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Potential Adverse Events: No known potential adverse events.

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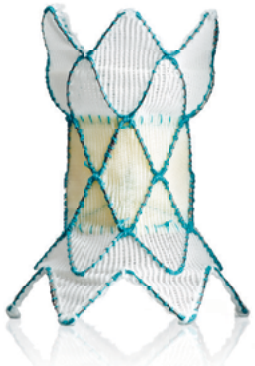

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Percutaneous Balloon Mitral Valvuloplasty in the Elderly

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Abstract

introduction and aim of the work:-mitral stenosis still shows it self older population in developed countries.Percutaneous balloon mitral Valvuloplasty (PBMV) is the treatment of choice for severe and /or hemodynamically significant mitral stenosis. It has been developed as an alternative modality to surgical closed commissurotomy for mitral stenosis5. Elderly patients with mitral stenosis are a special entity who suffered a severe form of the disease and in the same time at higher risk of surgical mitral valve replacement, the aim of the current study is to assess and analyze clinical and echocardiographic consequences after percutaneous balloon mitral Valvuloplasty in those patients. Study design Uncontrolled prospective longitudinal single center study performed in patients referred for percutaneous balloon mitral valvuloplasty. Patients :-The current study included 40 elderly consecutive patients who underwent PBMV in Sohag university hospital cathlab unit. results and conclusion:-There was no in hospital mortality , only one patient developed severe MR , technical failure was encountered in 2 patients, because of failure of the balloon ability to traverse tight valves.This study agreed that PBMV can be applied to those with less favorable valve morphology, PBMV is a safe and effective procedure and optimal results can be achieved in patients with higher wilkin's score if they are carefully selected and operated at experienced centers.

Key Words

Percutaneous • Mitral • Valvulopasty • Elderly

Introduction

The prevalence of mitral stenosis is reflective of the prevalence of rheumatic fever in a particular region [1, 2]. Rheumatic valvular heart disease continues to be endemic in developing countries, with mitral stenosis being the most frequent valve disorder [3]. Although the incidence and severity of rheumatic mitral stenosis has declined in developed countries, the disease is still highly prevalent in many poor and densely populated areas of the globe, remaining a major public health issue and reflecting the socioeconomic status of the region, especially among the elderly. By contrast, mitral stenosis progresses much more rapidly in developing countries and may lead to symptoms in younger patients, including young adults, adolescents, and even children under 5 years of age in some countries [4]. However, mitral stenosis is still pervasive within older populations in developed countries.

Percutaneous balloon mitral valvuloplasty (PBMV), which was developed as an alternative to surgical closed commissurotomy [5], is the treatment of choice for severe and/or hemodynamically significant mitral stenosis. Elderly patients with mitral stenosis are a special population who suffer from a severe form of the disease and at the same time are at higher risk of surgical mitral valve replacement. Therefore, the aim of the current study was to assess the clinical and echocardiographic consequences of PBMV in elderly patients.

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Materials and Methods

Study Design

The study was an uncontrolled, prospective, longitudinal, single-center study of patients referred for PBMV. This study was approved by the Scientific Ethical Committee of Sohag Faculty of Medicine. Informed written consent was obtained from all included patients.

Patients

We recruited 40 consecutive elderly patients who underwent PBMV in Sohag University Hospital. Patients were included if they were over 60 years of age; had symptomatic moderate to severe mitral stenosis, a mitral valve area <1.5 cm^2, and no higher than grade 2/4 mitral regurgitation by echocardiography; refused to undergo surgery; and were considered high risk due to comorbid conditions (e.g., renal insufficiency, chronic pulmonary disorder, liver cirrhosis, malignancy). Patients were excluded if they met any of the following criteria: highly unfavorable mitral valve morphology (i.e., Wilkin's score ≥11), higher than grade 2/4 mitral valve regurgitation, presence of thrombus in the left atrium or left atrial appendage, presence of concomitant valve disease requiring surgical intervention, scheduled for coronary artery bypass surgery, presence of infective endocarditis, interatrial septum thickness >4 mm, or occurrence of cerebrovascular stroke within the previous 3 months.

Procedures

Before and 1 month after the procedure, all patients underwent clinical assessment including New York Heart Association (NYHA) functional classification, paroxysmal or persistent atrial fibrillation, history of previous PBMV or surgical commisurotomy, and history of thromboembolic events. Body mass index, sinus rhythm, atrial fibrillation, and general signs of heart failure were recorded.

Before, immediately after, and 1 month after the procedure, all patients underwent a complete two-dimensional echocardiographic study. Mitral valve area was measured by direct plannimetry of the valve orifice. From the parasternal short-axis view, the smallest area of the valve orifice obtained in early diastole was chosen for analysis. Peak and mean diastolic pressure gradients across the mitral valve were measured from Doppler spectral analysis of diastolic transmitral mitral flow in apical four-chamber view. The degree of mitral regurgitation as determined by pulsed Doppler echocardiography, color-flow Doppler echocardiography mapping, or both was graded as none (0), mild (1), moderate (2), or severe (3 or 4).

Mitral valve morphologic features were categorized according to a semi-quantitative echocardiographic score as described by Wilkin's et al. [6]. Left atrial diameter and size were measured in parasternal long- and short-axis views. Other chamber sizes and valve abnormalities were also assessed.

Transesophageal echocardiographic examination was performed immediately before the procedure using the same cardiac ultrasound machine for all patients. Assessment was performed following standard methods to exclude the presence of thrombi in the left atrium or left atrial appendage and to measure mitral annular diameter and interatrial septal thickness. A blinded observer reviewed each echocardiogram.

PBMV was performed using the Inoue balloon technique. Balloon size was selected according to body surface area considering anatomy (1 to 2 mm smaller in unfavorable cases) and reached after several stepwise inflations. Hemodynamic measurements of the right and left heart, including simultaneous left atrial and left ventricular pressure recordings, were made immediately before and after valvuloplasty. PBMV was defined as successful when the Doppler mitral valve area was

Table 1. Clinical features of studied population.

Features	Statistical analysis
Gender	
Females	80%
Males	20%
Height	
Mean (SD)	159.57 (8.50%)
Median (range)	159 (145-178%)
Rhythm	
Sinus	75%
Atrial fibrillation (AF)	25%

Table 2. Willkin's Score of studied population.

Features	Statistical analysis
Thickness	
1	5%
2	45%
3	50%
Mobility	
1	10%
2	75%
3	15%
Subvalvular	
1	10%
2	50%
3	35%
4	5%
Calcification	
1	5%
2	50%
3	35%
4	5%
Total score	
7	10%
8	20%
9	40%
10	30%

