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Potential Adverse Events: No known potential adverse events.

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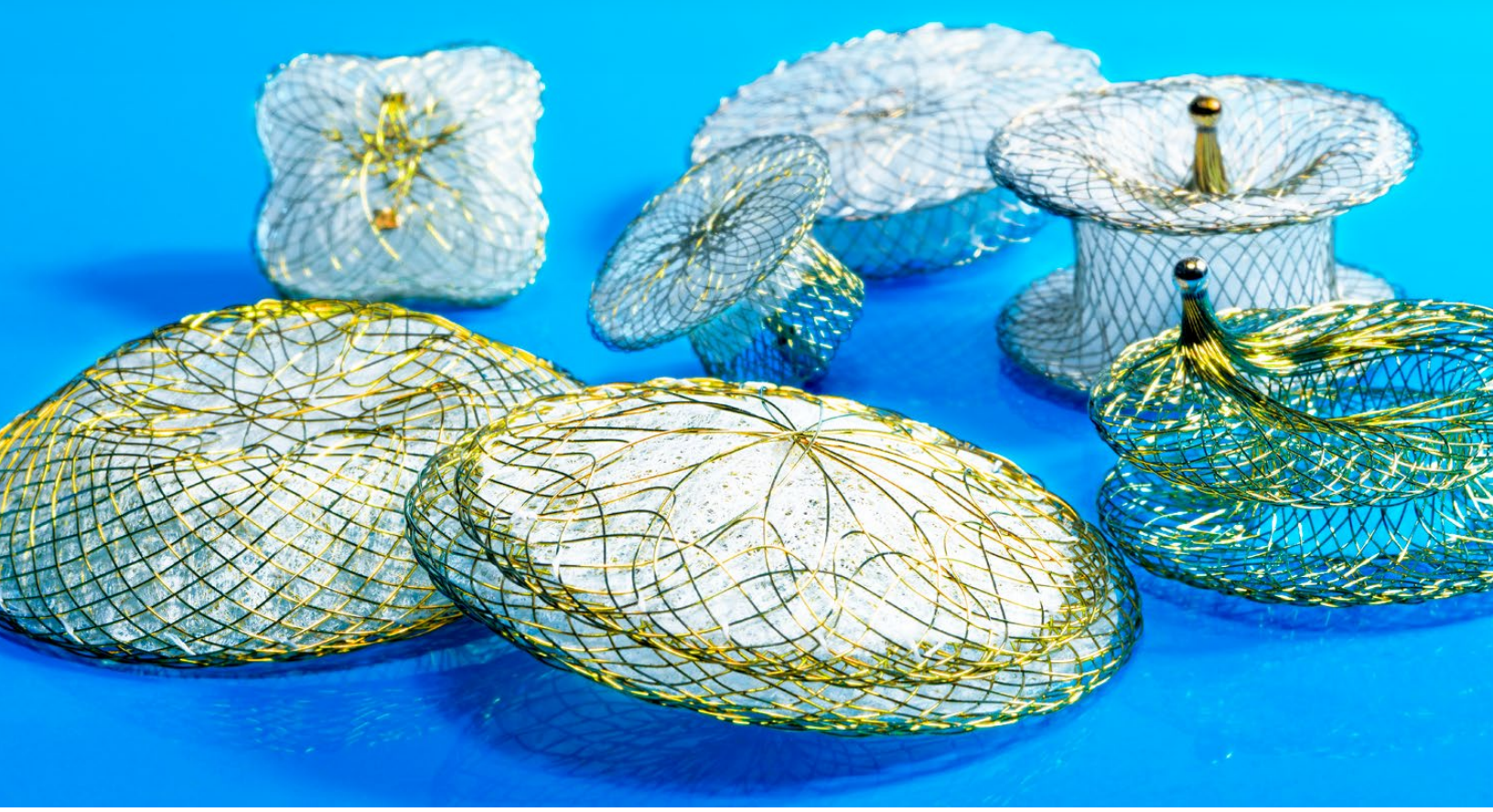

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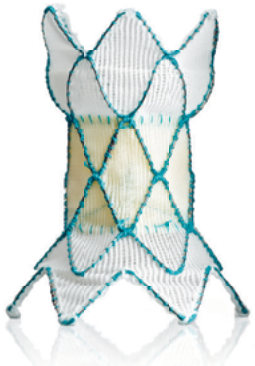

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Transcatheter Pulmonary Valve Replacement: The Venus P valve-Current Status

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Abstract

There is growing appreciation for the long-term adverse impact of right-sided dysfunction of the pulmonary valve in patients with congenital heart disease. Although pulmonary valve stenosis or regurgitation is often tolerated over the short and intermediate terms, the long-term consequences are numerous and include, but are not limited to, right-sided heart failure, arrhythmias, and sudden cardiac death. Surgical right ventricular outflow tract (RVOT) interventions have been performed for many decades as an initial therapy, but comorbidities associated with repeated surgeries are a concern. Transcatheter pulmonary valve replacement is safe, effective, and performed at an increasing number of centers around the world. It offers an alternative to traditional surgical techniques and may potentially alter the decision-making process whereby valvular replacement is performed prior to the development of long-term sequelae of RVOT dysfunction. However, only ~15% of potential patients with RVOT dysfunction are suitable for currently approved implantable valves (i.e., Melody valve from Medtronic and Edwards Sapien valves from Edwards Lifesciences). These two valve systems are designed and approved for patients with a conduit or bioprosthetic valve between the right ventricle and pulmonary artery, and they exclude most patients who undergo transannular patch repair techniques. The Venus P-valve (Venus Medtech, Shanghai, China) is a recently developed self-expanding transcatheter heart valve designed to adapt to a dilated RVOT and in such it provide patients with a percutaneous interventional option after transannular patch repair.

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Key Words

Transcatheter pulmonary valve replacement • Venus P valve-Current status • Congenital heart disease

Introduction

Progress in surgical interventions for congenital heart disease over the past few decades has allowed more children to survive well into adulthood. Most of these patients will require multiple surgical procedures over their lifetime. This can be associated with increased morbidity and mortality [1, 2] due to chest adhesions, bleeding, cardiac ischemia, arrhythmia burden, heart failure, and multi-organ dysfunction [3-7]. Since the introduction of the first balloon-expandable valve in the pulmonary position by Bonhoeffer et al. [8] in 2000, advances in interventional cardiology and transcatheter pulmonary valve replacement (tPVR) have revolutionized the management of these patients. The availability of these minimally invasive and effective therapies may allow for earlier treatment of right ventricular outflow tract (RVOT) dysfunction before the onset of irreversible ventricular remodeling and dysfunction. Moreover, transcatheter options can reduce the need for multiple surgical interventions over a patient's lifetime, thereby minimizing the morbidity of this growing patient popu-

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lation [9]. tPVR is categorized as a class II American Heart Association recommendation for conduits with moderate-to-severe stenosis or regurgitation, provided the patient meets inclusion and exclusion criteria for the available valves [10]. However, only ~15-20% of potential patients with RVOT dysfunction are suitable for currently approved implantable valves [11]. The design and approval of currently available valves (i.e., Melody valve and Edwards Sapien valves) are for patients with a conduit or bioprosthetic valve between the right ventricle (RV) and pulmonary artery (PA), leaving most patients without a percutaneous interventional option after transannular patch repair.

Limitation of Currently Available Devices

Presently, two commercial transcatheter heart valves systems exist: the Melody valve (Medtronic Inc., Minneapolis, Minnesota, USA) and the Edwards Sapien XT valve (Edwards Lifesciences, Irvine, California, USA). The maximum diameter is 22 mm for the Melody valve and 26 mm for the Sapien XT valve [12]. The Edwards valve has evolved over recent years, with an increased range of sizes including the 29-mm Sapien XT and, more recently, the Sapien S3 valve. Both valves are mounted on a balloon-expandable stent platform and require pre-stenting to create an optimal landing zone and reduce the chances of stent fracture or stenosis (for the Melody valve) of the framework after valve implantation [11-13]. For RVOT diameters >26–27 mm or patients with native RVOT post-transannular patch or very expansile RVOT, pre-stenting followed by implantation of these valves is very challenging. Despite these limitations, in the absence of a larger diameter valve, operators have achieved some success of implanting the presently available valves in patients with a native/patched RVOT. Meadows et al. [14] reported on 31 patients with RVOT patch repair who underwent implantation of the Melody valve; at a median follow-up of 15 months, no patient had greater than mild pulmonary regurgitation (PR). Eight patients developed more than mild pulmonary valve obstruction, six of whom experienced stent fractures. Boudjemline et al. described successful implantation of the Melody valve in 13 patients with patched RVOT using technical variations such as implantation of multiple coaxial stents to reduce the diameter (i.e., "Russian doll technique") or anchoring multiple overlapping stents in one PA branch to allow implantation of the valve into the meshwork protruding into the main PA (thereby jailing the opposite PA) [15]. Similar results were reported for the Sapien valve [16]. The 29-mm Sapien XT was recently used successfully for large native or patched RVOT with excellent results [17]. Although several reports of other challenging techniques using the two approved valves describe good immediate hemodynamic results, no data on long-term follow-up are available. Therefore, more recent efforts have concentrated on designing a self-expanding system to provide valve competence despite significant dilation of the native RVOT [18, 19].

Self-Expanding Platform Design (The Venus P-Valve)

The Venus P-valve (Venus Medtech, Shanghai, China) is a recently developed self-expanding transcatheter heart valve designed to adapt to a dilated RVOT [20].

Valve and Delivery System

The Venus P-valve consists of a stent using a Nitinol frame. The valve leaflets are made of porcine pericardium preserved in low-concentration solutions of buffered gluteraldehyde that are hand-sewn to the multi-level self-expanding Nitinol frame. The frame has proximal and distal flares to anchor the valve in the RVOT and PA bifurcation, respectively. The proximal flare is completely covered by pericardial tissue, whereas the distal flare is an open cell wire frame allowing access into the PA branches. The middle part is tubular and straight, fully houses the valve, and is intended to be expanded in the main PA. For ease of identification, there are two radiopaque platinum markers at the proximal and distal flare junctions with the straight segment. The valve is located approximately 5 mm distal to the proximal marker. The diameters and lengths of the straight segment range from 18 to 34 mm (in 2-mm increments) and from 20 to 35 mm (in 5-mm increments), respectively. After cardiac magnetic resonance (CMR) and angiographic evaluation, the valve length can be selected to match the length of the main PA to reduce the possibility of obstruction of the RV body or PA branches and to

reduce paravalvar leak. The proximal and distal flare diameters are 10 mm larger than the diameter of the straight segment. There are two small "ears" at the proximal part of the valve for attachment to the delivery system. The frame is made of a single Nitinol tube by laser-cutting. This design improves frame integrity; however, manufacturing of different sizes to fit patient anatomy is more time-consuming and costly. There are five (previously six) open cells in the distal flared part to allow easy access to the branch PA, with a wire across to decrease the chance of fracture of the distal stent. Figure 1 depicts the current design of the valve. The delivery system (Figure 2) consists of a 20–22-F capsule and a 16-F, 100-cm-long shaft with a rotating handle for deployment of the valve. The valve prosthesis is loaded into the capsule by submerging the Nitinol frame in sterilized cold saline solution and crimping the frame with a crimper provided by the manufacturer.

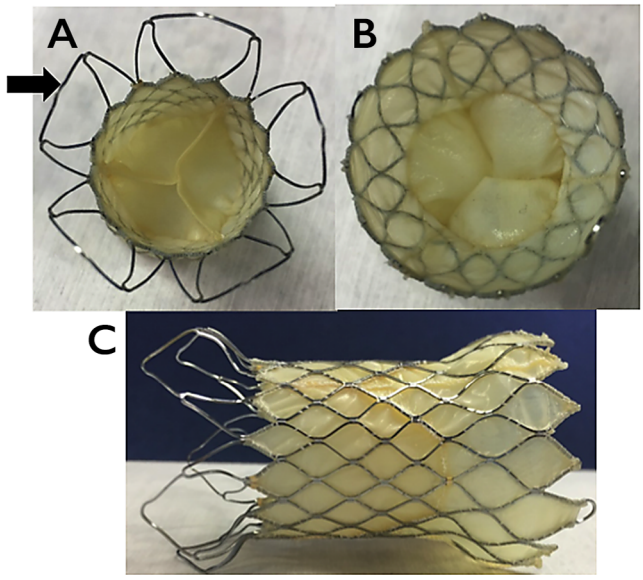

Figure 1. The Venus P-valve Nitinol frame. *Panel A*. Note the new design of the valve with five open cells (**black arrow**). *Panel B*. The valve viewed from below (from the right ventricle outflow portion), with *Panel C* being the straight segment of the valve.

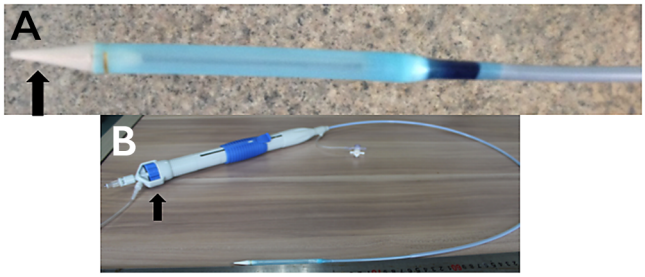

Figure 2. Delivery system with its components. *Panel A*. Carrot at the tip (**arrow**). *Panel B*. Shaft and handle with rotating knob (**arrow**) to allow slow valve deployment.

