

Percutaneous closure of a paravalvular leak after mitral valve replacement and transcatheter aortic valve implantation

Transkater aort kapak implantasyonu ve mitral kapak replasmanından sonra görülen paravalvüler kaçağın perkütan kapatılması

Introduction

Transcatheter closure of a paravalvular leak (PVL) is an attractive alternative in patients at high risk for repeat surgery (1). We report our experience with percutaneous transcatheter closure of a medial PVL in a patient after transcatheter aortic valve implantation (TAVI).

Case Report

A 74-year-old multi-morbid patient (EuroScore of 35%) with was admitted to our hospital with congestive heart failure. Twenty-nine years ago she underwent mitral valve (MV) replacement with a Björk Shiley mechanical prosthesis (Shiley, USA). The transthoracic echocardiography (TOE) revealed a severe aortic valve (AV) stenosis (AV area of 0.8 cm²), a pulmonary hypertension (mean pulmonary artery pressure (PAP) 58 mmHg) and a good left ventricular ejection fraction. There was no dysfunction of the MV prosthesis but a severe paravalvular mitral regurgitation (MR) from medial part of the mitral annulus (Fig. 1A, B, Video 1 and 2. See corresponding video/movie images at www.anakarder.com). As a first step we performed a TAVI via the transfemoral route. A CoreValve® prosthesis (Medtronic CoreValve, USA) was implanted successfully without impingement on the MV. TOE after months showed an unchanged severe MR. We decided for percutaneous closure of the PVL. A steerable Agilis-L sheath (St. Jude Medical Inc., USA) was required after puncture of the posterior part of the atrial septum to facilitate access to the medial part of the mitral annulus (Video 3. See corresponding video/movie images at www.anakarder.com). The PVL was crossed with an Asahi-Miracle 4.5 wire (Abbott Vascular, USA) under support of a right Williams catheter. The Miracle wire was changed for an Amplatzer® superstiff wire (Boston Scientific, USA) and a 4x8 mm Amplatzer® Vascular Plug III occluder (AGA Medical Corporation, USA) was deployed (Fig. 2A, B, Video 4. See corresponding video/movie images at www.anakarder.com). TOE revealed complete closure of the paravalvular leak (PVL) with no residual leak, a good function of the CoreValve® and the prosthetic MV with decrease of the PAP to 15 mmHg (Fig. 1C, D, Video 5. See corresponding video/movie images at www.anakarder.com). Patient recovery was uneventful, with stable results after a follow-up of 6 months.

Discussion

TAVI in patients with AV stenosis in the setting of previous mechanical MV replacement is technically challenging, because of the risk of interference and subsequent functional impairment of the mechanical prosthesis (2). Clinically significant PVL is an uncommon, ranging from 5% to 17% after surgical valve replacement (1). The technique is still challenging due to the lack of specifically engineered instruments for the procedure and requires considerable operator expertise (1, 3). This is the first report of a TAVI and successfully closure of a medial mitral PVL using the steerable Agilis sheath and an Amplatzer Vascular Plug III.