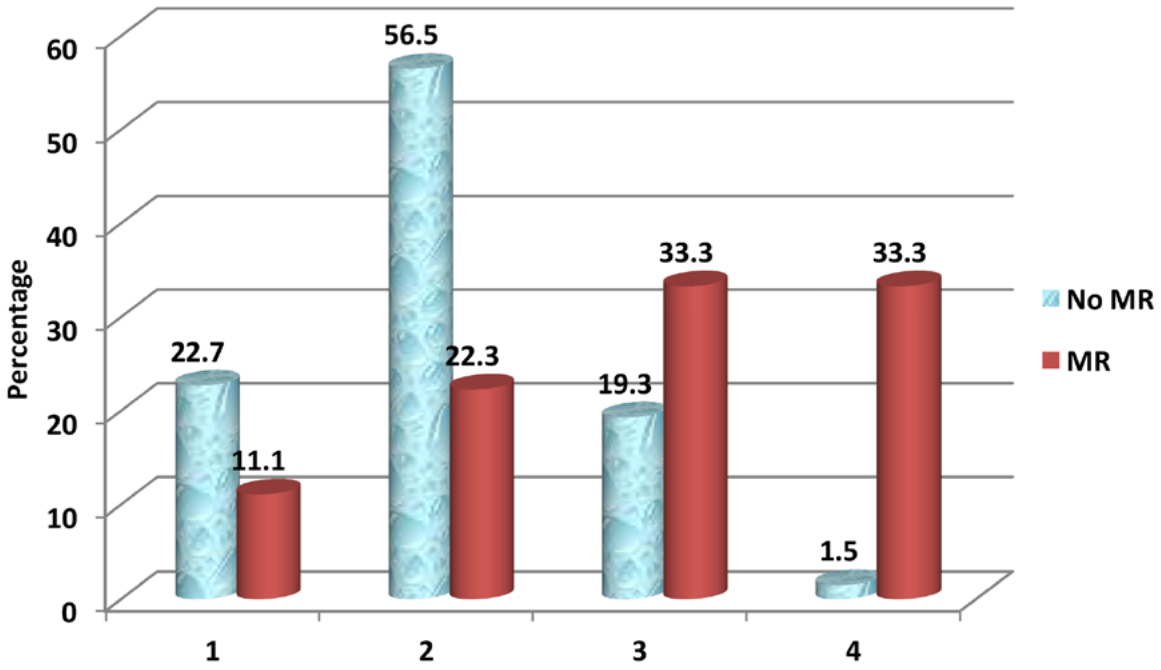

Figure 1. Comparison between those with and without MR as regard W.S. Calcification

≥1.5 cm^2 and there was no more than one grade increase in mitral regurgitation or no increase in gain above 50% of baseline without major complications. Major complications were defined as more than grade 2/4 mitral regurgitation, cerebrovascular stroke, cardiac tamponade, thromboembolic events, or balloon rupture with unwanted sequelae or periprocedural death.

Statistical Analysis

Data were analyzed using STATA/IC version 12.1. Quantitative data are reported as mean and standard deviation. Normally distributed or not normally distributed data were analyzed using Student's t-tests or Mann-Whitney tests, respectively. Qualitative data are reported as frequency and percentage and were analyzed using Chi-square tests or Fisher's exact tests. Statistical significance was set at $p < 0.05$.

Results

Pre-Operative Characteristics

The female-to-male ratio of included patients was 4:1 (Table 1). The predominant symptom before the procedure was heart failure, which was observed in 95% of patients. Symptoms of heart failure were severe (class III or IV) in 65% of patients. A history of cerebrovascular stroke was present in one patient (2.5%). Approximately 60% of patients were categorized as NYHA functional class III, 35% as class II, 4% as class I, and 1% as class IV.

Intraoperative Events and Procedure-Related Complications

Most patients (75%) exhibited sinus rhythm during the procedure, whereas some patients (25%) exhibited atrial fibrillation. Technical failure occurred in two patients due to failure of the balloon to traverse tight valves.

Non-significant increases in mitral regurgitation occurred in three patients, and one patient experienced severe mitral regurgitation that required surgical valve replacement (Table 4). Minor complications included vasovagal reaction in two patients, prolonged hypotension in one patient, and arrhythmia (i.e., multifocal ventricular ectopic beats) requiring treatment in one patient. Bleeding from the right femoral vein associated with balloon catheter insertion occurred in two patients (5%), with neither patient requiring blood transfusion. There was no in-hospital mortality (0%).

Mean mitral regurgitation grade increased significantly from 0.91 ± 0.54 before PBMV to 1.11 ± 0.56 post-dilatation ($p < 0.01$). Before valvuloplasty, 26 patients had no mitral regurgitation, 12 patients had grade 1, two patients had grade 2, and no patients had grade 3 or 4 mitral regurgitation as evaluated by Doppler studies. After valvuloplasty, mitral regurgitation remained unchanged in most patients. However, the degree of mitral regurgitation increased by one grade in four patients (10%): three increased from grade 1 to grade 2, and one increased from grade 2 to grade 3.

When we investigated factors associated with the development of significant mitral regurgitation (i.e., grade 2 or higher), we found a significant increase in the incidence of significant mitral regurgitation among female patients, patients with atrial fibrillation rather than sinus rhythm, and patients who un-

Table 3. General outcomes of studied population.

Statistical analysis	
Outcome	
Success	75%
Failure	25%
New Mitral regurgitation or increased grade	10%
Patients with Significant increase " ≥2+"Mitral regurgitation	2.5%

Table 4. Technical failures and Complications related to percutaneous balloon mitral valvuloplasty.

Complication	(n)	%
Technical failure	2	5%
Vasovagal reaction	2	5%
Prolonged hypotension	1	2.5%
Arrhythmias requiring treatment	1	2.5%
Balloon rupture, no sequaele	0	0%
Bleeding from RT femoral vein	2	5%
Significant hematoma	1	2.5%
Mitral regurgitation	4	10%
Severe mitral regurgitation	1	2.5%
Atrial septal defect	0	0%
Cardiac tamponade	0	0%
Thromboembolism	0	0%
Death	0	0%

derwent redo PBMV or had previous surgical commissurotomy (Table 5). Significant mitral regurgitation was non-significantly associated with a higher Wilkin's score. However, upon further analysis of Wilkin's score parameters, we found that calcification, especially commissural calcification, and higher subvalvular involvement were significantly associated with significant mitral regurgitation ($p < 0.0001$; Table 5 and Figure 1).

Post-Operative Outcomes

We defined significant symptomatic improvement as an improvement by one or more NYHA functional classifications achieved by 1-month follow-up. We observed a significant change in NYHA functional classification from 2.56 ± 0.58 pre-PBMV to 1.09 ± 0.29 post-PBMV ($p < 0.0001$). One patient changed from class IV to II, 10 patients changed from class III to I, 10 patients changed from class II to I, and 15 patients changed from class III to II.

Table 5. Factor affecting occurrence of significant Mitral regurgitation (SMR).

	Significant (≥+2) increase or new SMR		
Features	No	Yes	P value
Gender			
Females	(80.69%)	(55.56%)	0.03
Males	(91.31%)	(44.44%)	
Height	162.28±8.13	165.14±7.01	0.16
L A diameter	5.01±0.49	5.12±0.84	0.41
Rhythm			
Sinus	(77.9%)	(27.7%)	<0.0001
Atrial fibrillation (AF)	(22.1%)	(72.3%)	
Previous BMV/ surgery			
No	(88.97%)	(66.67%)	0.02
Yes	(11.03%)	(33.33%)	
W.S. Thickness			
1	(21.38%)	(16.67%)	0.64
2	(55.17%)	(50.00%)	
3	(23.45%)	(33.33%)	
W.S. Mobility			
1	(27.59%)	(16.67%)	0.37
2	(64.14%)	(66.67%)	
3	(8.28%)	(16.67%)	
W.S. Subvalvular			<0.0001
1	(18.6%)	(22.2%)	
2	(69%)	(16.7%)	
3	(11.7%)	(55.5%)	
4	(0.06%)	(5.6%)	
W.S. Calcification			
1	(22.7%)	(11.1%)	<0.0001
2	(56.5%)	(22.3%)	
3	(19.3%)	(33.3%)	
4	(1.5%)	(33.3%)	
Pre-operative MVA	0.96±0.18	0.90±0.13	0.17
Pre-operative PG	26.34±5.34	25.00±4.32	0.31
Pre-operative PASP	52.95±14.06	51.00±11.07	0.57

L A= Left Atrial; W.S. = Wilkins score; BMV = balloon mitral valvotomy; MVA = mitral valve areas; PG = prostaglandin; PASP = pulmonary artery systolic pressure.