Pre-Procedural Evaluation

Due to the wide variety of post-operative anatomical variants existing within this group of patients, the most crucial step for Venus P-valve implantation is the initial detailed anatomical assessment of the RVOT. Schievano et al. [21] evaluated variations in postoperative RVOT morphology in 83 patients using CMR, assessing implications for tPVR. Five different morphological subtypes were identified, and although type I morphology (i.e., pyramid-shape) was most commonly seen in those undergoing transannular patch, type II–V morphologies were also seen within this subgroup.

CMR is important for understanding anatomy and initial device size selection. It is advisable to perform cardiac catheterization before valve implantation for better valve selection, especially as different valve sizes may not be available on the shelf. This should be done for complete hemodynamic assessment and measurement of RVOT and PA dimensions. Angiograms in the main PA and RVOT can be done in the antero-posterior or right anterior oblique with cranial angulation and lateral projections. Measurements of the maximum systolic diameter of the RVOT, the main PA at the mid-part and its bifurcation, the maximum systolic diameter of the proximal and distal PA branches, and the length from the RVOT to PA bifurcation can be obtained. Simultaneous selective left coronary angiography (or ascending aorta angiography) and inflation of a sizing balloon in the main PA should be routinely performed to assess expansibility and diameters as well as proximity of the left coronary artery system to the RVOT. Important differences of up to 4.7 mm between CMR and angiographic balloon measurements have been noticed. Undersizing may lead to possible migration of the valve. To prevent valve migration, the implanted valve diameter should be 2–4 mm larger than the maximum diameter of the main PA on balloon interrogation.

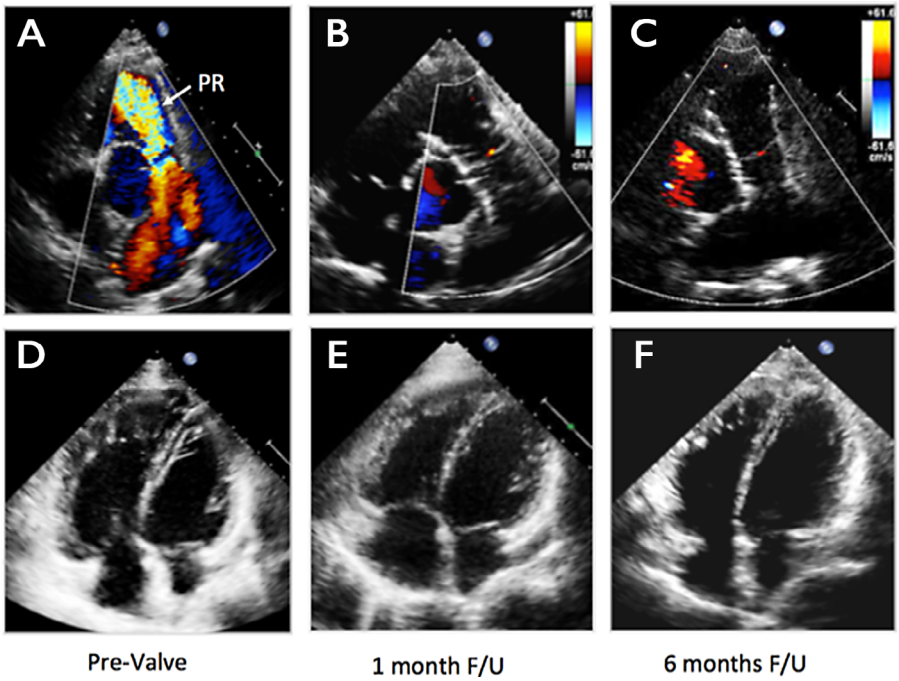

Figure 3. Series of color (*Panels A, B, C*) and two-dimensional transthoracic echocardiography four-chamber view images performed at baseline (*Panels A, D*), 1-month follow-up (*Panels B, E*), and 6-month follow-up (*Panels C, F*) in a 20-year-old female patient after surgical closure of ventricular septal defect and right ventricle outflow obstruction using a patch. She had severe pulmonary regurgitation. These images demonstrate sustained resolution of free pulmonary regurgitation and significant remodeling of the right ventricle. The **white arrow** in the *Panel A* image indicates severe pulmonary regurgitation, and the images in *Panels B and C* demonstrate a competent Venus P-valve (26 mm) with trivial if any regurgitation. The *Panel D* image shows a large right ventricle, and the images in *Panels E and F* show significant remodeling of the right ventricle at 1- and 6-month follow-up, respectively.

Procedure and Follow-Up

It is advisable to perform the procedures under general endotracheal anesthesia. Access should be via both right and left femoral veins (right femoral for device deployment and left for angiographic control), and the femoral artery should also be accessed to assess coronary artery proximity to the RVOT. Heparin 100 U/kg should be administered to maintain activated clotting time of >250 s. Intravenous antibiotics should be given at the beginning of the procedure followed by two doses 8 hours apart. Transesophageal echocardiography (TEE) or intracardiac echocardiography can be performed during the procedure to monitor the RVOT before and after valve implantation. However, this step is optional. Hemodynamic assessment and detailed angiography including balloon sizing can be repeated if needed. A 260-cm long 0.035" Lunderquist extra-stiff guide wire (Cook Medical, Bloomington, Indiana, USA) or any other stiff wire can be positioned in the PA, preferably in the distal left lower PA branch. Intended valve diameter selection should be 2–4 mm larger than the balloon inflation diameter at its waist, whereas mid-body length selection should be equivalent to the distance from the RVOT to PA bifurcation.

After preparation of the valve by rinsing with 2,000 ml normal saline for at least 10 min, the valve is manually crimped in a bath of cold normal saline onto a 20–22-F delivery system. The valve assembly is then passed through a 22-F Check-Flo® Performer Extra-Large Introducer sheath (Cook Medical) and manipulated over the Lunderquist guide wire. The distal carrot tip of the assembly then is advanced into the proximal left PA. Frequent check angiograms through a pigtail catheter placed in the main PA should be done as the distal flare of the valve is slowly deployed by clockwise rotation of the releasing knob. The valve position can be adjusted after check angiograms

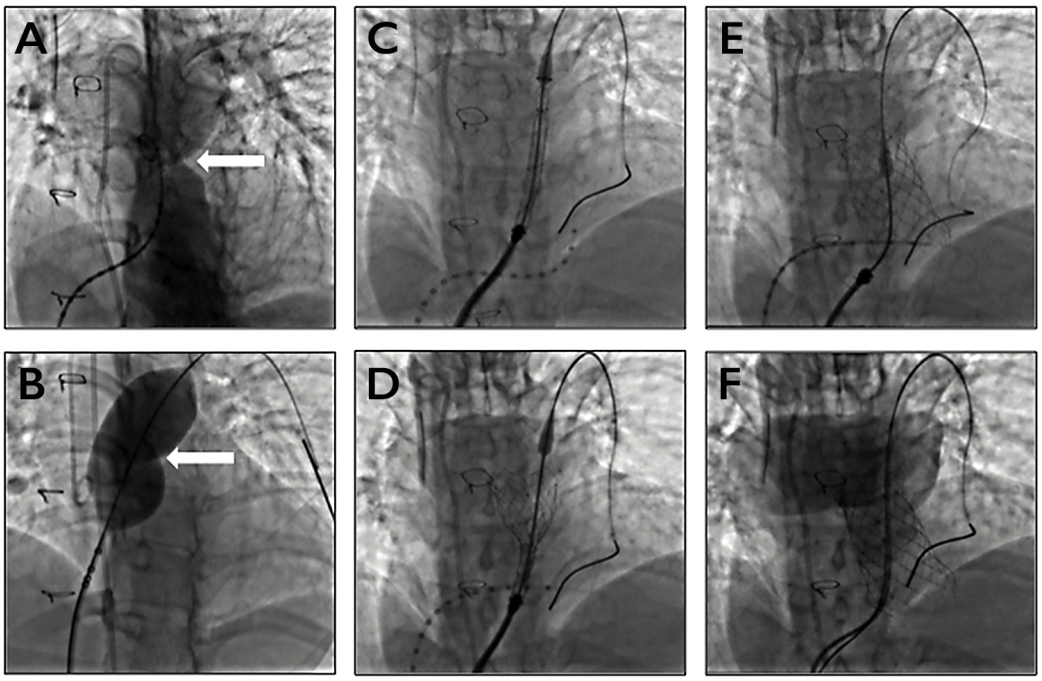

Figure 4. Series of cineangiographic images in frontal projection in a 20-year-old female patient (same as in Figure 3) with severe pulmonary regurgitation. *Panel A*. Angiography of the main pulmonary artery showing severe regurgitation and mild narrowing (**arrow**). *Panel B*. Cinefluoroscopy during balloon sizing and simultaneous ascending aortography showing indentation at the pulmonary annulus (**arrow**) measuring 22–24 mm. *Panel C*. Cinefluoroscopy of the valve assembly in the main pulmonary artery. *Panel D*. Cinefluoroscopy during gradual deployment of a 26-mm Venus P-valve. *Panel E*. Cinefluoroscopy after complete valve deployment. *Panel F*. Cineangiography of the main pulmonary artery showing competent valve.

are performed before it is fully deployed. When the middle segment of the valve is exposed, if it is fully opposed to the main PA, and because this stent segment is fully covered, there may be hypotension or bradycardia from low pulmonary blood flow for a brief period. The valve has to be deployed rapidly to allow normalization of cardiac output. After deployment and release of the valve, RV and PA pressures should be measured and angiography performed in the main PA to assess valve function. Hemostasis of the femoral venous access site can be achieved using direct pressure or a figure-of-eight suture. Alternatively, prior to introducing the large sheath, one or two Perclose ProGlide 6-F Suture-Mediated Closure Systems (Abbott Vascular, Santa Clara, California, USA) may be used [22].

After the procedure, the patient should be monitored in a cardiac intensive care unit or cardiac ward, depending on the hospital system. The patient should receive 81 mg aspirin for 6–12 months. Electrocardiogram (ECG), chest X-ray, and transthoracic echocardiography (TTE) should be performed before discharge. Patients should be followed up in the outpatient clinic at 1-, 3-, and 6-month intervals and yearly thereafter. The follow-up includes clinical evaluation, ECG, chest X-ray, and TTE. In addition, 6 months after the procedure, a CMR can be performed to assess RV volume and performance. Figures 3 and 4 show a patient with severe PR who underwent tPVR using a 26-mm Venus P-valve. Note the sustained competent valve function and remodeling of the RV upon follow-up.

Clinical Experience

The early results with this valve are encouraging. The first clinical human experience using a self-expanding percutaneous stent valve (Venus P-valve) was reported by Cao et al. [20]. In this study, five patients (four females) with a mean weight of 54.9 kg were selected for attempted valve deployment. Patients were NYHA class II (n = 3) or class III (n = 2) at baseline. Pulmonary insufficiency was grade 4 in all

cases, with mean RV end-diastolic volumes (RVEDVi) of 155 ml/m^2 on CMR. Mean minimum "annular" diameter on TTE was 22.8 mm and mean RVOT diameter was 31.8 mm. The valve was successfully implanted in all patients, with implanted valve diameters ranging from 26 to 32 mm. The mean fluoroscopy time (FT) was 22.8 min. Upon mean follow-up of 3.4 months, PR grade was 0 (n = 3) or 1 (n = 2) in all cases. NYHA class improved at least one class in all cases, and RV volumes assessed by TTE normalized in all three patients with follow-up to 3 months. This first cohort of patients is still being followed up and shows continued good valve function up to 3 years post-procedure.

Promphan et al. reported one of the earliest clinical human experiences using the Venus P-valve [23]. In this study, six patients (four males) with a median age of 18.5 years and mean body weight of 53.8 kg were selected. All patients were NYHA class II and had severe PR with mean RVEDVi of 146 ml/m^2 on CMR. The valve was successfully implanted in all patients, with implanted valve diameters ranging from 24 to 32 mm. The mean FT was 29.8 min. No patients had significant RVOT gradient or PR immediately after valve implantation. Only one patient had unexpected mild proximal valve migration to the RV body during withdrawal of the delivery system, causing mild paravalvar leak and significant tricuspid regurgitation. At 6-month follow-up, the median RVEDVi decreased from 146 to 108 ml/m^2. Additionally, the Doppler systolic peak gradient across the valve ranged from 4 to 40 mmHg, there was no evidence of stent fracture on fluoroscopy or structural valve failure, and patients' symptoms improved significantly.

Recently, Husain and colleagues reported their experience implanting the Venus P-valve in Europe [24]. In this study, five patients with a median age of 14 years and mean body weight of 88.4 kg were selected. Two patients were NYHA class II, and three patients were class I. All patients had significant PR with a mean RVEDVi of 131 ml/m^2 on CMR. The valve was successfully implanted in all patients, with implanted valve diameters ranging from 28 to 32 mm. The mean procedure duration was 136.2 min. No patients had significant RVOT gradient or PR immediately after valve implantation. There was no mortality and no major morbidity. Only one patient had jailing of the right PA requiring right PA stent implantation. Post-procedural follow-up (median follow-up 8.5 months, range 3–15 months) with TTE and CMR showed no restenosis or regurgitation with significant improvement in RVEDVi.

