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Case Report

Atrial septal defect in the elderly

Agnieszka Kuczaj [a,*], Mirosław Polok [b], Ewa Nowalany-Kozielska [a], Piotr J. Stryjewski [c]

[a] II Clinical Ward of Cardiology, Silesian University of Medicine, Zabrze, Marii Curie Skłodowskiej 10, Poland
[b] Division of Radiology, Multidisciplinary Hospital in Zabrze, Zabrze, Marii Curie Skłodowskiej 10, Poland
[c] Cardiology Department, Chrzanow City Hospital, Poland

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ABSTRACT

Atrial septal defect (ASD) can be diagnosed in the old-age patients. Patients have unspecific symptoms such as exertional shortness of breath, reduced functional capacity, palpitations and pulmonary infections. Diagnostic work-up of ASD consists of the ECG, echocardiography, cardiac tomography or cardiac magnetic resonance. In the cases of concomitant pulmonary artery hypertension right heart catheterization is needed. Patients with ASD are candidates for surgical/catheter interventional treatment at any age.

1. Introduction

It is difficult to make appropriate diagnosis in adult patients with congenital heart defects, including patients with atrial septal defect (ASD) and anomalous pulmonary venous drainage. The diagnostic process is both complicated and long-lasting. Clinical signs related to ASD may be uncharacteristic and may suggest the lung disease. Due to poor quality of visualization which makes it impossible to detect ASD, transthoracic echocardiography is not always conclusive. Furthermore, in light of unspecific symptoms, the observed signs of probable pulmonary hypertension might be linked to the suspected pulmonary disease.

Hence, we present two clinical cases i.e. a patient with ASD of the second type (ASD II) with concomitant partial anomalous pulmonary venous return and a patient with isolated ASD of the second type. In both cases patients complained of shortness of breath occurring over long periods of time and were primary investigated for respiratory disease. Both patients were previously hospitalized at pulmonary wards due to the suspicion of asthma and chronic obstructive pulmonary disease (COPD). Diagnoses from prior discharge summary cards were as follows: mild bronchial asthma, suspicion of COPD, heart failure and pulmonary arterial hypertension.

* Corresponding author.
E-mail address: agnieszka.kuczaj@gmail.com (A. Kuczaj).
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2. Case one – a patient with ASD of the second type with concomitant partial anomalous pulmonary venous return

A 68-year-old female with diagnosed bronchial asthma was admitted to the cardiology ward in order to undergo further diagnostics of pulmonary hypertension detected in an outpatient clinic. The patient complained of progressive exertional chest dyspnea with palpitations lasting about five years.

At the time of presentation the patent was in NYHA heart failure II/III functional class. Blood pressure was 130/80 mmHg. Physical examination showed no signs of cyanosis, peripheral edema or right heart insufficiency. On auscultation a prominent systolic murmur was detected together with a loud second sound with maximal intensity in the second left intercostal space.

A routine chest x-ray in the posteroanterior view showed bilaterally enlarged pulmonary hila with dominant right pulmonary hilum. The enlarged vascular shadow was strongly pronounced in the lateral view (Fig. 1).