Of the 10 patients with chronic atrial fibrillation, one reverted spontaneously to regular sinus rhythm after successful PBMV.

The primary end-point of procedural success commonly accepted by investigators is a final valve area >1.5 cm^2 without moderate or severe mitral regurgitation. We found that mitral valve area as assessed by planimetry increased significantly in all patients after valvuloplasty. The mean valve area was 0.96

Table 6. Hemodynamic and echocardiographic data of studied population.

Hemodynamic and echocardiographic data	Mean (SD)	P1
MVA		
Pre PBMV	0.96 (0.18)	
Post PBMV	1.93 (0.29)	<0.0001
The transverse diameter of the mitral valve		
Pre PBMV	1.54 (0.37)	
Post PBMV	2.43 (0.96)	<0.0001
The anteroposterior diameter of the Mitral valve		
Pre PBMV	0.75 (0.25)	
Post PBMV	0.93 (0.20)	<0.0001
Mean transmitral Pressure gradient		
Pre PBMV	13.26 (5.27)	
Post PBMV	5.53 (3.15)	<0.0001
Left atrial size	0	0%
Pre PBMV	4.8(0.7)	
Post PBMV	4.2(0.6)	<0.0001
PASP		
Pre PBMV	52.83 (13.85)	
Post PBMV	29.15 (8.94)	<0.0001
Mitral regurgitation		
Pre PBMV	0.91 (0.54)	
Post PBMV	1.11 (0.56)	<0.01

PBMV = Percutaneous Balloon Mitral Valvuloplasty; MVA = mitral valve areas; PASP = pulmonary artery systolic pressure.

± 0.18 cm^2 before valvuloplasty and 1.93 ± 0.29 cm^2 after valvuloplasty ($p < 0.0001$; Table 6). The transverse diameter of the mitral valve was 1.54 ± 0.37 cm before valvuloplasty and 2.43 ± 0.96 cm after valvuloplasty ($p < 0.0001$). Also, trans-mitral diastolic mean pressure gradient decreased from 13.26 ± 5.27 mm Hg before valvuloplasty to 5.53 ± 3.15 mm Hg after valvuloplasty ($p < 0.0001$).

Pulmonary hypertension frequently complicates mitral stenosis and may significantly influence clinical findings and prognosis. The increase in pulmonary arterial pressure is often out of proportion to the degree of left atrial hypertension, which reflects a major increase in pulmonary vascular resistance [28]. In the present study, the average pulmonary artery systolic pressure decreased significantly after 1 month from 52.83 ± 13.85 mm Hg to 29.15 ± 8.94 mm Hg ($p < 0.0001$).

At 1-month follow-up, the left atrium anteroposterior dimension decreased in 85% of patients (from 4.8 ± 0.7 cm to 4.2 ± 0.6 cm; $p < 0.0001$) and remained unchanged in 15% of patients.

Table 7. Factor affecting occurrence of significant Mitral regurgitation (SMR).

Features	Outcome		P value
	Failure	Success	
Gender			
Females	(50.00%)	(86.87%)	0.01
Males	(50.00%)	(13.13%)	
Height	166±7.76	157.24±8.25	0.002
L A diameter	4.88±0.83	4.60±0.49	0.11
Rhythm			
Sinus	(30.00%)	(88.89%)	<0.0001
Atrial fibrillation (AF)	(70.00%)	(11.11%)	
Previous BMV/ surgery	(90.00%)	(89.90%)	0.99
No	(10.00%)	(10.10%)	
Yes			
W.S. Thickness			
1	(20.00%)	(29.29%)	0.78
2	(70.00%)	(58.59%)	
3	(10.00%)	(12.12%)	
W.S. Mobility			
1	(20.00%)	(36.36%)	0.36
2	(70.00%)	(60.61%)	
3	(10.00%)	(3.03%)	
W.S. Subvalvular			
1	(30.00%)	(71.72%)	0.01
2	(40.00%)	(23.23%)	
3	(30.00%)	(5.05%)	
W.S. Calcification			
1	(30.00%)	(62.63 %)	0.051
2	(40.00%)	(30.30 %)	
3	(30.00%)	(7.07%)	
Pre-operative MVA	1.03±0.18	0.96±0.18	0.22
Pre-operative PG	24.1±4.07	26.47±5.36	0.18
Pre-operative PASP	52.2±13.82	52.89±13.92	0.88

L A= Left Atrial; W.S. = Wilkins score; BMV = balloon mitral valvotomy; MVA = mitral valve areas; PG = prostaglandin; PASP = pulmonary artery systolic pressure.

Factors Influencing Procedural Success

Total echocardiographic score was the strongest predictor of procedural success, with lower scores associated with a greater likelihood of a successful outcome (Table 7). Of the different components of Wilkin's score, the presence of subvalvular disease and calcification were most strongly associated with success rate, whereas valvular thickening and mobility were most strongly associated with dilatation.

Discussion

Despite a dramatic decline in the incidence of rheumatic fever, the disease continues to affect many people [7]. Balloon mitral valvotomy, which was first performed in 1982 by Kanji Inoue in Japan, produces excellent results equivalent to those obtained with open or closed surgical valvotomy. In countries with a high prevalence of rheumatic heart disease, such as Egypt, mitral stenosis is a common presentation, and PBMV is a particularly valuable treatment modality. Here, utilizing a prospective, single-center study design, we describe the outcomes of PBMV for elderly patients with mitral stenosis, including those with high Wilkin's scores, relative contraindications, and refusal of valve replacement.

In earlier studies, percutaneous balloon commissurotomy was used in patients with pliable noncalcified mitral valves [2], while more recent studies report the efficacy of balloon commissurotomy even in calcific disease (70% of our patients had Wilkin's scores of 9 or 10) [9]. The results of the present study confirm those of a previous multicenter trial by Inoue et al. [5], which showed that PBMV using the Inoue balloon catheter technique significantly increases mitral valve area in patients with severe mitral stenosis. In association with an increased valve area, we observed significantly reduced mitral valve gradient, left atrial pressure and size, and mean pulmonary artery systolic pressure. These hemodynamic benefits were mirrored by clinical improvements in patients' symptoms and significant downgrades in NYHA functional classification. Therefore, consistent with previous studies [6, 7, 10, 11, 12], our results show that PBMV produces immediate hemodynamic and clinical improvements in most patients.

In our experience, PBMV has a high technical success rate and an encouraging safety record. In the present study, trans-septal catheterization was successful and uncomplicated, and there was no in-hospital mortality. These results are comparable to those from Palacios et al., who reported only one death (3%) and one thromboembolic episode (3%) after valvuloplasty [13]. McKay et al. also reported only one death (2%) and two embolic cerebrovascular accidents (3%) in a large series involving 63 patients, speculating that their success was due to the nature of the Inoue balloon, especially its flow-directed passage from the left atrium to the left ventricle [9]. In the present study, an Inoue balloon also achieved a smooth delivery in most patients.