In 2017, Garay et al. reported successful implantation of the Venus P-valve in 10 patients (seven female) [25]. The patients' mean age was 32 years and mean weight was 59.6 kg. Seven patients were NYHA class II, and three patients were class III. All patients had moderate to severe PR with mean RVEDVi of 139 ml/m^2 on CMR. The valve was successfully implanted in all patients, with implanted valve diameters ranging from 26 to 32 mm. The mean FT was 29 min. There were no procedure-related complications and no evidence of paravalvular leak in any of the patients. During a mean follow-up of 12 months (range 4–21 months), all patients remained NYHA class I. TTE and CMR 6 months after implantation of the valve showed sustained and significant reduction of PR in all patients. In six patients, the median pulmonary regurgitant fraction was 1% (range 0–5%) and the RVEDVi was 78 ml/m^2 (range 66–100 ml/m^2). No stent fracture was demonstrated on fluoroscopic follow-up at 6 months.

Current Status of the Valve

As of August 2017, the Venus P-valve has been implanted in 110 patients with very good initial results worldwide (China, United Kingdom, India, Thailand, Ireland, Indonesia, Chile, Jordan, Qatar, and Argentina). All patients are undergoing rigorous follow-up protocols, and obtained data will be published in the future. The manufacturer has submitted a protocol to the notified bodies in a few European countries to evaluate the valve in patients after transannular patch repair with severe PR. Data obtained will be used to seek a CE mark approval.

Summary

It is challenging to deal with patients who have undergone tetralogy of Fallot repair using the transannular patch repair technique and who have a larger dilated RVOT that exceeds the size of commercially available balloon expandable valves. Several techniques have been described to adapt these valves for patients with native RVOT; although they show good

immediate hemodynamic effect, there are no long-term follow-up data. In addition, these techniques require extensive pre-stenting. Later generations of the Sapien valve (Sapien XT and Sapien 3) are FDA-approved for use in the aortic position and are being used by multiple centers for off-label implantation in the pulmonary position. Larger diameter Sapien valves (26 and 29 mm) allow for implantation in large dysfunctional native or patched RVOTs [26].

With a self-expanding platform design, the Venus P-valve can conform to a dilated and curved structure of the RVOT. Furthermore, there is no need for pre-stenting to create a landing zone. Initial studies reveal that Venus P-valve implantation is feasible and safe in patients with severe PR after previous correction of tetralogy of Fallot, in whom a transannular patch has been used. The valve is durable in the short-term and has not shown any stent fracture or valve malfunction 6–12 months after implantation. The valve restores early, sustained pulmonary competence with RV remodeling and improved clinical symptoms. Further long-term studies are warranted.

Conflict of Interest

The authors have no conflict of interest relevant to this publication.

Comment on this Article or Ask a Question

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Stroke Prevention With Carotid Compression in Patients Undergoing Transcatheter Aortic Valve Replacement: a Multi-Center Study

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Abstract

***Background*: Transcatheter aortic valve replacement (TAVR) has revolutionized the management of severe aortic stenosis. However, TAVR is associated with several complications, including stroke. Carotid compression has been suggested as a simple maneuver to reduce embolic events during various cardiac procedures. Therefore, we examined the association between carotid compression and the incidence of transient ischemic attack (TIA) and stroke in patients undergoing TAVR.**

***Methods*: This is a retrospective multicenter study of patients who underwent TAVR with and without carotid compression. Primary outcomes were stroke or TIA 72 hours after the procedure and between 72 hours and 30-day follow-up. Data analysis was performed using a propensity score technique with inverse probability weighting.**

***Results*: A total of 306 TAVR patients were included in the study. Group I (n = 188) and II (n = 118) included patients who did not or did undergo carotid compression during TAVR, respectively. The mean age was 82.5 ± 8.2 years in Group I and 78.5 ± 7.6 years in Group II. There was no significant difference in combined stroke or TIA rate 72 hours after the procedure (1.1% vs. 1.9%, P = 0.50) or between 72 hours and 30-day follow-up (2.3% vs 1.6%, P = 0.67).**

***Conclusions*: Carotid compression during the TAVR procedure is not associated with the incidence of composite TIA or stroke after TAVR.**

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Key Words

Transcatheter aortic valve replacement • Carotid compression • Stroke • Transient ischemic attack • Severe aortic stenosis

Introduction

Transcatheter aortic valve replacement (TAVR) has emerged in recent years as an attractive option for treating patients with symptomatic severe aortic stenosis (AS) who are deemed to be at intermediate or high risk for surgical aortic valve replacement (SAVR) [1-3]. As TAVR continues to evolve, it requires improvements in device technology and implantation techniques to minimize the rate of complications such as stroke. Stroke is a devastating complication that has been attributed to procedural factors [4]. The 30-day occurrence of all strokes in the PARTNER 1A tri-

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al was significantly higher among TAVR patients than among SAVR patients (5.5% vs. 2.4%, $P = 0.04$) in the high-risk cohort [1]. Published 5-year outcomes of the PARTNER 1 trial showed a similar incidence of stroke or transient ischemic attack (TIA) between TAVR and SAVR patients (15.9% vs. 14.7%, $P = 0.3$) [5]. More recent data show stroke rates of 3.4% at 30 days in the FRANCE 2 Registry study [6] and procedural/in-hospital stroke rates of 1.8% in the Transcatheter Valve Treatment Sentinel Pilot Registry [7]. Although the incidence of stroke is declining [8], stroke is strongly associated increased morbidity and mortality [9, 10].

The mechanism of stroke after TAVR is presumably embolic in most cases and may differ depending on the timing of stroke and route of access. However, a history of carotid disease may be one predictor of post-procedure strokes [11-13]. Multiple studies utilizing magnetic resonance imaging, trans-cranial Doppler, and filtered and retrieved material have documented nearly universal embolic phenomena during TAVR [14-17]. Hypothetically, stroke prevention can be achieved by minimizing the amount of microemboli showering from the aorta to the brain during TAVR implantation. Anecdotally, several TAVR programs in the United States and Europe perform carotid compression during TAVR to minimize the risk of stroke or TIA [18]. The rationale for this concerns both direct occlusion of the arteries during a period of embolic showering and a change in flow diverting emboli from entering cerebral circulation. Here, we examined the association between carotid compression during TAVR and post-procedural incidence of TIA and stroke.

Materials and Methods

Patient Population

The study included patients with symptomatic severe AS who underwent TAVR at the University of Utah Hospital, Washington University Medical Center, or Banner Health Center in Phoenix, Arizona between February 1, 2012 and April 1, 2016. Collected data were part of the Transcatheter Valve Therapy registry. For the purposes of this study, the intervention of interest was whether the patient received carotid compression during the TAVR procedure as documented in the clinical operative report. Patient eligibility for TAVR was decided at each site by the heart team, which included interventional cardiologists, cardiothoracic surgeons, imaging cardiologists, and cardiac anesthesiologists. Members of the heart teams at all three centers were similarly trained and qualified to treat TAVR patients. Enrolled patients were divided into two groups: Group I included patients who underwent TAVR without carotid compression, and Group II included patients who underwent TAVR with carotid compression. The valve technologies included in the study were SAPIEN XT and S3 (Edward Lifesciences, Irvine, California, USA). To control for any potential differences in pathophysiology related to access approach, only patients who underwent a TAVR procedure via the transfemoral access route were included in this study. Patients whose pre-procedure carotid stenosis status had not been measured were not included in the study, nor were patients with bilateral carotid disease. All patients for whom anticoagulation medication was not contraindicated received dual anti-platelet therapy with aspirin and clopidogrel for 6 months after TAVR. Intravenous heparin was administered during the procedure to obtain an activated clotting time goal of >300 s. Taking into account contraindications, there was no significant difference between groups with regard to the administration of anticoagulation medication at discharge (98.4% for Group I vs. 97.5% for Group II, $P = 0.95$).

At the University of Utah Hospital, it is standard practice to perform carotid compression on patients receiving SAPIEN valves; however, carotid compression is not used at the other two centers, therefore data from these centers served as a control. Carotid compression is defined as bilateral external compression of the carotid arteries and is performed coincident with rapid pacing. In this study, carotid compression was initiated 2 s before rapid pacing and released 3 s after rapid pacing was discontinued, with the average duration of carotid compression lasting 30–45 s. Carotid compression was completed manually with enough pressure to feel the carotid compressed and was completed by the assisting physician. Stroke and TIA were defined according to Valve Academic Research Consortium (VARC) criteria [19]. TIA was defined as a new focal or global neurological deficit with symptom resolution within 24 hours of onset in the

Table 1. Non-adjusted baseline characteristics.

Variable	No Carotid compression *n=188*	Carotid compression *n= 118*	p value
Age	81.9 ± 8.1	78.8 ± 7.3	**<0.001**
Females	48.5%	39.4%	**0.05**
Caucasian race	93.1%	97.1%	0.06
Diabetes	43.5%	40.6%	0.52
Hypertension	96.1%	89.7%	**0.003**
Atrial fibrillation	48.9%	36.6%	**0.008**
Prior Stroke or TIA	23.8%	18.9%	0.20
Prior MI	36.8%	23.4%	**0.002**
Prior CAD (CABG or PCI)	55.1%	49.4%	0.22
Current Tobacco Use	6.9%	7.4%	0.83
COPD	34.4%	27.4%	0.11
Prior PVD	40.4%	30.3%	**0.02**
Carotid Stenosis (L/R)	22.7%	17.1%	0.14
Baseline LVEF (>30%)	93.3%	96.6%	0.13
BMI	28.5 ± 6.9	28.3 ± 6.2	0.80
STS Score	8.47 ± 4.4	9.24 ±5.9	0.12

Table 2. Non-adjusted procedure characteristics.

Variable	No Carotid Compression	Carotid Compression	p value
LOS Post-Procedure (days)	6.8 ± 5.6	6.3 ± 4.8	0.28
Prior aortic valve procedure	18.3%	8.0%	**0.002**
Pre-procedure anticoagulants	43.8%	28.0%	**<0.001**
Access Site • TF • TA + Other	58.7% 41.3%	70.3% 29.7%	**0.01**
Status • Elective • Urgent/ Emergency	85.0% 15.0%	92.6% 7.4%	**0.01**
NYHA Class • Class I • Class II • Class III • Class IV	2.0% 6.0% 46.0% 46.0%	0% 5.7% 81.7% 12.6%	**<0.001**
Valve-in-valve	6.9%	5.7%	0.60
Annulus size	22.4 ± 2.3	23.4 ± 2.7	**<0.001**
Procedure Time (minutes)	141.2 ± 63.2	116.5 ± 63.2	**<0.001**
ICU Hours	54.5 ± 91.8	72.7 ± 90.7	**0.03**

absence of other possible causes and was confirmed by a neurologist. Stroke was defined as rapid onset of a focal or global neurological deficit lasting ≥24 hours (or <24 hours if therapeutic interventions were performed) in the absence of other identifiable non-stroke causes and was confirmed by a neurologist. In keeping with the VARC recommendation, a disabling stroke (also known as a "major stroke") was defined as a stroke with degree of disability ≥2 on the modified Rankin scale. Post-procedure neurologic events ("post-procedure cardiovascular accidents (CVA)") were defined as any TIA or stroke documented within 72 hours of TAVR, whereas a late neurologic event ("late CVA") was defined as any TIA or stroke documented between 72 hours of TAVR (or at discharge) and 30 days after TAVR. In this study, high-volume TAVR is defined as >100 TAVR cases annually, with all three centers meeting this definition. This study was approved by the institutional review boards at all three centers.

Statistical Analysis

Baseline Comparison of Treatment Groups. Continuous variables are presented as mean ± standard deviation (SD) or percentage based on their distribution. In the overall sample, continuous and categorical variables were compared between groups using Student's *t*-tests and Chi-square tests, respectively. Differences were considered statistically significant if $P < 0.05$.

Development of Propensity Score. One difficulty in using observational data is that there may be different distributions of confounding variables between treatment and control groups. Propensity score analysis with inverse probability weighting (IPW) is a method that can help address imbalances between treat-

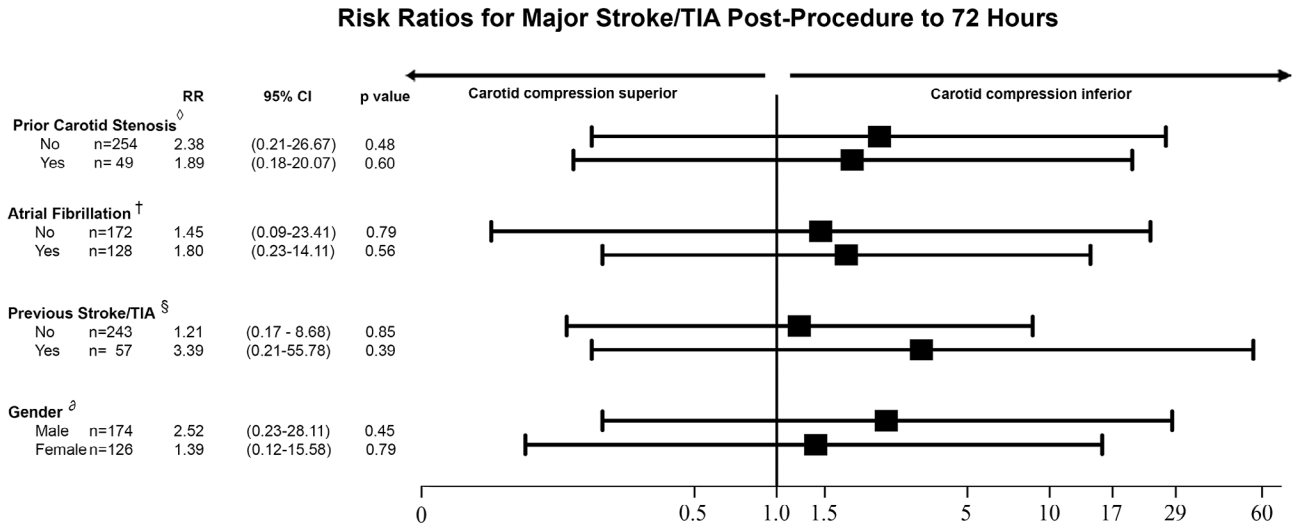

Figure 1. Effects of carotid compression major stroke/TIA post-procedure to 72 hours in subgroups. Carotid compression was shown to be neither superior nor inferior to no carotid compression with regard to combined risk of major stroke or TIA between procedure and 72 hours in subgroups known to have higher risk for stroke or TIA. Due to the low number of overall stroke and TIA events in the population studied, analysis could only be performed on the above subgroups.

