuspid valve insufficiency. LV – left ventricle, RV – right ventricle, LA – left atrium, RA – right atrium

with mean heart rate of 60 bpm. The patient received digoxin (0.1 mg q.d.), furosemide (40 mg q.d.), spironolactone (100 mg q.d.), trandolapril (0.5 mg q.d.), acetylsalicylic acid (75 mg q.d.), carvedilol (6.25 mg b.i.d.) and levothyroxine (25 µg q.d.).

TTE demonstrated dilation of both left atrium (diameter 58 mm; area 58 cm²), and left ventricle (72/47 mm), and preserved left ventricular ejection fraction (LVEF 54%). Yet, the size of right ventricle remained within reference values (25 mm). These results were comparable with those from the year 1990. Precise evaluation of the vicinity of pulmonary trunk led to the discovery of continuous flow between aorta and pulmonary trunk in the site of formerly implanted surgical patch, along the lateral wall of pulmonary trunk towards the pulmonary valve (Figure 1).

Transesophageal echocardiography (TEE) allowed for precise visualization of the lesion, including adequate measurement of the diameter and flow velocity through the false vascular communication (Figure 2). The diagnosis of PDA recanalization was established. Moreover, TEE revealed dilation of mitral valve annulus (38 mm), lack of mitral valve leaflet coaptation, and thickening of chordae tendinae with patchy calcifications. Doppler ultrasound indicated increased diastolic mitral flow velocity (V_{max} 1.6 m/s) and mitral regurgitation (mitral regurgitation area of 12–14 cm²). Pulmonary veins distention was also documented (Figure 3).

In addition, aortic valve leaflets also showed signs of degeneration, including cusp thickening, increased aortic leaflet mobility with patchy calcifications, and normal aortic annulus diameter. Doppler ultrasound of aortic valve confirmed increased maximal flow velocity (V_{max} 1.9 m/s) and the 2nd degree of aortic valve regurgitation (vena contracta 5–6 mm; left ventricle outflow tract diameter 29 mm).

The assessment of tricuspid valve denoted the 2nd degree of tricuspid regurgitation with elevated estimated right ventricular systolic pressure (RVSP 60 mm Hg) (Figure 3), as well as increased pulmonary artery trunk diameter (36 mm) (Figure 4).

Thoracic computed tomography (CT) confirmed former echocardiographic findings. CT revealed PDA with parietal calcifications (length 18 mm; diameter 10 mm) located roughly 11 mm from the origin of left subclavian artery [Figure 4].

Coronary angiography allowed for the exclusion of coronary artery disease.

In the course of right heart catheterization (RHC) following values of pressure were acquired: right atrium – 7 mmHg, right ventricle 52/3 mmHg (mean 18 mmHg), pulmonary artery 45/21 (mean 31 mmHg), aorta 162/50 mmHg (mean 87 mmHg). Blood oximetry confirmed systemic-to-pulmonary shunt on the level of pulmonary artery. Blood oximetry values were as follows: right atrium – 64%, right ventricle 68%, pulmonary artery trunk – 82%, aorta – 95%. The ratio of pulmonary-to-systemic blood flow (Q_p/Q_s) was 2:1. The patient was qualified for cardiac surgery procedure involving surgical PDA closure and mitral valve repair.

Review of literature

The reported case represents a scenario of a very late diagnosis of PDA, which persisted additional 15 years despite surgical repair, leading to the progression of adverse myocardial remodeling related with predominantly left ventricular, but also secondary right ventricular overload.

The natural course of this congenital heart defect can be various [1]. In this particular case the anomaly was asymptomatic. In the moment of initial diagnosis (at the age of 40) signs of left atrial and left ventricular volume overload were present. Yet a different manifestation of PDA was atrial fibrillation.

The patient was initially referred for surgical PDA ligation. The percutaneous approach was not available at the moment of primary diagnosis. Presumably due to the specific morphology of PDA, an atypical surgical approach involving trans-pulmonary introduction of occluding patch was selected. Noteworthy, relatively simple procedure of PDA ligation in children represents a much more serious and complex surgical procedure in adult patients, which is linked to chronic adverse intra-wall remodeling of PDA (parietal calcifications).