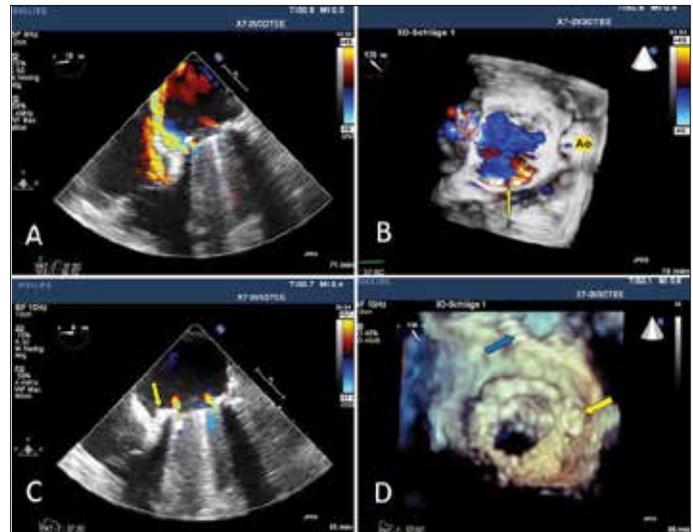


Figure 1. A) 2D-TOE shows a severe eccentric mitral regurgitation. B) Real-time 3-D color Doppler TOE demonstrates a paravalvular mitral regurgitation jet (arrow) originating from medial aspect of the mitral sewing ring (Ao-aorta). C) TOE reveals a completely disappearance of mitral regurgitation following Amplatzer occluder implantation (arrow) for PVL. D) 3D-TOE after successful deployment of Amplatzer Vascular Plug III occluder (yellow arrow). The CoreValve prosthesis is marked with by a blue arrow

TOE - transesophageal echocardiography

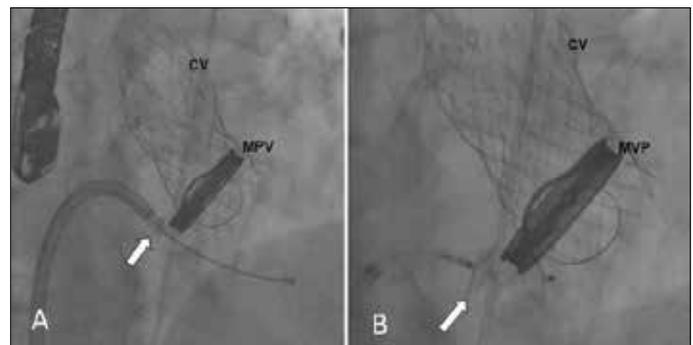


Figure 2. A) Fluoroscopy shows the guidewire (arrow) crossing through the PVL (left anterior oblique projection). B) Fluoroscopy shows the deployment of Amplatzer Vascular Plug III occluder (arrow) into the PVL. CV - CoreValve, MPV - mitral valve prosthesis, PVL - paravalvular leak

Conclusion

With our staged approach, we could demonstrate that TAVI is feasible in high-risk patients with mechanical MV and that mitral PVL closure is possible in patients after TAVI without the risk of disturbing the valve functionality.

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Video 1. Assessment of paravalvular leak (at 6 clock) with 3D-TOE
Video 2. 2D-TOE shows a severe eccentric mitral regurgitation
Video 3. Utility of 3D-TOE in guiding the steerable Agilis-L sheath
Video 4. Fluoroscopy shows the deployment of Amplatzer Vascular Plug III occluder into the paravalvular leak

Video 5. Complete disappearance of mitral regurgitation following Amplatzer occluder implantation

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Available Online Date/Çevrimiçi Yayın Tarihi: 27.05.2013

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Intrauterine idiopathic severe ductal constriction diagnosed by fetal echocardiography: a cause of hydrops fetalis

Intrauterin dönemde fetal ekokardiyografi ile tanı alan ve hidrops fetalise yol açan idiyopatik ciddi duktus darlığı

Introduction

The premature closure or constriction of the ductus arteriosus (DA) is rare and can result in right heart failure, fetal hydrops, persistent pulmonary hypertension and death (1-3). Thus, the recognition of this condition is of vital importance.

We diagnosed idiopathic severe ductus constriction in a fetus whose mother had no risk factors for DA restriction. The case is described with the features of prenatal and postnatal fetal echocardiography.

Case Report

A 29-year-old gravida 2, para 1 woman was referred to our department at 33 weeks of gestation for fetal echocardiography. Obstetric ultrasound examination showed normal fetal growth with a small pleural effusion, and a moderate amount of ascites and polyhydramnios. The patient had no history of medication use during pregnancy, including anti-inflammatory drugs.