◊ Interaction between prior carotid stenosis and carotid compression. p=0.34
† Interaction between atrial fibrillation and carotid compression. p=0.24
§ Interaction between previous stroke/TIA and carotid compression. p=0.38
∂ Interaction between gender and carotid compression. p=0.85

ment and control groups when there is a likelihood of potential selection bias due to nonrandom treatment assignment [20]. As matching is not employed with IPW, this method has the benefit of balancing propensity scores without needing to drop any patients for whom suitable matches cannot be found. A propensity score is the conditional probability of receiving a treatment based on observed potential confounding variables. In this study, the propensity score represents a patient's probability of receiving carotid compression or no carotid compression as a function of baseline and procedural characteristics.

Several covariates that have been linked to the risk of stroke post-TAVR were included in the propensity score model, including new onset of atrial fibrillation, gender, and chronic kidney disease [21]. An initial propensity score model was estimated using 27 variables described in Tables 1 and 2. To estimate propensity scores, logistic regression models were used in which treatment status was regressed on the baseline characteristics listed in Tables 1 and 2. Continuous baseline variables were linearly related to the log-odds of receipt of carotid compression in the initial specification of the propensity score model. Prior research on variable selection for propensity scores suggests that it is desirable to include variables that affect the outcome [22], therefore variables that appeared to impact the outcome were included in the initial propensity score model. Due to our smaller sample size, we chose to remove irrelevant covariates to avoid introducing "noise" into the treatment effect estimates [22]. Propensity scores were modeled as a function of age, pre-procedure hypertension, pre-procedure atrial fibrillation, previous aortic valve procedure, NYHA classification, anticoagulation medication within 24 hours before the TAVR procedure, TAVR procedure time, and pre-procedure aortic valve annulus size. After a propensity score was calculated for each subject, we evaluated the overlap in the range of propensity scores between groups as well as the balance (i.e., distributions) across groups. Although there is no standard guideline for how much imbalance is acceptable, standardized differences for covariates ranging up to 0.25 are acceptable [23]. Balance was achieved with our covariates across groups (Table 3).

Table 3. Covariate balance across control and treatment groups after weighting on the propensity score.

Covariate	No Carotid Compression (standardized mean)	Carotid Compression (standardized mean)	Standardized Difference (%)
Age	80.96	80.29	8.5
Female	0.45	0.45	0.0
Hypertension	0.94	0.93	3.8
Atrial fibrillation - pre-procedure	0.44	0.48	-9.6
COPD	0.31	0.29	5.3
Baseline EF (<30%)	0.05	0.03	11.4
BMI	28.74	28.19	7.9
Chronic Kidney Disease	0.54	0.53	2.5
Prior aortic valve procedure	0.14	0.08	19.9
Anticoagulants pre-procedure	0.40	0.46	-13.3
Procedure time	131.27	133.77	-4.0
Annulus size	22.80	22.65	5.8
Atrial fibrillation – new, post-procedure	0.02	0.00	10.0

Data Analysis. Adjusted risk ratios for post-procedure and late stroke were estimated with 95% confidence intervals (CIs) using a Poisson regression model. All statistical analyses were performed with STATA software (Stata Statistical Software: Release 14, StataCorp, College Station, Texas, USA).

Results

Patient Demographic and Procedural Characteristics

A total of 306 TAVR patients from three high-volume TAVR centers were included in the study. Group I consisted of 188 patients, and Group II consisted of 118 patients. There were 118 patients (38.6%) from the University of Utah, 142 (46.4%) from the University of Washington, and 46 (15.0%) from Banner Health Center. Patient demographic characteristics are shown in Table 1. Mean age was 82.5 ± 8.2 years in Group I and 78.5 ± 7.6 years in Group II. There were more males in both Group I and II (54.3% and 63.6%, respectively). Procedural characteristics are shown in Table 2. The average length of stay was 5.8 ± 4.6 days in Group I and 5.1 ± 3.8 days in Group II.

Unadjusted Results

Before propensity adjustment, post-procedure CVAs were observed in 1.6% of patients in Group I and 2.5% of patients in Group II, with a risk ratio (RR) of 1.59 (95% CI, 0.33–7.78, $P = 0.57$). Late CVA rates were 2.7% in Group I and 2.5% in Group II, with a RR of 0.96 (CI 95%, 0.23–3.94, $P = 0.95$).

Adjusted Results

After propensity adjustment, post-procedure CVAs were observed in 1.1% of patients in Group I and 1.9% of patients in Group II, with a RR of 1.77 (95% CI, 0.34–9.39, $P = 0.50$). Late CVA rates were 2.3% for Group I and 1.6% for Group II, with a RR of 0.72 (95% CI, 0.16–3.18, $P = 0.67$).

Subgroup Analysis

We evaluated the association between specific clinical characteristics and carotid compression with regard to disabling stroke and TIA risk after the procedure. Due to the small number of events, subgroup analysis could be completed only for the following baseline characteristics: carotid stenosis, atrial fibrillation, previous stroke or TIA, and gender. There were no changes in risk of disabling stroke at discharge among analyzed subgroups (Figure 1).

Discussion

We found that patients who received or did not receive carotid compression during TAVR exhibited no significant difference in risk of disabling stroke after the procedure. While the data do not show any benefit in the use of carotid compression, they also do not show evidence of harm associated with carotid compression. It is possible that carotid compression may limit microemboli. The clinical significance, of any, of microemboli detected by transcranial Doppler that do not manifest in overt TIA or stroke is still unknown [24].

TAVR is the gold standard therapy for inoperable patients with symptomatic severe AS [1, 3]. Recently, TAVR has been indicated for intermediate risk patients [2]. The use of TAVR in a wider range of patient populations has paralleled improvements in valve technology aimed at reducing the risk of complications such as vascular injury and stroke [25]. The 30-day stroke rate in the PARTNER 1A trial was significantly higher among patients in the TAVR group than among patients in the medical therapy group (6.7% vs. 1.7%, $P = 0.04$) or the SAVR group (5.5% vs. 2.4%, $P = 0.04$) [1, 3]. In the PARTNER 1A trial, 38% of strokes occurred within 2 days and 58% within 30 days in patients undergoing TAVR. Risk factors for stroke in the PARTNER 1A trial were use of TAVR and small native aortic valve area [9]. The risk of post-TAVR stroke in other studies ranged between 1.7% and 8.4% [7, 26, 27]. This wide difference in reported stroke rate in the literature is likely secondary to inconsistencies in the definitions of acute neurological events. However, a systematic review of the literature shows that stroke rates have declined over the last decade as delivery systems have become smaller, the systematic use of heparin has increased, and technical experience has improved [7, 25, 28].

The mechanism of stroke in TAVR is multifactorial but largely thought to be secondary to embolization. This may be in part due to liberation of calcific material during valve implantation. Other factors contributing to the development of stroke are manipulation of wires and catheters at the level of the aortic arch and root during the transfemoral approach and manipulation of the apex during the transapical approach [29]. Although studies have implicated embolization during the procedure as a potential cause of stroke [30], and bilateral carotid disease is a predictive factor for post-procedure stroke [12], the main source of emboli remains unclear. Clinically silent emboli to the brain have been detected in the majority of patients after TAVR [14, 16, 24, 31]. Several studies have used transcranial Doppler to identify high-intensity transient signal (HITS) as a surrogate for microembolization. Procedural HITS was identified in all patients, with the highest HITS detected for the transfemoral approach with the self-expanding Medtronic Core Valve, mainly during implantation [14, 29, 30]. In our study, neuroimaging testing was not used to determine the impact of carotid compression on the burden of asymptomatic microembolization. The valve design did not seem to alter the risk of stroke after TAVR. The FRANCE 2 Registry showed no statistically significant difference between the balloon-expanding Edwards SAPIEN valve and the self-expanding Core Valve in terms of stroke outcome [32].

The use of carotid compression to influence cerebral embolization during cardiac procedures is controversial, with little published data in support of this practice. Asahi et al. [33] used magnetic resonance angiography to examine the effects of unilateral carotid compression on cerebral flow patterns in two human volunteers. They demonstrated clear changes in perfusion patterns and flow directions within the cerebral vasculature that recovered with decompression. Hillebrand et al. [18] studied 20 patients undergoing a variety of open cardiac surgeries and performed transcranial Doppler of the middle cerebral artery. They found that digital carotid compression reduced the incidence of cerebral emboli during aortic cannulation and declamping. The lack of efficacy of carotid compression during TAVR in the present study may be explained by the fact that microemboli occur during every step of TAVR, not just during valve deployment, as shown by TCD monitoring [14], although there is clearly a peak during valve positioning and deployment. In addition, there is no consensus on the timing and level of pressure that should be applied to potentially prevent stroke.

Another approach to preventing stroke after TAVR is the use of embolic protection devices. There is evidence to suggest that the use of embolic protection devices is associated with a smaller volume of silent ischemic lesions and a smaller total volume of lesions, but there is no related decrease in clinically relevant strokes [34] and no significant change in neurocognitive function [15, 16, 24].

Although the specific causes of ischemic stroke in TAVR patients have yet to be fully identified, several factors may increase a patient's risk of post-procedure stroke, including chronic kidney disease, new onset atrial fibrillation [11, 21], post-deployment balloon dilation, and pure aortic stenosis without regurgitation [26]. Because stroke increases a patient's risk for mortality [35] and can negatively impact quality of life for patients and their families [36], and there is evidence

that early treatment of ischemic stroke results in better patient outcomes [37], it might be worthwhile to consider post-procedure treatment plans specifically designed for patients determined to be at a higher risk of post-procedure stroke.

Some limitations of this study must be considered. This study is a retrospective analysis of a relatively small number of patients. Although this is a multi-center study, carotid compression was performed at only one of the three study sites, and the overall number of outcome events was small. The study did not involve neuroimaging testing to verify the impact of carotid compression on the burden of clinically silent emboli. Larger cohort trials are needed to validate the results of this study.

In conclusion, stroke prevention by means of the non-invasive technique of using carotid compression during TAVR may not impact the incidence of TIA or disabling stroke after TAVR. Further research into the relationship between carotid disease and post-procedure stroke in TAVR patients is warranted.

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Conflict of Interest

The authors have no conflict of interest relevant to this publication.

Comment on this Article or Ask a Question

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The First Case of Successful Stenting of the Dissection of the Ascending Aorta and the Aortic Arch that Occurred During Surgical Correction of the Supralvular Aortic Stenosis in a Child with the Williams Syndrome

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Abstract

Congenital supravalvular aortic stenosis (SVAS), in vast majority of cases is a manifestation of Williams syndrome. Surgical correction of this pathology is a "gold standard" for treatment in these patients. One of the most dangerouse potential complications in surgical repair of this disease is acute dissection of the ascending aorta, arising in 0.7% of cases. This complication can be attributed to both surgical errors and or due to the anatomical features of the aortic wall. We report on a pediatric patient with Williams syndrome, 2.3 yr, 11.9 kg, who underwent surgical repair for supravalvar aortic stenosis using the Doty technique. Aortic dissection was diagnosed using aortography. This was managed by implanting two Valeo stents.

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Key Words

Williams syndrome • Aortic dissection • Stenting

Introduction

Congenital supravalvular aortic stenosis (SVAS), in the vast majority of cases, is a manifestation of Williams syndrome or other pathological condition associated with mutation of the 7q11.23 gene [1-4]. In patients with Williams syndrome or SVAS, other obstructive vascular lesions are often present such as coarctation of the aorta and peripheral stenoses of the pulmonary arteries. Patients with Williams syndrome also often have a bicuspid aortic valve and mitral valve prolapse, as well as various heart rhythm disturbances [6-8].

SVAS is a complex pathology that may present in a wide variety of clinical and morphological forms. The most common form is aortic lumen obstruction localized directly above the aortic valve [5, 9]. The severity of stenosis is the main factor determining the severity of hemodynamic burden and thus the clinical manifestation of this pathology. In most cases, the clinical picture of the disease manifests during childhood or adolescence, and most patients require surgical correction of the defect, which is performed under cardiopulmonary bypass [1, 10, 11].