Considering patient's symptomatology, residual shunt might have been already present in the perioperative period, while docu-

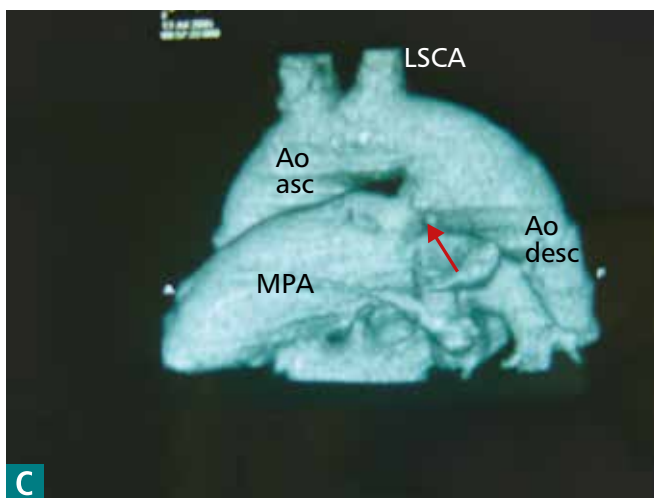
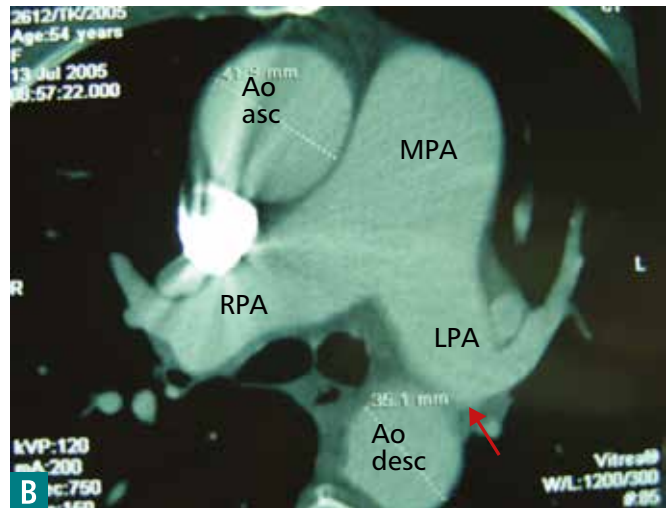
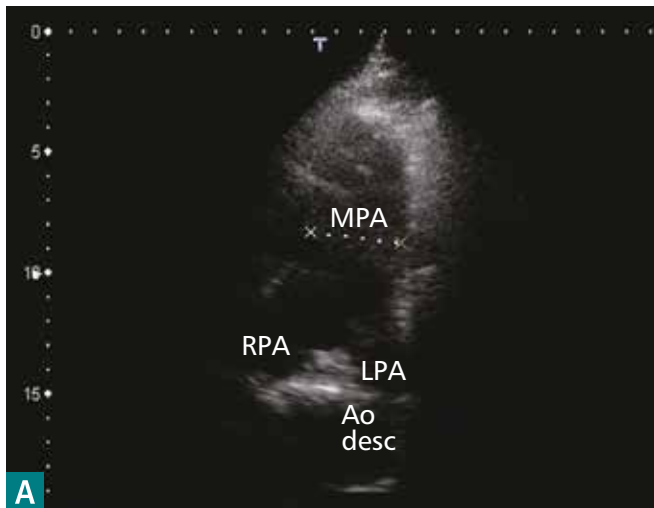


Figure 4. Panel A. Transthoracic echocardiography – parasternal short-axis view – severe dilation of pulmonary artery trunk. Panel B. Comparable respective view acquired by thoracic computed tomography. Panel C. Thoracic computed tomography – 3D reconstruction with apparent patent ductus arteriosus (red arrow) below the origin of left subclavian artery. MPA – main pulmonary artery trunk; LPA – left pulmonary artery; RPA – right pulmonary artery; Ao asc – ascending aorta; Ao desc – descending aorta, LSCA – left subclavian artery

mented fever could be related with postoperative inflammation. The reason for the lack of adequate follow-up remains unknown, since both symptomatology and continuous loud murmur should trigger prompt hospital readmission and implementation of adequate management.

The indications for surgical re-intervention were based on several premises. The PDA diameter of 10 mm represents a limitation to percutaneous approach (trans-catheter PDA closure should be applied for PDA with diameter of < 8 mm with the success rate of 85%) [1–3]. The concomitant indication for mitral valve repair adjudicates in favor of surgical management. The invasively measured pulmonary artery pressure was only mildly elevated, which is of utmost significance, as severe pulmonary hypertension represents an absolute contraindication for PDA closure.

It should be underlined that surgical management in case of PDA with diameter >8mm is associated with higher rate of successful closures (95%) than percutaneous treatment, but at the same time surgical ligation is related with higher mortality rate (pulmonary hypertension; PDA morphology) [1, 4]. Calcified or aneurysmal PDA constitutes a great surgical challenge on account of low elasticity and high frailty of the operated tissue, which results in the increased risk of perioperative bleeding [5].

On the basis of the presented case, one should draw a conclusion that all patients following PDA closure should be diligently followed up so as to exclude possible PDA recanalization. Paucity of data exists regarding the risk of late endarteritis related with residual shunts. In such a clinical scenario, antibiotic prophylaxis of endocarditis is indisputably warranted.

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