An increase in mitral regurgitation is one possible complication after percutaneous balloon commissurotomy. However, in most cases, the degree of mitral regurgitation slightly increases after PBMV without requiring surgical intervention. The mechanism of the increase or new appearance of mitral regurgitation is reported to be excessive tearing of the commissures(s) or the posterior/anterior leaflet at the noncommissural part, incomplete closure of a calcified leaflet, localized rupture of the subvalvular apparatus, or shortened chordate tendineae after splitting of the commissure(s). Although the incidence of mitral regurgitation has slightly decreased in the past few years, the appearance or worsening of mitral regurgitation after balloon mitral valvotomy is still a major concern [14, 15, 16, 17, 18]. Although approximately half of patients undergoing balloon mitral valvotomy exhibit a small increase in mitral regurgitation [19, 20], severe mitral regurgitation is relatively rare, with a frequency ranging from 1.4 to 9.4% [13, 21]. There are even some reports of a decrease in mitral regurgitation after balloon mitral valvotomy [22, 23, 24]. In the present study, we observed no change in mitral regurgitation in the majority of patients and severe mitral regurgitation (≥3 grade) in only one patient (2.5%).

We found that elderly patients with a higher calcification score and more subvalvular involvement were more likely to exhibit an increase in mitral regurgitation, whereas total Wilkin's score did not predict the occurrence of mitral regurgitation. This is consistent with the finding of Aslanabadi et al. that calcification is the most important component of Wilkin's classification in that it can predict mitral regurgitation [25]. By contrast, others reported that an increase in mitral regurgitation is not predicted by any valvular or subvalvular apparatus features, patient clinical characteristics, or technical aspects of the procedure [26, 27].

Limitations

Our study has several limitations. The study was performed at a single center with a relatively small sample size, which limits the generalizability of the results to all patients with mitral stenosis. Therefore, multicenter

studies using the same protocol with a larger number of patients are needed. Because all of our patients underwent PBMV with the Inoue balloon technique, it is unknown whether our data can be safely extrapolated to patients undergoing double-balloon PBMV or percutaneous metallic valvotomy. Furthermore, we depended only on Wilkin's score to assess mitral stenosis, which has several limitations, including a limited ability to differentiate nodular fibrosis from calcification, an inability to account for uneven distribution of pathological abnormalities, an inability to assess commissural involvement, and frequent underestimation of subvalvular disease.

Conclusions

Elderly patients with rheumatic heart disease and mitral stenosis show a tendency toward a higher degree of calcification and fibrosis of the mitral valve. This may be attributed to a greater likelihood of repeated episodes of active rheumatic disease, which highlights the importance of administering rheumatic fever prophylaxis to younger patients undergoing PBMV procedures. Our study confirmed that PBMV is a safe and effective procedure that can be used for those with less favorable valve morphology. Without questioning the value of Wilkin's score or its cut-off point, our study shows that successful PBMV can be accomplished in patients with a Wilkin's score between 9 and 10. In conclusion, PBMV and redo PBMV can be employed as a palliative technique in patients with mitral regurgitation at a high risk of morbidity and mortality due to the presence of significant comorbid disease.

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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Successful Percutaneous Device Closure for Giant Atrial Septal Defect with Massive Pericardial Effusion and Pulmonary Hypertension in an Elderly Patient

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Abstract

An 82-year-old man suffering from shortness of breath, leg edema, and appetite loss visited our outpatient clinic. Although he was diagnosed with atrial septal defect (ASD) requiring surgical repair 40 years ago, he had refused an operation at that time. Echocardiography revealed a 37-mm ASD and massive pericardial effusion. Cardiac catheterization showed significant left-to-right shunt flow with Qp/Qs of 4.6 and pulmonary artery pressure of 93/35/52 mmHg. Pulmonary vascular resistance was calculated as 8.3 Wood units. Surgical treatment was no longer indicated due to his condition, and percutaneous treatment with an Amplatzer septal occluder (ASO) was planned instead. Diuretics, a PDE3 inhibitor and nasal oxygen, were administered preoperatively for 1 month. A 38-mm ASO, the maximum occluder size available in Japan, was implanted successfully. No acute decompensation occurred after ASD closure, and the patient's symptoms improved after ASO implantation. Cardiac catheterization on postoperative day 13 revealed no evidence of residual shunt, and pulmonary artery pressure decreased to 63/20/33 mmHg. As postoperative therapy, a PDE5 inhibitor, endothelin receptor blocker, and PGI_2 analog were administered for residual pulmonary hypertension. Because pericardial effusion did not disappear after pericardiocentesis, surgical pericardiostomy was performed 6 months after ASD closure, which reduced PA pressure to 34/16/24. Appropriate pre- and postoperative medical therapy, device closure with an ASO, and pericardiostomy were effective in this frail patient with a giant ASD with pulmonary hypertension and massive pericardial effusion.

Key Words:

Atrial septal defect • Pulmonary hypertension • Catheter • Intervention

Introduction

Atrial septal defect (ASD) is a common congenital heart disease in adults. The main indication for ASD closure is the presence of significant shunting as evidenced by right heart volume overload with or without symptoms [1]. Severe fixed pulmonary hypertension is considered a contraindication for surgical repair of an ASD [2]. However, development of percutaneous closure techniques and medical management may make secundum ASD closure feasible and alleviate symptoms [3].

Case Presentation

An 82-year-old Japanese man suffering from shortness of breath, leg edema, and appetite loss

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visited our outpatient clinic. He was diagnosed at 43 years of age with secundum ASD requiring surgical repair but had rejected treatment at that time. At 76 years of age, he was referred from a community hospital to our institute due to shortness of breath on exertion. By this time, surgical intervention was no longer indicated because echocardiography revealed severe pulmonary hypertension (estimated right ventricular systolic pressure, 102 mmHg). He was treated conservatively with heart failure medications such as furosemide, digoxin, and carvedilol. However, because his symptoms had worsened considerably over the past several years, he was referred to our institute again and hospitalized while alternative treatments were considered.

Although his vital signs were stable (blood pressure, 125/69 mmHg; pulse rate, 69 beats per minute with irregularity), his transcutaneous oxygen saturation ($SatO_2$) in room air had decreased to 95%. During a 6-min walk test, his $SatO_2$ on 1 l/min of nasal oxygen declined to 88%. On physical examination, his jugular veins were dilated, and facial and pretibial edema were observed. Rales were audible in both lower lung fields. His heart sounds revealed fixed splitting of the second heart sound and a systolic murmur at the second intercostal space and left-sternal border. Laboratory data showed a total bilirubin of 1.3 mg/dl and B-type natriuretic peptide of 407 pg/ml. A chest X-ray revealed cardiomegaly, enlargement of the bilateral pulmonary artery, and pleural effusion (Figure 1). An electrocardiogram showed atrial fibrillation and complete right bundle branch block. Transthoracic echocardiography (TTE) revealed enlargement of the right heart, hypoplasty of the left ventricle (Figure 2A), and massive pericardial effusion (Figure 2B). In addition, there was an ostium secundum-type ASD with left-to-right shunting and moderate tricuspid regurgitation on transesophageal echocardiography (TEE) (Figure 2C). The size of the defect and the rims were also measured by TEE. His ASD showed a maximal size of 36 × 37 mm, and each rim was long enough for device closure (Figure 3). Cardiac catheterization revealed Qp/Qs of 4.6, pulmonary artery pressure of 93/35/52 mmHg, and pulmonary vascular resistance