Although surgery is safe and successful in most cases, there is the rare potential complication of dissection of the ascending aorta after its surgical reconstruction. This complication can be attributed to both surgical errors and anatomical features of the aortic wall. The incidence of this complication is 0.6% [12-13]. Here, we describe a child with Williams syndrome

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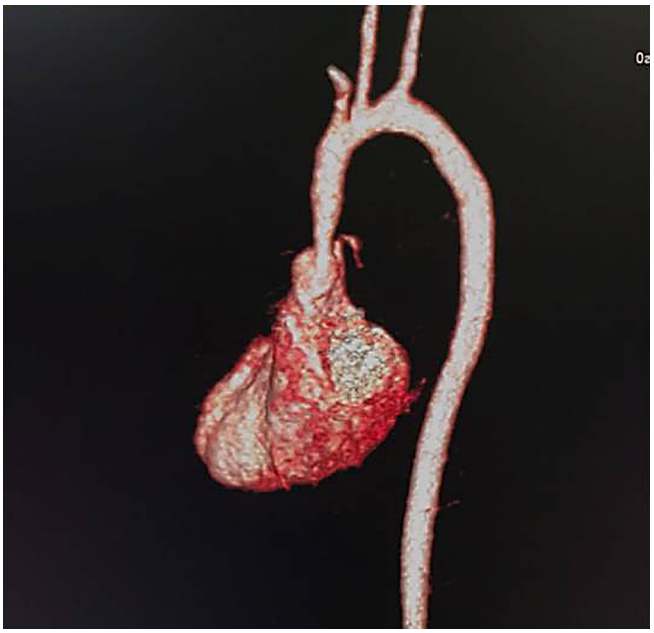

Figure 1. Multi-slice computed tomography of the heart with contrasting. 3D-reconstruction (VRT). A pronounced narrowing of the sinotubular zone of the ascending aorta in a patient with Williams syndrome is visualized.

and SVAS who underwent successful hybrid stenting of an extensive dissection of the ascending aorta extending to the aortic arch.

Case Presentation

Patient K. was 2.25 years and weighed 11.9 kg. He was diagnosed with Williams syndrome with severe SVAS. Echocardiography revealed the presence of concentric hypertrophy of the left ventricle with normal ejection fraction of 68%. The peak gradient across the ascending aorta was 100 mmHg. He also had moderate stenosis of the branch pulmonary arteries, with a peak gradient across the entire right ventricle outflow tract of 11 mmHg. He also had a patent foramen ovale. His echocardiogram showed left axis deviation and left ventricle hypertrophy. Chest X-ray demonstrated cardiomegaly, mainly due to left heart hypertrophy, with a cardiothoracic ratio of 67%.

Multislice computed tomography was performed to clarify the anatomy and determine the localization of the lesion and severity of the stenosis (Figure 1). We observed severe aortic stenosis at the level of the sino-tubular junction measuring 4.5 mm. The isthmus and aortic arch diameters were 6 mm. Brachiocephalic vessels originated from separate origins, with brachocephalic trunk, left common carotid artery, and left subclavial artery diameters of 3.0, 2.8, and 3.8 mm, respectively.

Due to his clinical symptoms of fatigue associated with severe stenosis and left ventricle hypertrophy, we decided to perform surgical repair of the ascending aorta using the Doty technique. Median sternotomy was performed, and under cardiopulmonary bypass with moderate hypothermia (28°C), the ascending aorta was opened via a longitudinal incision toward the non-coronary sinuses of Valsalva almost to the aortic valve annulus. The second incision crossed the stenotic sinotubular zone, forming a reverse "Y" shape in the direction of the right coronary sinus anterior to the intracoronary commissure. Visually, we noticed a thickening of the aortic wall to a diameter of 3.5 mm. A reverse "Y"-shaped xenopericardial patch was made and fixed to the edges of the aortic incision using a premilene suture starting from the right coronary sinus. The suture site was reinforced with medical hemostatic glue [14].

The patient was weaned off bypass without much difficulty. However, there was a significant hemodynamic difference in systolic pressure between the ascending aorta and the radial arteries to the right and left of 110 mm Hg. Dissection of the aorta was suspected. To confirm this, using a mobile angiocardiographic unit (OEC 9900, GE Healthcare, Chicago, IL, USA), we performed ascending aortography using the right femoral arterial access. The systolic pressure gradient between the ascending and descending aorta was 177 mm Hg. Aortography revealed the presence of aortic dissection, with an intimal flap distal to the aortic patch on the ascending aorta with extension to the aortic arch and brachiocephalic vessels (Figure 2).

Given a high risk of surgical correction under cardiopulmonary bypass, a hybrid approach was proposed. We decided to perform stenting of the dissected part of the aorta. A 6-F Mullins sheath (Cook Medical, Bloomington, IN, USA) was placed in the ascending aorta. A standard diagnostic guidewire was exchanged for a 0.035" Amplatz super-stiff guide wire with a 1-cm soft tip (Boston Scientific, Marlborough, MA, USA) to deliver the stent into the ascending aorta so that it would completely cover the zone of dis-

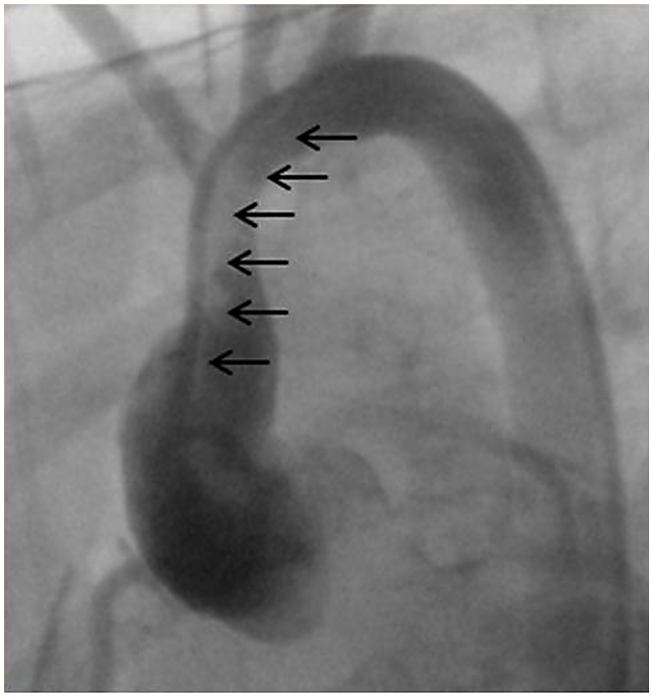

Figure 2. Aortography in the left oblique projection. There is a dissection of the ascending section and the aortic arch (indicated with arrows). The dissection extends to the mouth of the brachiocephalic trunk and the left common carotid artery.

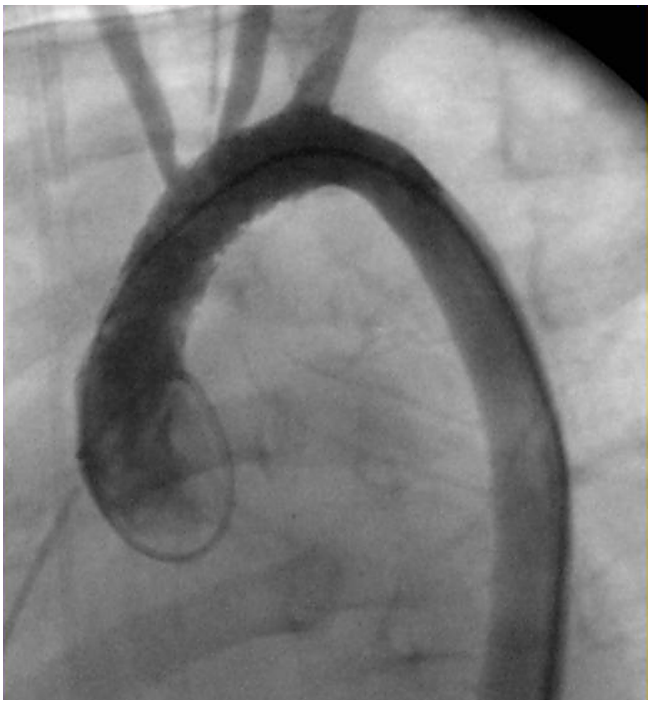

Figure 3. Aortography in the left oblique projection. After the implantation of the two Valeo stents into the ascending aorta and the aortic arch, there are no signs of aortic narrowing and dissection, the brachiocephalic vessels are completely passable.

section to the point of origin of the brachiocephalic vessels. A 18-mm Valeo stent (Bard, Murray Hill, NJ, USA) was attached to a 8-mm balloon deployed to 10 atm. After stent implantation, however, there was no significant change in systolic pressure gradient. Therefore, a second 26-mm Valeo stent on a 8-mm balloon was implanted covering the whole surface of the aortic arch. Repeat aortography revealed proper implantation of both stents covering the entire zone of dissection. Patency of the brachiocephalic arteries was preserved (Figure 3). Invasive pressure measurement after stent implantation showed minimal residual systolic pressure of 15 mm Hg. The child left the operating room on adrenaline at a dose of 0.1 μg/kg/min.

The child was extubated after 20 hours. Anticoagulant therapy was initiated using heparin at a rate of 200 U/kg/day for 3 days followed by aspirin at a dose of 50 mg/day. The patient was discharged home on postoperative day 13. Unfortunately, the parents refused postoperative follow-up consultation in our center due to a stated good clinical status of their child and distant place of residence.

Discussion

Surgical correction of SVAS in patients with Williams syndrome is safe and effective. However, a rare acute complication of dissection of the ascending aorta can lead to an unfavorable prognosis. There are no clear recommendations or algorithms for action in cases of dissection. In adult patients with acute dissection, prosthetic material can be used to repair the dissection. In children, however, this is more difficult and sometimes not feasible. Therefore, in such cases, using endovascular techniques to eliminate acute aortic dissection is potentially a promising solution. We did not find descriptions of similar clinical cases in young children in the literature. Available reports discussed planned endovascular and hybrid interventions to eliminate residual stenosis of the ascending aorta and aortic arch after previously performed reconstructive surgical interventions [15-16].

Despite good immediate results in our case, a future increase in systolic pressure gradient is possible due to growth of the child and neointimal proliferation [15, 16]. This complication often occurs after stenting of vessels, especially in children with Williams syndrome or other genetic defects accompanied by connective tissue dysplasia, which are characterized by a tendency to increased proliferation of the endothelium [17]. However, in our case of hybrid management of an urgent obstruction of the ascending aorta and aortic arch, we used Valeo stents, which have the capacity for further expansion to an adult size if and when needed [18]. Therefore, our case demonstrates the possibility of intraoperative diagnosis and elimination of a formidable complication such as acute aortic dissection using endovascular technologies in pediatric cardiac surgery.

Conflict of Interest

The authors have no conflict of interest relevant to this publication.

Comment on this Article or Ask a Question

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Non-TAVR Bailout for Acute Severe Aortic Regurgitation After Balloon Aortic Valvuloplatsy by Using Fogarty® Catheter

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Abstract

Acute severe aortic regurgitation (AR) after balloon aortic valvuloplasty (BAV), which rarely occurs, can cause catastrophic deterioration of a patient's hemodynamics. The emergent conversion of transcatheter aortic valve replacement (TAVR) or open heart surgery can be considered as bailout procedures for this situation; however, these are not ideal or safe options. We developed a novel bailout method for acute AR without TAVR or surgical conversion. The patient was an 82-year-old woman with severe aortic stenosis who had been treated for interstitial pneumonia for more than 10 years and in whom BAV was scheduled for worsening dyspnea. Through BAV performed using a retrograde approach with an 18-mm balloon, we treated the entrapment of the non-coronary leaflet, the vital sign of which had deteriorated due to severe AR. We attempted to manipulate a pigtail catheter to push back the entrapped leaflet; however, the attempt failed. Next, we advanced a Fogarty catheter into the space between the wall of the sinus of Valsalva and the entrapped non-coronary leaflet with the aim of pushing it back. Inflating the balloon restored the leaflet movement, which successfully reduced AR and obtained hemodynamic stability. This procedure successfully avoided further invasive bailout procedures.

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Key Words

Balloon aortic valvuloplasty • Complication • Acute aortic regurgitation • Bailout procedure • Transcatheter

Introduction

Balloon aortic valvuloplasty (BAV) was the only interventional therapeutic option for patients suffering from severe aortic stenosis (AS) for whom open heart surgery is not indicated. With the emergence of transcatheter aortic valve replacement (TAVR) as an alternative treatment option for such situations, the indications for BAV as a bridging therapy for TAVR have expanded.