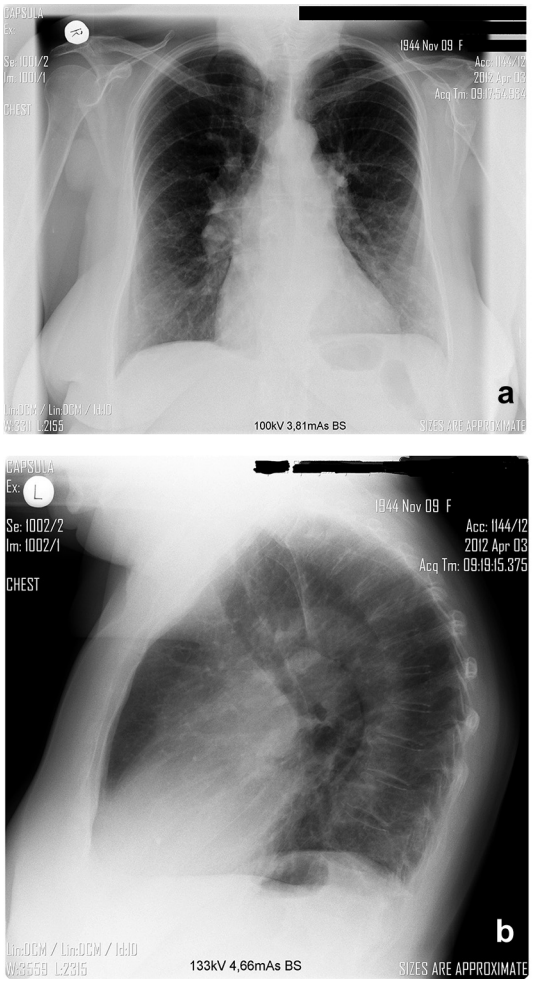

Fig. 1 – Chest x-ray (posteroanterior and lateral views). The posteroanterior (a) view shows bilaterally enlarged pulmonary hila with dominant right lung hilum. Lateral view (b) shows strongly pronounced enlarged vascular shadow.

Resting 12-lead ECG showed sinus rhythm, incomplete right bundle branch block, ST segment depression of 1 mm in V5–V6, flat-negative T waves in I, aVL, V5–V6. Coronary angiography performed during previous hospitalization in another medical center two years ago due to the observed ECG alterations excluded coronary artery disease as a cause of the reported complaints. The examination showed normal coronary artery vessels and the revision of coronary angiography during hospitalization was abandoned due to the prior finding.

24-h Holter ECG monitoring showed multiple supraventricular extrasystoles, multiple supraventricular pairs and episodes of paroxysmal supraventricular tachyarrhythmia (the longest consisted of 7 beats with the mean frequency of 132 bpm). Spirometry showed slightly decreased values suggesting pulmonary obstructive disease of moderate grade. A 6-min walk test showed significant a limitation of exercise tolerance (distance 257 m, Borg 3) with saturation decrease from 96% to 94% after the exercise.

The value of N-terminal pro-B-type natriuretic peptide (NTproBNP) (3530 pg/ml) confirmed the presence of heart failure. Transthoracic echocardiography showed an enlarged right ventricle (54 mm; parasternal axis) with a thickened right ventricle wall (8 mm), right ventricle/left ventricle index 51/32; 4 chamber view, echocardiographic signs of pulmonary hypertension (right ventricular systolic pressure: 95 mmHg) calculated from the tricuspid regurgitation velocity and the inferior vena cava diameter, acceleration time: 80 ms, eccentricity index 1.27 (systole)/1.57 (diastole), small pulmonary regurgitation, tricuspid annular plane systolic excursion: 18 mm and the left to right shunt with the ratio of 6:1 calculated from the continuity equation.[1] A probable place of the shunt was not identified and confluence of pulmonary veins was not sufficiently visible.

Transesophageal echocardiography (TEE) was recommended to the patient in order to visualize the confluence of pulmonary veins and to detect the potential ASD. The patient refused the TEE investigation fearing the discomfort accompanying the procedure.

In order to assess the grade of pulmonary hypertension and the significance of the shunt, invasive pulmonary circulation was measured. The measurement confirmed the shunt advancement and very high oxygen saturation in the pulmonary artery of 94.2% with oxygen partial pressure of 68.7 mmHg. Arterial blood oxygen saturation from pulse oximetry was 96.3%, venous blood oxygen saturation measured invasively in the right atrium and the right ventricle was 92.1%, saturation measured in the inferior vena cava was 81.6%, and 84.7% in the superior vena cava. Calculated mixed venous blood oxygen saturation was 83%.

The grade of pulmonary hypertension was assessed as moderate and irreversible (mean pulmonary artery pressure – mPAP 36.33 mmHg, mean arterial pressure – mAP 106 mmHg, pulmonary capillary wedge pressure – PCWP 15 mmHg, pulmonary venous resistance – PVR 3.96 Wood units, transpulmonary gradient – TPG 21.8 mmHg, CO 5.39 l/min; with nitric oxygen supply: mPAP 30.67 mmHg, mAP 99.67 mmHg, PCWP 15 mmHg, PVR 2.85 Wood units, TPG 15.7 mmHg, CO 5.49 l/min). Hemodynamic investigation results suggested the presence of the left–right shunt.