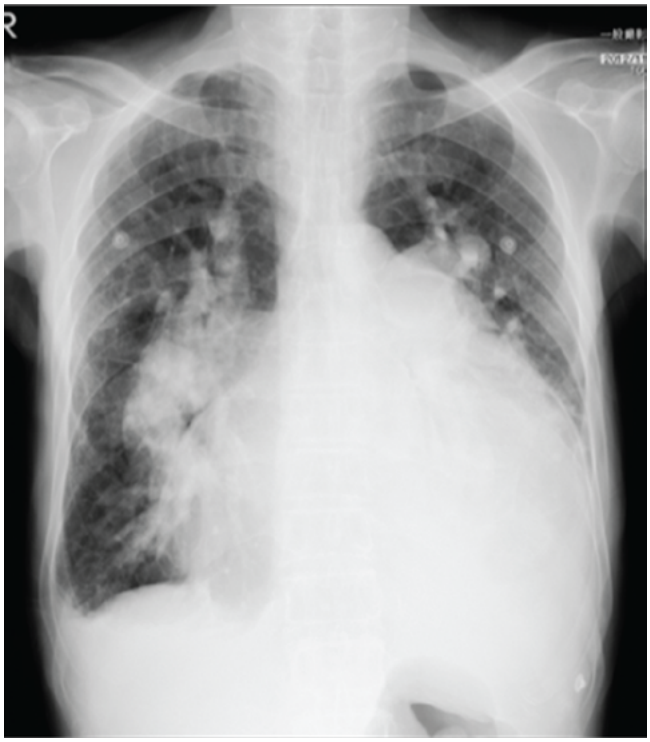

Figure 1. Chest x-ray on admission. Cardiomegaly, dilatation of the pulmonary arteries, and pleural effusion were observed.

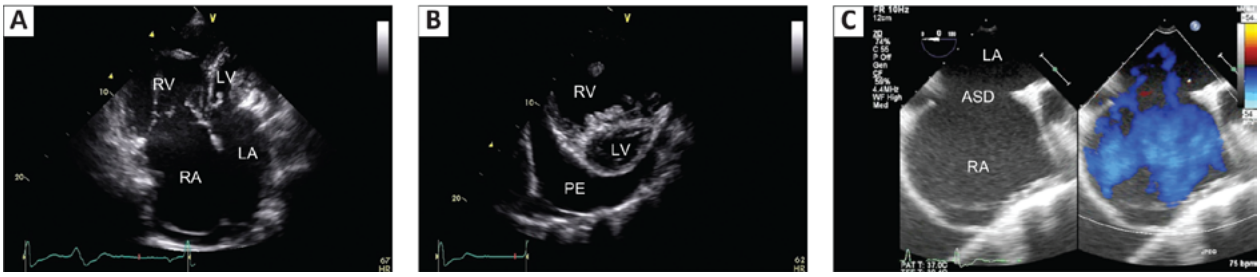

Figure 2. *Panel A.* Four-chamber view of transthoracic echocardiography (TTE). A large right and left atria and ASD were observed. *Panel B.* Short-axis view of TTE. A dilated right ventricle and hypoplastic left ventricle with massive pericardial effusion were observed. *Panel C.* Transesophageal echocardiography findings. A dilated right atrium and left-to-right shunt flow through the ASD were observed. ASD = atrial septal defect; LA = left atrium; LV = left ventricle; PE = pericardial effusion; RA = right atrium; RV = right ventricle.

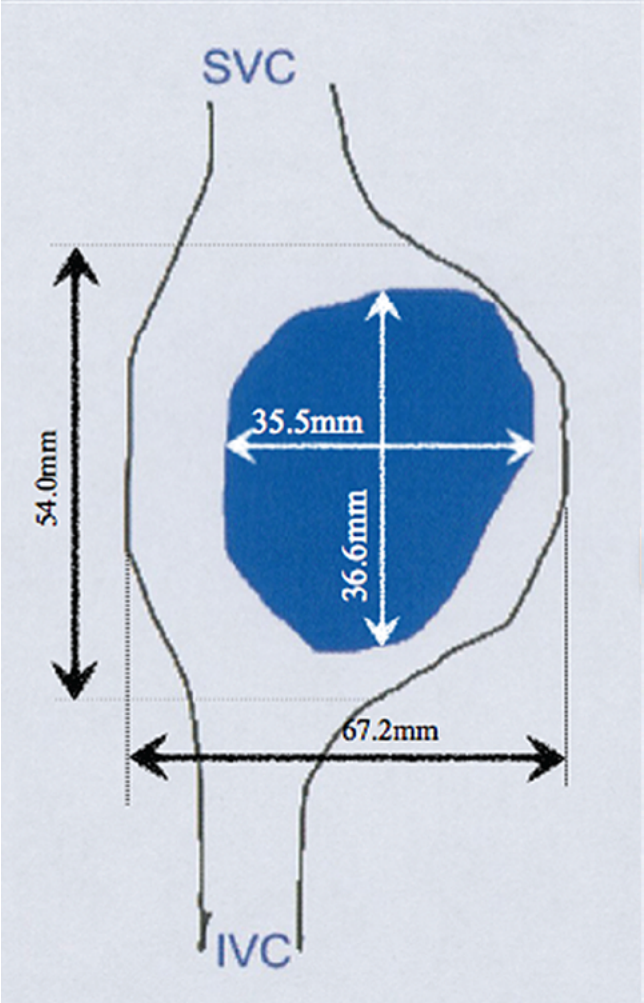

Figure 3. Transthoracic echocardiography findings of atrial septal defect (ASD). The ASD occupies large part of the atrial septum. SVC = Superior vena cava; IVC = Inferior vena cava.

(PVR) of 8.3 Wood units. His PVR index was 5.4 Woods units · m^2, and pulmonary-to-systemic blood pressure ratio was 0.87.

Mainly due to his frailty, percutaneous treatment with an Amplatzer septal occluder (ASO) was planned. Two different diuretics, a PDE3 inhibitor (milrinone 0.2 μg/kin/min) and nasal oxygen, were administered as preoperative therapy in preparation for acute decompensation (Figure 4). After 1 month of medical treatment, we performed percutaneous device closure for atrial septal occlusion. We performed the operation while monitoring intra-cardiac pressure. When we occluded the defect with an ASO temporarily, we observed that pulmonary capillary wedge pressure and left ventricular end diastolic pressure did not rise markedly. Finally, we successfully implanted a 38-mm ASO, which was the maximum size available in Japan at the time (Figure 5). No pulmonary edema occurred after ASO implantation, and the patient's symptoms improved without oxygen administration. When cardiac catheterization was performed on postoperative day 13, there was no evidence of residual shunting, and pulmonary artery pressure had decreased to 63/20/33 mmHg. As pericardial effusion was still present after successful ASD closure, pericardiocentesis was performed, and 1,500 ml of fluid was removed. A PDE5 inhibitor (sildenafil 60 mg), endothelin receptor blocker (ambrisentan 5 mg), and PGI_2 analog (beraprost 120 μg) were administered to treat the remaining pulmonary hypertension. The patient was subsequently discharged from the hospital on postoperative day 41 and returned home on foot.