Acute aortic regurgitation (AR) is a rare complication of BAV that is reported as to occur in 0.8–1.5% of all procedures [1-2]. This complication is clinically important, as a sudden hemodynamic collapse can lead to a fatal outcome. However, the risk of AR is difficult to predict before BAV, and emergent actions are therefore required. The emergent conversion of TAVR or open heart surgery can be considered as bailout procedures for this situation [3-4]. However, these are not ideal or sufficiently safe, as they require additional manpower, preparation time, and availability of valve devices. Furthermore, it is obviously not adequate for institutions without experience to rapidly switch and

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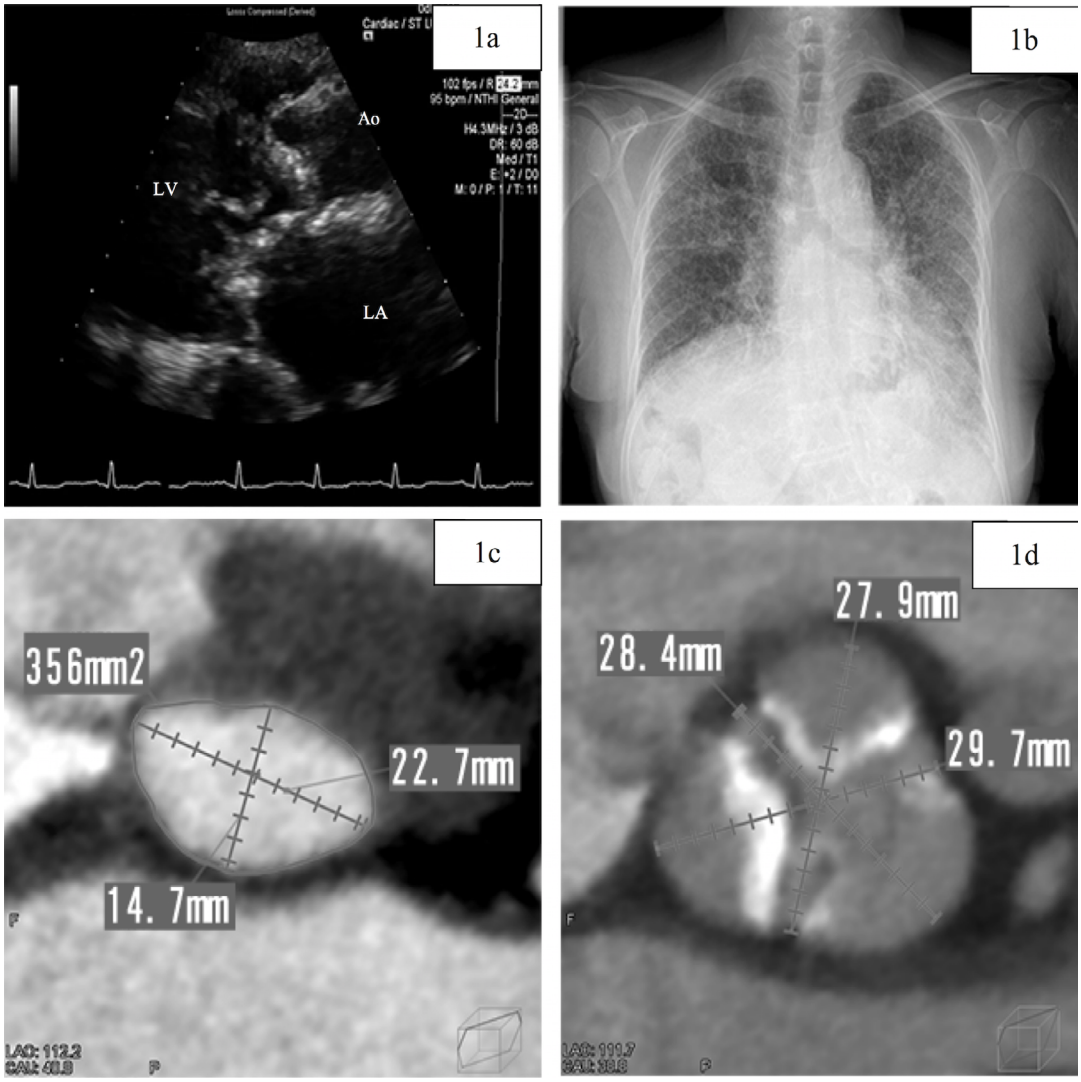


Figure 1. Periprocedural images. *Panel A*. Parasternal transthoracic echocardiogram image obtained in the systolic phase. *Panel B*. Chest X-ray. Axial computed tomography scan of the aortic annulus *(Panel C)* and sinus of Valsalva *(Panel D)*. Ao = aorta; LV = left ventricle; LA = left atrium.

prepare for TAVR [4]. Furthermore, poor outcomes have been reported in cases of emergent conversion to open heart surgery [5]. As such, there is need for a novel quick bailout technique that does not involve greater invasion.

We successfully treated acute AR without TAVR or surgical conversion. A Fogarty catheter was used to push back the entrapped aortic valve leaflet that was the cause of acute AR. This action reduced the severity of the AR from severe to mild, which successfully stabilized the patient's hemodynamics, thereby avoiding intubation or the need for additional TAVR or surgical conversion. We believe this procedure could reduce the need for emergent TAVR or surgical intervention and improve patient outcomes.

Case Presentation

An 82-year-old Japanese woman was referred to our valvular heart disease department due to a heart murmur and a newly developed worsening of dyspnea on effort. Transthoracic echocardiography (TTE) revealed severe aortic valve stenosis, septal bulge, and mild-moderate mitral stenosis. She had been

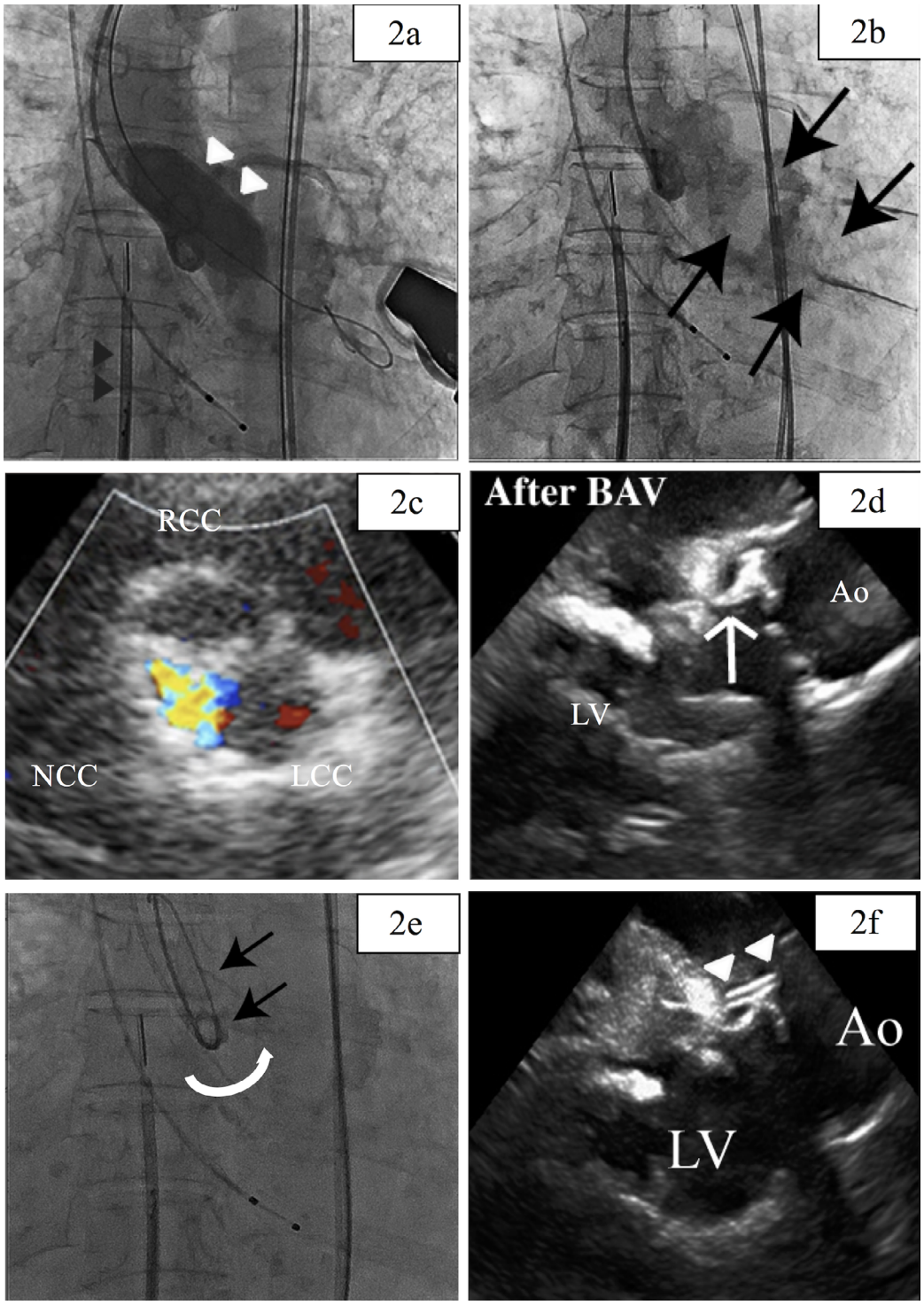


Figure 2. Balloon aortic valvuloplasty (BAV), acute aortic regurgitation (AR), and the bailout procedure with pigtail rotation. *Panel A*. BAV with an 18-mm balloon (**white triangle**). The **black triangle** indicates intracardiac echocardiography (ICE). *Panel B*. Acute severe AR after BAV (**black arrow**). *Panel C*. Transthoracic echocardiogram image of acute AR from the non-coronary cusp. *Panel D*. ICE image of the entrapped non-coronary leaflet (**white arrow**). *Panel E*. Rotating the pigtail catheter (**white arrow**) with a stiff wire (**black arrow**). *Panel F*. ICE image confirming the pigtail positioning at the entrapped non-coronary leaflet (**white triangle**). RCC = right coronary cusp; LCC = left coronary cusp; NCC = non-coronary cusp; Ao = aorta; LV = left ventricle.

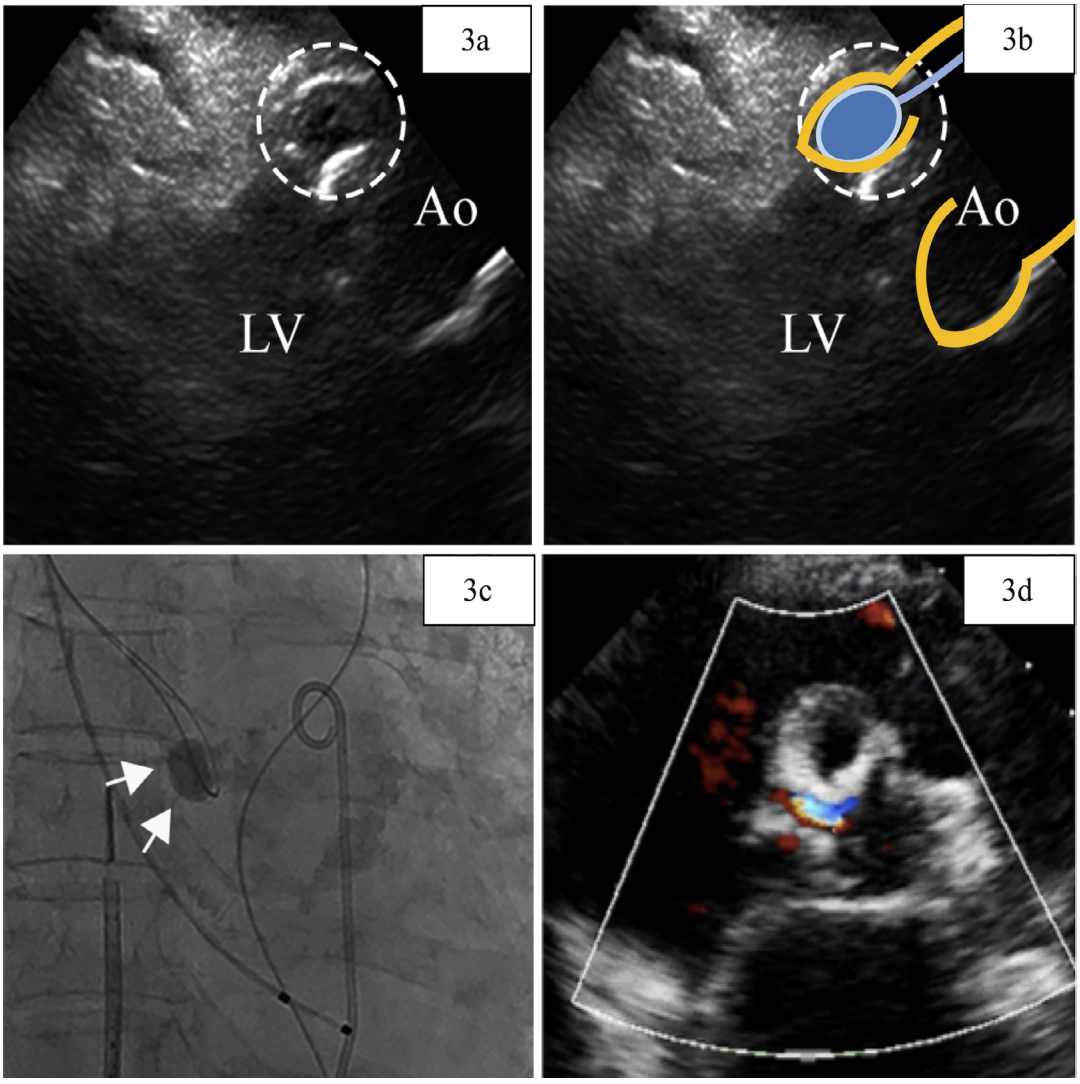
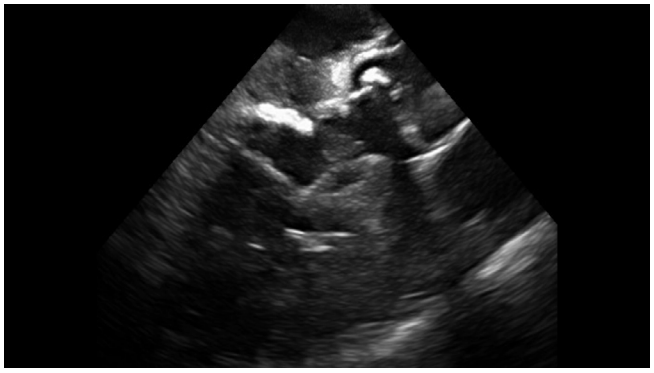


Figure 3. Intracardiac echocardiography image during inflation of the Fogarty catheter *(Panel A)* and a schematic image *(Panel B)*. The **blue circle** indicates the Fogarty catheter. *Panel C*. Inflation of the Fogarty catheter (**white arrow**). *Panel D*. Transthoracic echocardiography image of aortic regurgitation reduction. Ao = aorta; LV = left ventricle.

treated for hypersensitivity pneumonitis and interstitial pneumonia for approximately 10 years with home oxygen therapy and oral steroids. Her STS score was 16.5%. As she had both heart and lung problems, the cause of her symptoms was not clear. After discussion between the heart team and a pulmonologist, BAV was scheduled to confirm whether aortic valve intervention would reduce her symptoms. Preprocedural TTE revealed severe aortic stenosis with a 0.59 cm^2 valve area (planimetry); peak velocity, 3.8m/s; mean pressure gradient, 37.2 mmHg; and max pressure gradient, 59.0 mmHg. No AR, mild-moderate mitral stenosis, or septal bulge were observed. (Figure 1A)

Chest X-ray and lung computed tomography (CT) showed a reticular shadow pattern (Figure 1B). The annular area calculated on cardiac CT was 356 mm^2, with a diameter of 24.6 × 19.7 mm (Figure 1C and 1D).

