Case Report
Correction of right to left shunt by transcatheter treatment of pulmonary stenosis and atrial septal defect: a case report

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Abstract: Co-occurrence of atrial septal defect and pulmonary valve stenosis is a rare entity for adult population. It is possible to correct both the pulmonary stenosis and the defect in the atrial septum by transcatheter methods. We present a case of right to left shunt due to severe pulmonary stenosis and atrial defect which was successfully corrected by transcatheter pulmonary valvuloplasty and closure of the atrial septal defect.

Keywords: Pulmonary stenosis, atrial septal defect

Introduction
Atrial septal defect (ASD) is a relatively common congenital heart disease which is currently treated by transcatheter intervention [1]. Pulmonary stenosis (PS) has a rare occurrence in adult population [2]. Since the first introduction by Kan et al. in 1982, percutaneous balloon pulmonary valvuloplasty is a first choice treatment option for isolated PS in all age groups [3]. Co-occurrence of ASD and severe PS is rare. Treatment of both diseases by simultaneous transcatheter intervention has been performed previously [4]. But, in patients with concomitant ASD and severe PS, the development of pulmonary hypertension and related pulmonary vascular damage is potentially prevented. And, depending on the level of PS severity, the shunt can be reverted to right-to-left. We present a case of right to left shunt due to concurrent severe PS and ASD which was successfully corrected by transcatheter pulmonary valvuloplasty and closure of the ASD.

Case report
A 45 years of female was admitted with increasing resting dyspnea during the last fortnight, and cyanotic appearance was evident. Her cardiac examination revealed loud second heart sound and a 3/6 pansystolic murmur at upper left sternal border. Jugular vein distention, 2+ pretibial edema and cyanotic appearance were evident. NYHA functional capacity was class 4 was followed in intensive care unit. She has had diagnosed with ASD about four years ago, and due to right-left shunt no surgery was planned and had been followed by medical therapy. Normal sinus rhythm, incomplete right bundle block, inverted T wave in inferior derivations was evident in electrocardiography. Transthoracic echocardiography (TTE) was performed and left ventricular systolic and diastolic dimensions and functions were within the normal limits. The right atrium and ventricle was dilated, and D-shaped appearance of interventricular septum at short axis view was detected. In pulmonary valve level, peak gradient was 80 mmHg and mean pressure gradient of 50 mmHg was detected by Doppler echocardiography. Transoseophageal echocardiographic examination (TEE) revealed a 10 mm secundum type ASD (Figure 1), and atrial rims being > 5 mm. Thickened and calcified pulmonary valve was determined by TEE (Figure 2). Diagnostic cardiac catheterization revealed a 5 mmHg of right atrial pressure, 22 mmHg of systolic pulmonary arterial pressure, pulmonary capillary wedge pressure of 9 mmHg, and right ventricu-
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Figure 1. Bi-caval view of atrial septal defect in transeophageal echocardiography.

Figure 2. Appearance of stenotic pulmonary valve in transeophageal echocardiography.

Figure 3. Fluoroscopic image appearance of pulmonary valve balloon dilatation.

Pulmonary arterial blood oxygen saturation was 31%, 96% at pulmonary venous level, and 63% in aorta.

The Qp/Qs was calculated as 0.5. Then the ASD was temporarily occluded with balloon and the aortic blood oxygen saturation and right atrial pressure was re-examined. After the balloon occlusion the aortic saturation was significantly increased reaching to 96%. No change was observed in the right atrial pressure. Thus, treatment by simultaneously performed percutaneous balloon valvuloplasty and ASD closure was planned.

Pulmonary valve was dilated by 4 x20 mm balloon (NuMed, Cornwall, Canada Inc.) (Figure 3). Following balloon dilatation the RV systolic pressure gradient was decreased to 25 mmHg, and aortic O2 saturation increased to 90%. Afterwards, ASD was closed with 26 mm Amplatzer device (St. Jude Medical Corporation, Austin, TX) with guidance of TEE (Figure 4). Following the ASD closure the aortic O2 saturation increased to 96%. After the procedure, a control TTE was performed and it was determined that the peak systolic gradient was 27
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mmHg and mean gradient being 15 mmHg over the pulmonary valve (Figure 5). The functional capacity status of the patient was increased to class II level. The patient was asymptomatic at three months follow-up.

**Discussion**

Treatment of both PD and ASD by transcatheter method is commonly performed. When planning the treatment of ASD, the pulmonary arterial pressure should be considered. The pulmonary arterial pressure is a critical determinant of choice of ASD treatment. When the pulmonary arterial pressure is increased and the shunt is reversed right-to-left, the patient is no longer suitable for surgery and medical therapy is the only choice. But, in the current case the cause of right to left shunt was not increase in pulmonary arterial pressure, the actual cause was presence of severe PD. This was confirmed by the findings of normal pulmonary arterial pressure and increased right ventricular pressure during diagnostic catheterization. Right to left shunt through patent foramen ovale can occur in isolated severe PD. This shunt is among to the therapy indications for PD. The failure of notice of PD at previous evaluation was probably underlying the decision of unsuitability for surgery. There are cases in the literature reporting treatment of ASD and severe PD by transcatheter approach [4]. In some cases both procedures were simultaneously performed while in some others the procedures were performed in a staged manner [5]. The right to left shunt can be decreased even only by pulmonary valvuloplasty [6]. But, in our case the PD was severe and the ASD was not small, so the both repairment was performed simultaneously in one session.

In patients with congenital heart disease the development of right-to-left shunt is considered as a sign of irreversible pulmonary damage [1]; thus, leaving the patient inoperable. But, as in our case, the right-to-left shunt can be secondary to different diseases. Therefore, elucidation of the actual cause of these shunts is of importance. By performing diagnostic catheterization we revealed the cause of the shunt in our case. Additionally, during diagnostic catheterization temporary occlusion of the ASD by balloon will give clue for response to permanent treatment.

**Conclusion**

In conclusion, in concomitant ASD and severe PS both conditions can simultaneously be treated by transcatheter method. Depending on the severity of the PS, right to left shunt can also be developed in these patients. Clarification of the mechanism of the shunt is critical for efficient therapy planning.

**Disclosure of conflict of interest**

None.

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**References**