Three months after ASO implantation, the patient was re-hospitalized due to dyspnea on exertion because pericardial effusion appeared again, even after pericardiocentesis and intensive medical therapy (Figure 6). His pulmonary artery pressure was still high at 47/26/35 mmHg. Therefore, surgical pericardiostomy was performed 6 months after ASD closure. Finally, his left ventricular shape became round and enlarged (Figure 7), and pulmonary artery pressure decreased to 34/16/24 (Figure 6). Finally, he was discharged again and became asymptomatic.

Discussion

We successfully treated a patient with a giant ASD along with extreme pulmonary hypertension and massive pericardial effusion percutaneously with an ASO. Therefore, this case shows that appropriate and timely pre- and postoperative medical therapy combined with ASD closure can be effective.

In patients with significant ASD, left ventricular preload is reduced and a reduction of LV volume is observed over time [4]. In these cases, closure of an ASD with a large left-to-right shunt can increase left ventricular inflow, leading to left ventricular heart failure [5]. To avoid this catastrophic condition, two diuretics and an inotropic agent were administered intravenously for

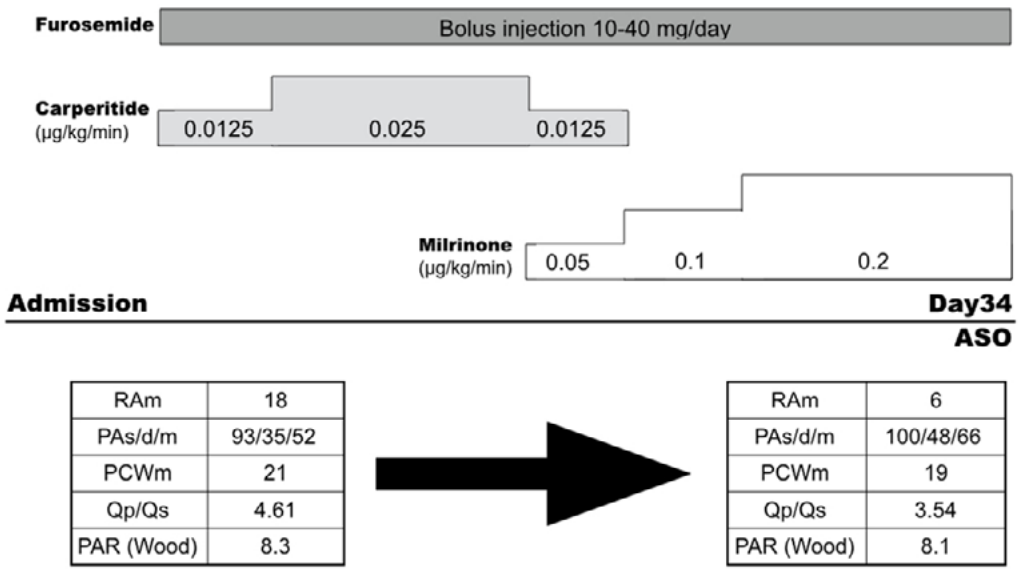

Figure 4. Preoperative medication with two diuretics and an inotropic agent. Although right atrium pressure was decreased, pulmonary artery pressure was not changed by the treatment. ASO = Amplatzer septal occluder; m = mean; PA = pulmonary artery; PAR = pulmonary artery resistance; PCW = pulmonary capillary wedge; Qp/Qs = pulmonary-to-systemic blood flow ratio; RA = right atrium; s/d/m: systolic/diastolic/mean.

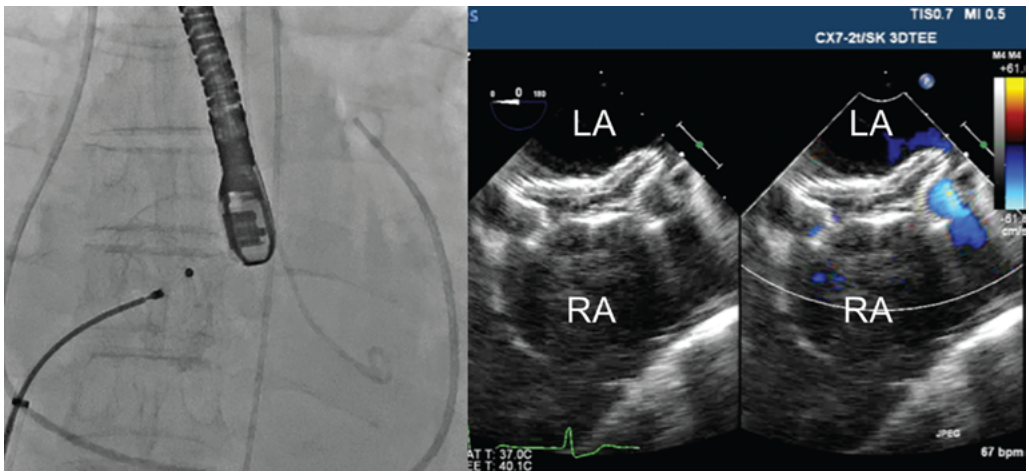

Figure 5. Angiographic *(left panel)* and TEE *(right panel)* findings during implantation of an Amplatzer septal occluder.

1 month before the operation. Although severe pulmonary hypertension was observed in this case, pulmonary artery dilators were not used before the procedure contrary to current recommendations for surgical treatment [6]. This is because a possible increase in left-to-right shunt due to a reduction in pulmonary artery resistance might increase left ventricular inflow, leading to left ventricular heart failure due to masked left ventricular restriction [7] at the time of ASD closure. In this case, ASD closure with an ASO was safely performed without any hemodynamic deterioration.

As the massive left-to-right shunt was completely eliminated by ASO implantation, pulmonary artery pressure was expected to decrease after the procedure, but this did not occur. Therefore, potent pulmonary artery dilators such as a PDE5 inhibitor, endothelin receptor blocker, and PGI_2 analog were started incrementally after the procedure. The present case is considered to be high risk because the PVR was greater than 5 Woods units m^2, and pulmonary artery pres-

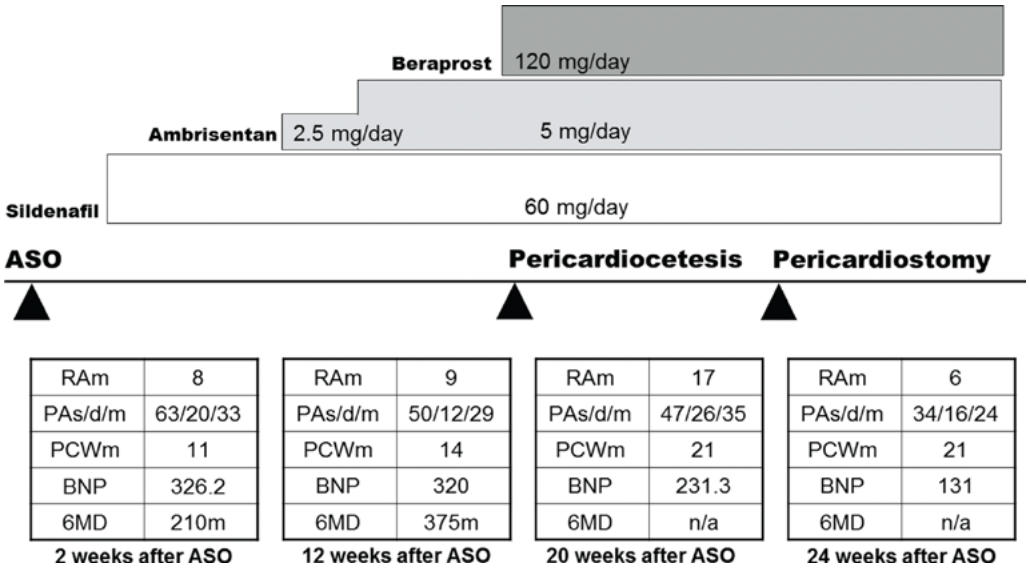

Figure 6. Medications and examinations after ASO implantation. 6MD = 6-min walk distance; ASO = Amplatzer septal occluder; m = mean; PA = pulmonary artery; PCW = pulmonary capillary wedge; RA = right atrium; s/d/m = systolic/diastolic/mean.