The BAV procedure was performed under local anesthesia with light sedation controlled by an anesthesiologist. A temporary pacemaker was inserted via the right jugular vein, an 11-F sheath in the left femoral artery, a 5-F sheath in the right femoral artery with a 5-F pigtail catheter seated in the non-coronary



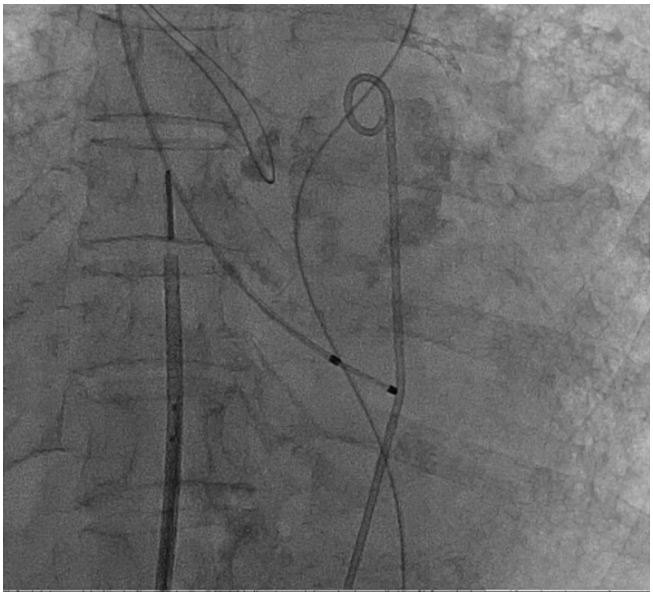
Video 1. Intracardiac echocardiography showing the entrapped non-coronary leaflet. Motion loss and entrapment of the non-coronary leaflet are observed. View supplemental video at https://doi.org/10.12945/j.jshd.2018.038.17.vid.01.

cusp, and a 10-F sheath in the right femoral vein with intracardiac echocardiography (ICE; ViewFlex, St. Jude Medical Japan Co., Ltd, Tokyo, Japan). Heparin was administered with a target activated clotting time of 200–300 s. The contrast media was diluted twice with saline.

The aortic valve was crossed with a 5-F AL1.0 diagnostic catheter (Terumo Medical Corporation, Tokyo, Japan) using a 0.035" Radiforcus straight wire (Terumo Medical Corporation), which was exchanged for a manually shaped Amplatz Super Stiff Wire (Boston Scientific Corporation, Tokyo, Japan). An 18-mm NuMed balloon (Trytech Co., Tokyo, Japan) was inserted into the aortic valve and inflated with rapid pacing (Figure 2A).

Immediately after ballooning, the patient's systolic blood pressure dropped to 60 mmHg, and she complained of chest discomfort. Aortic root angiography revealed severe AR (Figure 2B). ICE and TTE confirmed movement loss and entrapment of the non-coronary leaflet. AR color jet was mainly observed in this area (Figure 2C and 2D and Video 1). The pigtail catheter was inserted into the left ventricle without any resistance; this is referred to as the "free floating pig tail sign". The aortic-left ventricle pressure gradient was almost equivalent after BAV due to entrapment of the non-coronary leaflet. TTE and ICE revealed no pericardial effusion.

We were able to control the patient's blood pressure with catecholamine, and thus she did not require mechanical support. However, because her SpO_2 level dropped and central venous pressure increased, we added noninvasive positive pressure ventilation (NPPV).



Video 2. Cineangiography showing inflation of the Fogarty catheter. View supplemental video at https://doi.org/10.12945/j.jshd.2018.038.17.vid.02.

Our patient was considered unsuitable for emergency TAVR or surgical conversion for several reasons. First, TAVR was not clearly indicated, as we had not yet determined whether AS would be effective for improving the patient's symptoms. Second, as our institution had limited experience with TAVR at that time, we thought that the emergent preparation of TAVR would have the potential to cause further problems. Finally, the patient had a high STS score and lung disease, and surgical intervention was considered to be too risky. Thus, we decided bailout was required without conversion to TAVR or open surgery.

We first attempted mechanical push back using the pigtail catheter. We ensured insertion of the catheter between the entrapped non-coronary leaflet and the wall of the sinus of Valsalva by ICE and then rotated it repeatedly. However, we encountered huge resistance, with no movement of the non-coronary leaflet. We then tried to use a stiff wire in the pigtail to stiffen the catheter itself; however, this was not effective (Figure 2E and 2F).

We needed another option to push the non-coronary leaflet back from its entrapped position. We

attempted to use a Fogarty catheter (Edwards Lifesciences Corporation, Tokyo, Japan) because we thought that a sphere-shaped balloon would fit in the shape of the sinus of Valsalva and the coronary cusp and that it could be advanced into the narrowed space between the entrapped coronary leaflet and the wall of the sinus of Valsalva using an over-the-wire lumen.

A 4-F compatible, 9.0-mm balloon expansion size Fogarty catheter was selected. The 0.035" wire was switched to a 0.025" wire as the balloon was compatible with a 0.025" wire. Tracking with the wire, the Fogarty catheter was successfully advanced and inserted into the slit of the non-coronary leaflet and the wall of the sinus of Valsalva, which was confirmed by ICE and fluoroscopy. The balloon was gently inflated three times for ~10 s each time (Video 2). After the inflation, TTE and ICE showed that non-coronary leaflet movement had been restored and that the degree of AR was reduced to mild. Findings of aortic root angiography were also compatible with AR reduction. The patient was transferred to the intensive care unit without NPPV or catecholamine support.

On postoperative day 1, the patient's respiratory condition worsened, and she required NPPV again; however, right and left heart diagnostic catheterization on postoperative day 2 revealed no increases in pulmonary capillary wedge pressure or left ventricular end-diastolic pressure, which ruled out heart failure with AR. This was therefore diagnosed as an acute worsening of lung disease. The patient gradually recovered and was discharged on postoperative day 15 without further complications. Follow-up TTE showed mild AR; moderate AS with 0.8 cm^2 (planimetry); peak velocity, 3.3 m/s; mean pressure gradient, 26.2 mmHg; and max pressure gradient, 43.9 mmHg.

Discussion

To our knowledge, this is the first case report describing the successful bailout of acute AR after BAV using a Fogarty catheter. Postmortem images of acute AR due to leaflet entrapment after BAV were previously shown by Treasure et al. [7], who reported that malalignment of the irregular surfaces of the fractured calcific nodule entrapped the non-coronary leaflet in an open position and mechanically hindered the ability of the leaflet to close. Hara et al. reported that BAV created intraleaflet fractures within the aortic valve calcified deposit [8], and Mizuno et al. reported a case in which cracks made by BAV were observed in the chunky calcification at the coronary leaflet on postmortem images 6 months after BAV [9]. Based on these reports, we hypothesize that the entrapment in our case might have been caused by the inside-bending of cracked calcified nodular deposits, which locked the leaflet into an open position. ICE in this case clearly showed bending of the non-coronary leaflet.

The Fogarty catheter was suitable for our requirements in the present case for two reasons. First, as a sphere-shaped balloon was required to fit the shape of the wall of the sinus of Valsalva and the coronary cusp, a coronary or endovascular over-the-wire type balloon would not have been appropriate due to their rectangular shape. Second, it was considered difficult to advance the balloon into position accurately; however, this was a necessary aspect of the bailout procedure. An over-the-wire type balloon met these requirements, as this type of balloon can be tracked by the wire and manipulated and inserted into the slit of the entrapped leaflet and the wall of the sinus of Valsalva using a MultiPurpose or Judkins Right catheter, with echocardiography performed as a backup.

We believe that our method can decrease the need for emergency TAVR or surgical conversion, which may decrease further complications of acute AR after BAV. It might be possible to use this Fogarty catheter bailout technique to stabilize a patient's hemodynamics during preparation for TAVR or surgical conversion.

In conclusion, the Fogarty catheter can be safely used for bailout of acute AR caused by an entrapped coronary leaflet after BAV. We believe that using this technique as a bailout procedure can reduce the need for emergency TAVR conversion or surgery.

Acknowledgments

We thank Drs. Osamu Okada and Kohei Abe, who were involved in the treatment of the patient.

Conflict of Interest

The authors have no conflict of interest relevant to this publication.

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Rapid Reduction in Right Atrial Size after Amplatzer Septal Occluder Placement Results in Disc Distortion and Chest Pain Necessitating Device Removal

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Abstract

Patients with a significant shunt from a secundum atrial septal defect (ASD) undergo repair to avoid long-term complications of right ventricular failure, atrial arrhythmias, and pulmonary hypertension. Upon surgical or transcatheter ASD closure, there is an abrupt change in shunt volume, and cardiac remodeling occurs over a period of months to years. Frequently, right-sided dilation does not resolve completely. A 26-year-old man underwent device closure of a large secundum ASD and was observed to have a rapid and profound reduction in right atrial size over an 8-day period. He was asymptomatic for a week following the procedure and then began to experience worsening chest pain. He also experienced various dysrhythmias, including low atrial rhythm with pauses as well as atrial flutter. Echocardiography did not demonstrate pericardial effusion, but electrocardiogram-gated cardiac computed tomography angiography showed the right atrial disc digging into the posterior wall of the right atrium. Despite aggressive medical treatment with non-steroidal anti-inflammatory medication (i.e., opiates and steroids), the patient continued to have daily symptoms. Surgical device removal and ASD repair was carried out 29 days after device placement, which showed no gross evidence of device erosion but that the right atrial disc was compressed into the posterior wall of the right atrium. The patient's symptoms resolved following recovery.

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Key Words

Atrial septal defect • ASD closure • ASD device retrieval • Congenital heart disease

Introduction

We report a case of a rapid and profound change in the right atrial size of a patient who underwent device closure of a secundum atrial septal defect (ASD) using an Amplatzer septal occluder (ASO) resulting in chest pain and rhythm changes. Computed tomography angiography (CTA) showed the right atrial disc "digging" into the posterior wall of the right atrium. Upon surgical exposure of the device, the shape and position of the device was found to be distorted.

Case Presentation

A 26-year-old man with a history of a heart murmur from the age of 13 was referred to our institution for the management of a newly diagnosed secun-

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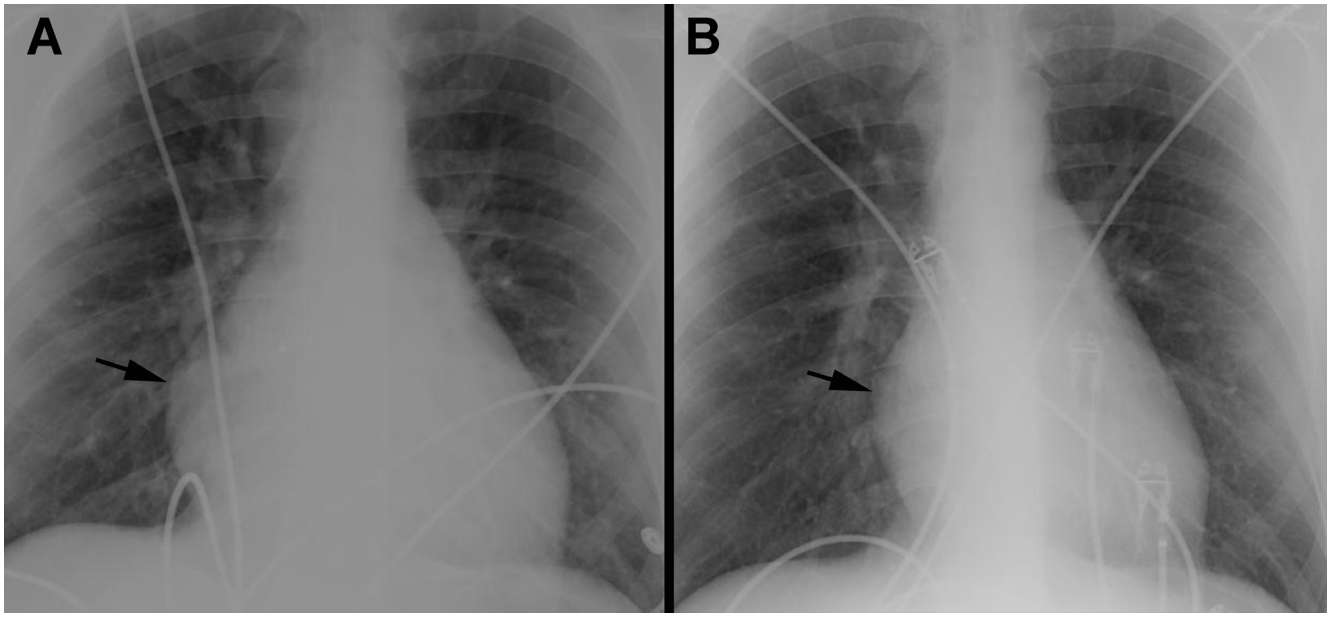
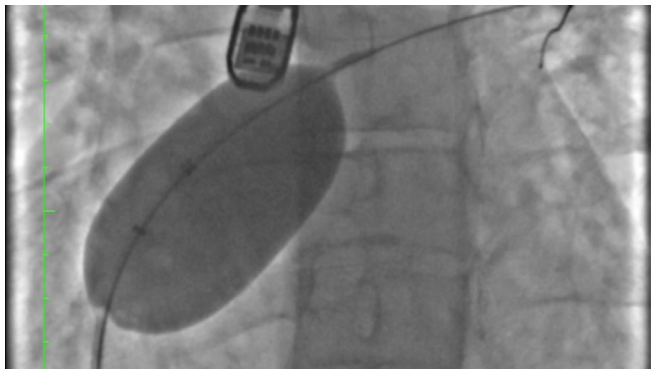