Fetal echocardiography ruled out structural cardiac defects; however, it demonstrated a prominent, dilated and hypertrophic right ventricle and an enlarged right atrium and moderate tricuspid regurgitation (Fig. 1 and Video 1. See corresponding video/movie images at www.

anakarder.com). Estimate of right ventricular pressure was 80 mmHg. A small pleural effusion and moderate ascites was also observed. The DA was difficult to visualize, and the flow pattern at the ductus insertion site was consistent with constriction of DA (Video 2. See corresponding video/movie images at www.anakarder.com). Ductal velocity was at least 2.68 m/sec during the systolic phase and 1.57 m/sec during the end-diastolic phase (Fig. 1). The pulsatility index was 0.68. We decide to deliver the baby due to hydrops fetalis, and a cesarean section was performed one day after the mother's examination. A male infant weighing 3.4 kg was delivered at 33+1 weeks of gestation with Apgar scores of 8 and 9 after 1 and 5 min, respectively. The baby was noted to be tachypneic soon after birth and required supplemental oxygen with an FiO₂ of up to 30%; however, mechanical ventilation was not required. The infant was noted to have hydrops with common ascites and subcutaneous edema. An echocardiogram was performed within one hour of birth, which revealed spontaneous closure of the DA. Thus, the prenatal diagnosis was confirmed. A repeat echocardiogram showed a decrease in right ventricular hypertrophy with functional improvement and a marked decrease in the severity of tricuspid regurgitation. There was no risk factor for hydrops fetalis in this infant with the exception of prenatal DA constriction. The neonate was without clinical complications after delivery. The infant's ascites and subcutaneous edema resolved gradually. He was discharged on day 8 weighing 2.9 kg. An echocardiogram performed at this time showed no abnormalities.

Discussion

In utero closure of DA is rare. The patency of DA in utero is largely maintained by high levels of circulating PGE₂ and locally produced PGE₁. Thus, it has been noted that maternal administration of PG synthase inhibitors, such as NSAIDs and corticosteroids, is associated with an increased risk of premature closure of the PDA (4, 5). Another cause of premature PDA closure is the ingestion of polyphenols (i.e., herbal teas, grape and orange derivatives, dark chocolate, berries) (6).

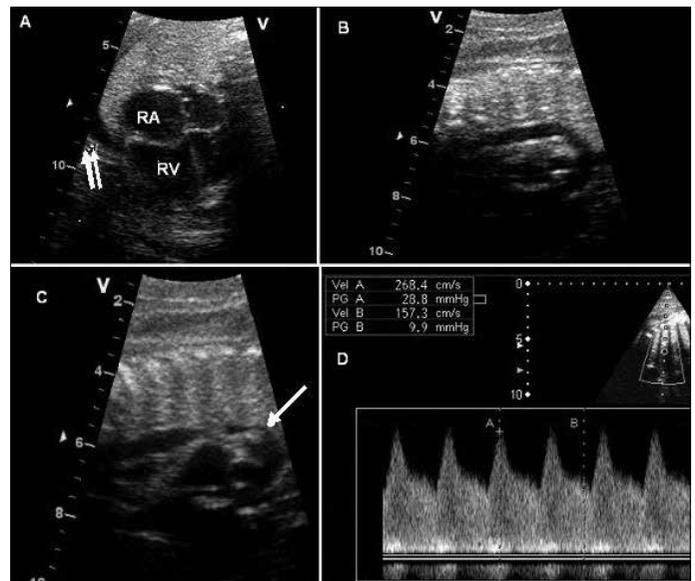


Figure 1. A) The four-chamber view shows marked right ventricular enlargement and hypertrophy as well as right atrial enlargement and a pleural effusion (white double arrow). B) Normal aortic arch in the sagittal view. C) Constriction of the ductus arteriosus in the sagittal view (single arrow). D) Pulsed wave Doppler flow showing a peak velocity of 2.60 m/sec in the systolic phase and 1.5 m/sec in the end-diastolic phase