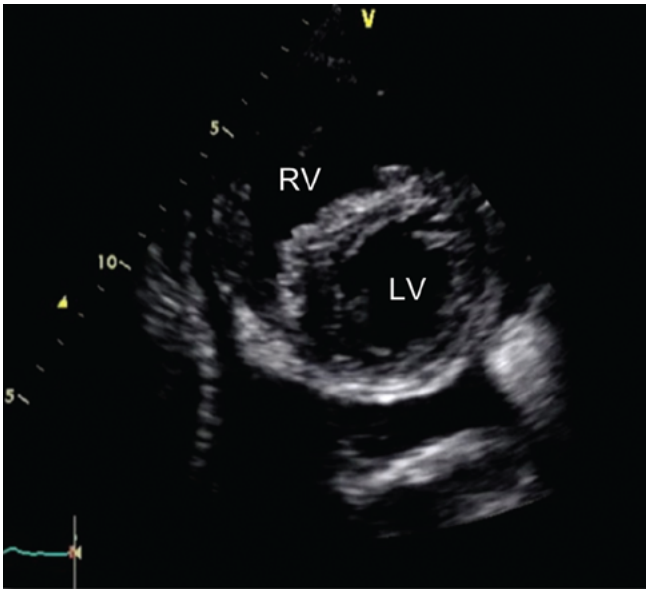

Figure 7. Short-axis view of transthoracic echocardiography after drainage of the pericardial effusion 6 months after atrial septal defect closure. The left ventricular shape became round, and the dimension of the left ventricle increased.

sure was greater than 70% of systemic pressure [8]. The pulmonary hypertension-specific medications that we administered are reportedly effective even in high-risk patients during long-term follow-up [8]. However, in our particular case, probably due to the presence of chronic massive pericardial effusion, the reduction in pulmonary arterial pressure fell short of our expectations. Because pericardiocentesis had no effect on pericardial effusion, pericardiostomy and drainage was performed according to previous reports [9]. Finally, the surgical procedure eliminated the effusion, which was followed by a decrease in pulmonary artery pressure to a nearly normal level and the disappearance of symptoms.

Conclusions

We report a case of giant ASD with massive pericardial effusion treated by ASO followed by the administration of pulmonary arterial dilators. Potent pulmonary hypertension drugs and percutaneous closure devices make high-risk ASD treatment possible, even in elderly and frail patients.

Conflict of Interest

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

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Intravascular Stent Implantation for Refractory Chylothorax Secondary to Congenital Superior Vena Cava Stenosis in an Infant

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Abstract

Chylothorax is a rare but dangerous cause of respiratory failure in the pediatric population. It most commonly presents after cardiac surgery or, alternatively, due to retrograde pressure on the thoracic duct from narrowing or obstruction in the innominate vein due to thrombus or neoplasm. We observed an unusual presentation of chylothorax in an otherwise healthy 3-month-old infant with congenital superior vena cava stenosis leading to acute respiratory collapse. After initially not responding to medical therapy, the patient was successfully treated with an intravascular stent.

Key Words:

Superior • Vena • Cava pediatric • Cardiology angioplasty interventional • Cardiology

Introduction

Respiratory failure secondary to chylothorax is a rare complication in patients with congenital heart disease [1]. In these patients, leakage of chyle into the thoracic space most commonly results from direct incidental damage to the lymphatic system during surgery or vascular obstruction in the innominate vein or superior vena cava (SVC) secondary to central venous line-associated thrombosis, postoperative vascular stenosis, or extravascular mass effect, all of which impede lymphatic decompression into the veins through the lymphatic duct. Very rarely, chylothorax is reported in infants with congenital stenosis of the SVC. In previous reports of chylothorax secondary to congenital SVC stenosis, patients have tended to present in the neonatal period and have almost always had additional complex congenital heart lesions that increased the index of suspicion for vascular anomalies as the underlying etiology [2, 3, 4]. Here, we present a unique case of an isolated congenital SVC stenosis leading to chylothorax and acute respiratory failure in an otherwise healthy 3-month-old. We describe the successful transcatheter stenting of the SVC and subsequent rapid resolution of the patient's effusion and symptoms.

Case Presentation

An ex-full-term and otherwise healthy 3-month-old boy was admitted to the hospital for acute respiratory failure in the context of a massive effusion surrounding the right lung visualized on chest x-ray. He was immediately intubated and the fluid drained with a tube thoracostomy. Fluid analysis showed high

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levels of triglycerides and cholesterol, consistent with a chylothorax. He was extubated shortly after thoracostomy tube placement and was stable on room air, although a significant amount of chylous fluid continued to drain from his thoracic cavity. A magnetic resonance lymphangiogram was obtained to evaluate for congenital lymphatic malformation or signs of genetic lymphedema-lymphangiectasia syndrome. However, imaging showed a normal thoracic duct with no obvious lymphatic malformation or intrathoracic mass. An upper extremity vascular Doppler ultrasound was negative for deep venous thrombosis in the right and left internal jugular, subclavian, and axillary veins. An echocardiogram had reportedly been notable for a patent foramen ovale and otherwise structurally normal heart at the referring hospital and was repeated upon admission. Transthoracic echo showed a narrowed right-sided SVC with low velocity but forward flow to the right atrium. There was retrograde flow in the unobstructed innominate vein and multiple large posterior venous structures that appeared to be decompressing the upper body central veins caudally to a dilated inferior vena cava. The echocardiogram was negative for additional intracardiac or vascular lesions. The patient showed no signs of plethora or upper body edema on exam. After the patient was stabilized, medical management was attempted, including octreotide infusion and low fat formula feeding followed by central line placement in the right subclavian vein and initiation of total parenteral nutrition (TPN) when the clinical response was deemed inadequate. Chylous drainage from the chest tube continued to be significant despite these measures. Given the echocardiographic finding of SVC obstruction as the likely etiology of continued chylous effusion, the patient was referred to the cardiac catheterization lab for angiography and intervention 14 days after admission.