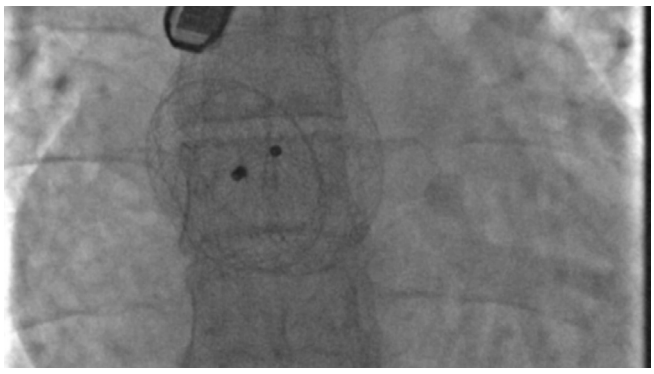
Figure 1. Chest X-rays demonstrating (*Panel A*) significant right atrial enlargement secondary to the patient's large secundum atrial septal defect (*Panel A*) and profound decrease in right atrial size 12 days after device placement (*Panel B*).

dum ASD. The patient initially presented to his primary doctor after a several-month history of decreased exercise tolerance and palpitations. On transthoracic echocardiogram, the patient was noted to have profound right atrial and right ventricular enlargement and a large ASD measuring 1.8 × 2.2 cm. Electrocardiogram (ECG) demonstrated a low atrial rhythm with a rate of 48 beats per minute and an Rsr' in the lead V1. A chest X-ray demonstrated severe right atrial enlargement (Figure 1A).

In the catheterization lab, hemodynamic data demonstrated a normal pulmonary vascular resistance of 0.4 Wood units and a Qp:Qs of 2.7:1. Transesophageal echocardiography showed that the defect measured 30 mm, and the stop-flow technique using a 34-mm-sized balloon did not demonstrate a waist on cineangiogram, so a 38-mm Amplatzer ASO device was chosen. An excellent result was achieved with both imaging modalities. Videos 1 and 2 show balloon-sizing of the defect and rotational angiography of the device in excellent position. The patient



Video 1. Balloon-sizing of the atrial septal defect using a 34-mm sizing balloon. Despite stop-flow, there was no waist on the sizing balloon. View supplemental video at https://doi.org/10.12945/j.jshd.2018.036.17.vid.01.



Video 2. Rotational cineangiogram after device deployment demonstrated excellent positioning of the device. View supplemental video at https://doi.org/10.12945/j.jshd.2018.036.17.vid.02.

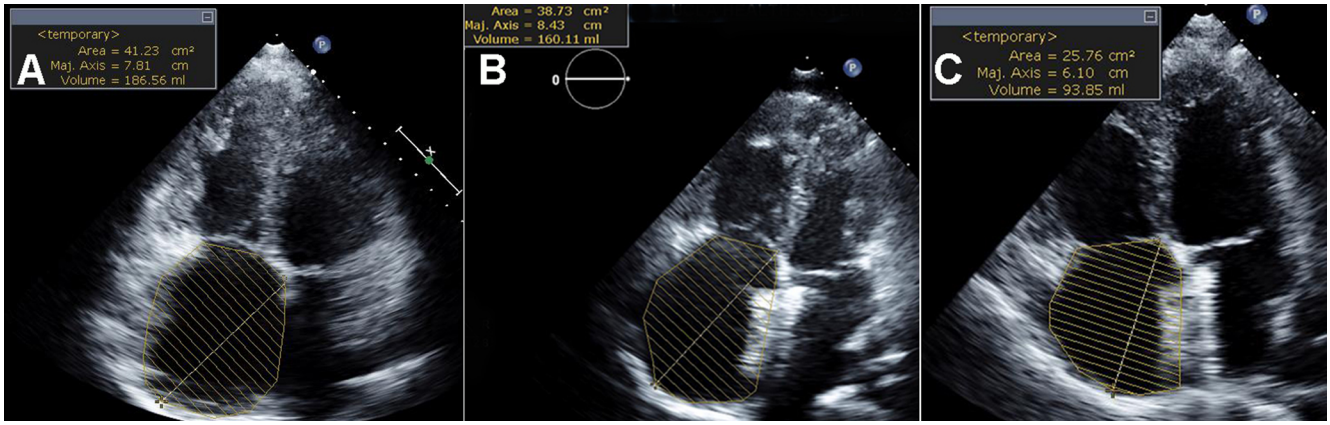


Figure 2. Echocardiogram images demonstrating right atrial volumes of 186 ml before device closure (*Panel A*), 160 ml the day after the procedure (*Panel B*), and 94 ml 8 days after the procedure (*Panel C*).

was discharged the next day after echocardiography showed excellent positioning of the device and no evidence of pericardial effusion.

The patient returned for routine follow-up 8 days later with complaints of palpitations and returned again 12 days later for an urgent visit with continued palpitations as well as chest pain and pressure. Repeat chest X-ray demonstrated a profound decrease in right atrial size (Figure 1B). Review of the patient's transthoracic echocardiogram demonstrated a decrease in right atrial volume from 186 ml pre-procedure to 160 ml the day after the procedure and 94 ml 8 days after the procedure (Figure 2).

Despite aggressive medical treatment with non-steroidal anti-inflammatory medication (i.e., opiates and steroids), he continued to have daily symp-

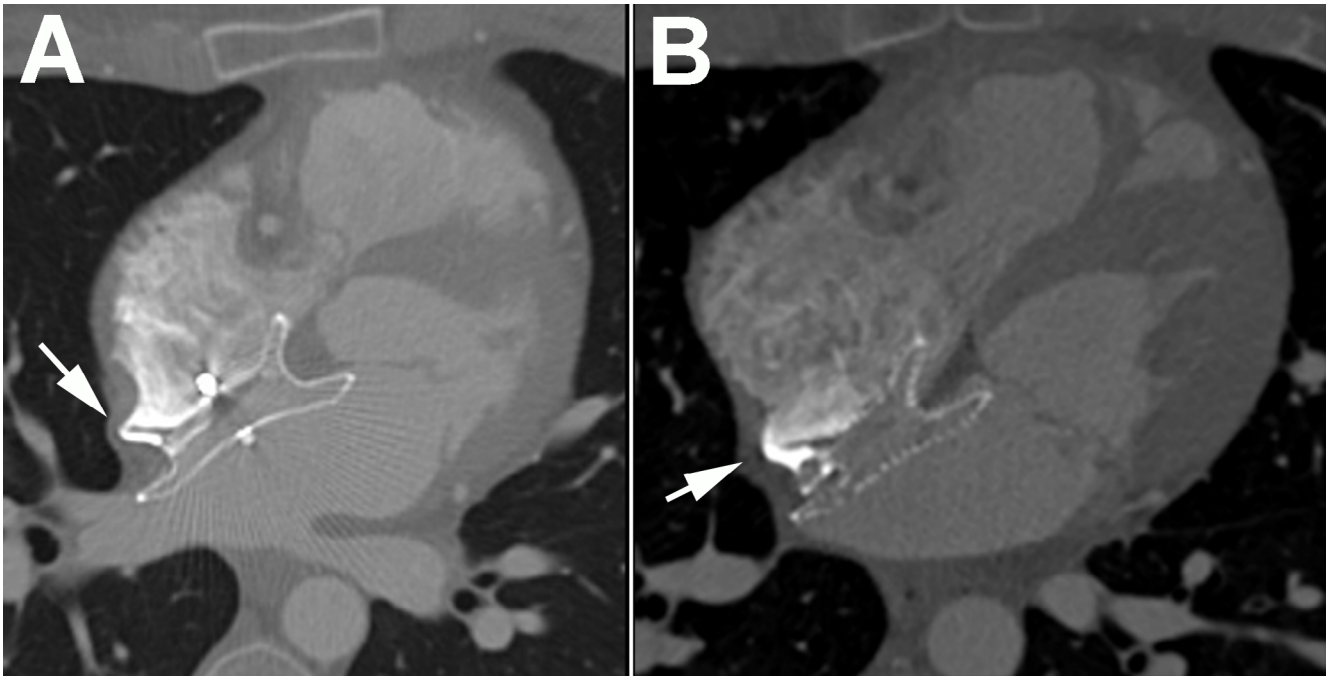


Figure 3. Computed tomography scan demonstrating the right atrial disc "digging" into the posterior wall of the right atrium and distortion of the right atrial wall (**arrow**). *Panel A*. Posterior right atrial wall distortion at the level of the pulmonary veins. *Panel B*. Anterior view with continued right atrial wall distortion (**arrow**).

toms. ECG demonstrated a low atrial rhythm with pauses greater than 2 s and brief runs of atrial flutter. ECG did not demonstrate pericardial effusion, but ECG-gated cardiac CTA showed the right atrial disc "digging" into the posterior wall of the right atrium (Figure 3).

Given his persistent symptoms and rhythm disturbance, we decided that device removal and surgical closure of the ASD was the best course of action. Subsequent explantation of the device demonstrated distortion of the right atrial disk (Figure 4). The patient also had a pacemaker placed for sinus node dysfunction and has had ablations for intraatrial re-entrant tachycardia.

Discussion

Transcatheter closure of secundum ASDs with various occlusion devices has become a standard of care in most pediatric and adult patients due to greater safety and efficacy, shorter recovery duration, and decreased hospitalization time compared with surgical closure [1]. Complications are rare with transcatheter devices, and most complications that require surgical removal and ASD patch closure are secondary to device embolization [2]. Rhythm abnormalities are also reported, with supraventricular tachycardia and atrioventricular block being the most common [1].

Right-sided remodeling following ASD closure remains difficult to assess given the geometric shape of the right atrium and right ventricle. Major deformational and geometrical changes of the right ventricle are completed in 24 hours, but remodeling continues for several months to years [2]. Echocardiography data using strain rate values and tricuspid annular plane systolic excursion suggest an immediate improvement in left ventricular function and decrease in right ventricular function, likely secondary to loading conditions [3-6]. Changes in the size and shape of the atria are not well described.

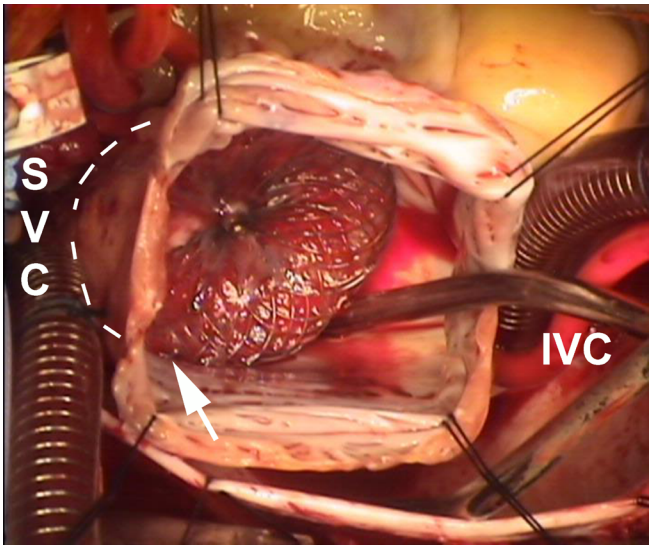


Figure 4. Surgical exposure of the right atrium revealed a distorted right atrial disc. **Dashed lines** demonstrate the course of the device in the right atrium. The **arrow** corresponds to the area of atrial distortion demonstrated on the computed tomography scan in Figure 3. SVC = superior vena cava; IVC = inferior vena cava.

In this case, chest X-ray demonstrated a profound change in right atrial size. We hypothesize that the combination of a large 38-mm ASO device and rapid change in the size of the right atrium caused a significant change in the shape of the device that provoked chest pain and rhythm disturbances. The conformational change was confirmed upon explantation of the device. Therefore, larger devices may have a higher potential to change shape and cause symptoms. Caution should be employed when utilizing larger ASD devices.

Conflict of Interest

The authors have no conflict of interest relevant to this publication.

Comment on this Article or Ask a Question

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