In the catheterization lab, the left femoral vein was accessed, and a hydrophilic catheter was placed in the stenotic SVC for hemodynamics and angiography. Right atrial pressure was normal, with a 5 mmHg gradient across the SVC. Angiography showed a severe long-segment SVC stenosis measuring 18 mm in length and 2.6 mm at its narrowest diameter (Figure 1). The upper body central veins were decompressing through a very dilated hemiazygous system. The SVC was nearly occluded by the right subclavian vein central line that had been placed 3 days prior to catheterization. Balloon

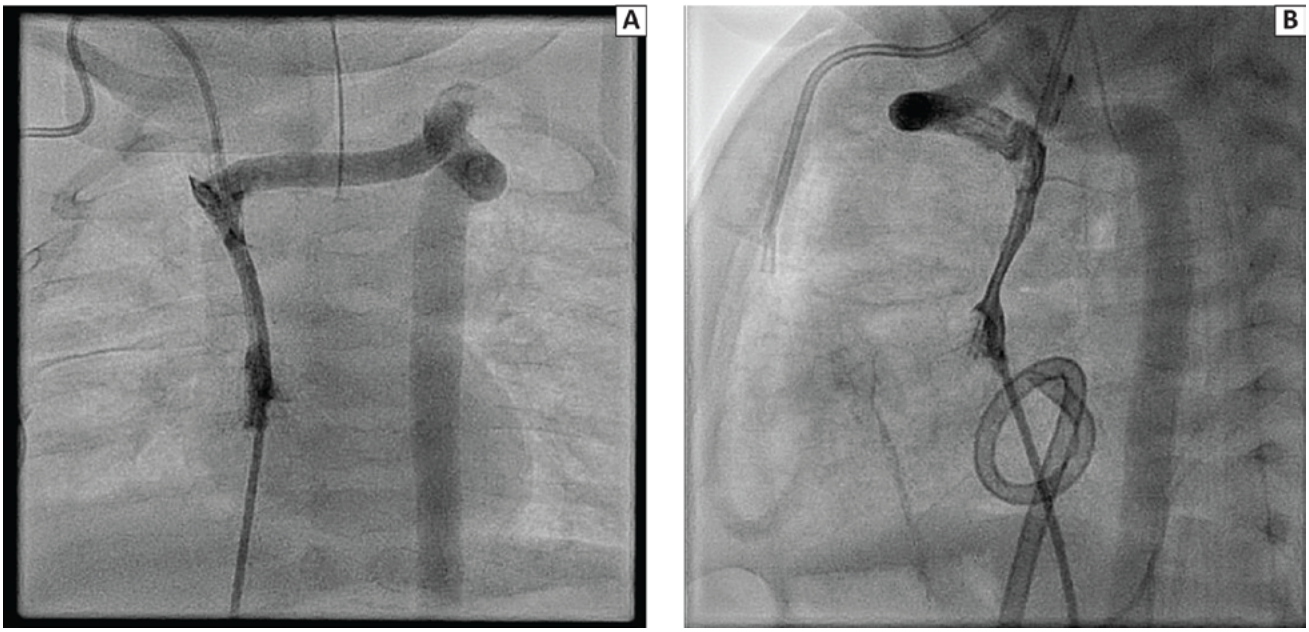

Figure 1. *Panel A.* Severe long-segment superior vena cava (SVC) stenosis and collateral decompression of upper body venous flow through a very dilated hemiazygos system. Note that the SVC stenosis was appreciated prior to insertion of the right subclavian central venous line. *Panel B.* Lateral view, with thoracostomy tube in view.

angioplasties were performed in the narrowed SVC using a 5 × 20 mm Sterling balloon (Boston Scientific, Marlborough, Massachusetts) over a .018 Platinum Plus wire (Boston Scientific, Marlborough, Massachusetts), but a tight waist persisted at 14 ATM of pressure (Figure 2). Dilations were then performed with a higher pressure 5 × 20 mm Dorado balloon (Bard Medical, Covington, Georgia) over the same .018 wire, which resulted in resolution of the tight waist at 24 ATM. However, on repeat angiography, although the SVC showed improved flow, there was significant vascular recoil and residual obstruction necessitating stent implantation. In anticipation of stent implantation, the central venous line from the right subclavian vein that crossed the SVC was pulled out of the SVC and into the left innominate vein using a snare catheter introduced from the hemiazygos vein. An 8F long sheath was placed in the SVC from the femoral vein over a .035 Amplatz Super Stiff wire (Boston Scientific, Marlborough, Massachusetts), and a Palmaz Genesis 1910 XD stent (Cordis, Fremont, California) mounted on a 6 × 20 mm Dorado balloon was deployed in the SVC and further dilated with a 7 × 20 mm Dorado balloon at 22 ATM. Afterward, there was a 1 mmHg gradient across the stent, and the SVC was widely patent, measuring 6 mm. At the conclusion of the procedure, there was excellent flow through the well-positioned stent, and the veins no longer decompressed through the hemiazygos system; rather, all venous flow entered the heart briskly through the SVC (Figure 3). Of note, the patient required balloon venoplasty of the right iliofemoral vein due to narrowing, likely from a previous line placement. After venoplasty, a PICC line was placed in the right femoral vein so that the tunneled subclavian line could be removed.

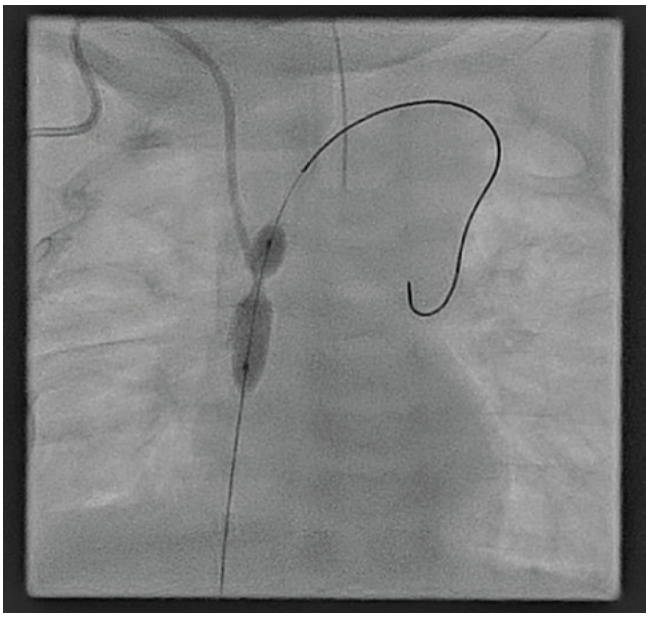

Figure 2. Balloon dilation shows a significant residual waist using a 5-mm Sterling balloon at 14 ATM of pressure. The stenosis ultimately required 22-24 ATM of pressure to resolve at full inflation.

The patient was transitioned off octreotide over the following 4 days. Medium-chain triglyceride formula was replaced with breast milk, and no recurrence of chylous pleural effusion was observed. The chest tube was removed on post-catheterization day 6.

To preserve and rehabilitate the obstructed right femoral vein, repeat catheterization was performed 11 days after initial catheterization, at which time the right femoral vein PICC was removed and angioplasty was performed to maintain the patency of the right iliofemoral vein. Repeat angiography of the SVC stent showed continued unobstructed flow across the SVC stent with no short-term recurrent narrowing (Figure 4). The patient was discharged 2 days later on low-dose aspirin for stent endothelialization.

At the most recent follow-up 5 months after discharge, echocardiogram showed excellent flow through a stably positioned SVC stent with a mean Doppler gradient of 3.8 mmHg. The dilated hemiazygous vein was no longer visualized. No recurrent pleural effusions were noted on follow-up chest radiography. The patient will require periodic repeat catheterizations with angioplasty of the SVC stent for somatic growth until the vessel reaches adult size.

Discussion

Chylothorax is a known cause of respiratory compromise and appears at an increased frequency in patients with congenital heart disease, in particular following intrathoracic surgical intervention. Chylothorax is most commonly due to incidental damage to the thoracic duct during a surgical procedure or, alternatively, to venous stenosis/obstruction causing

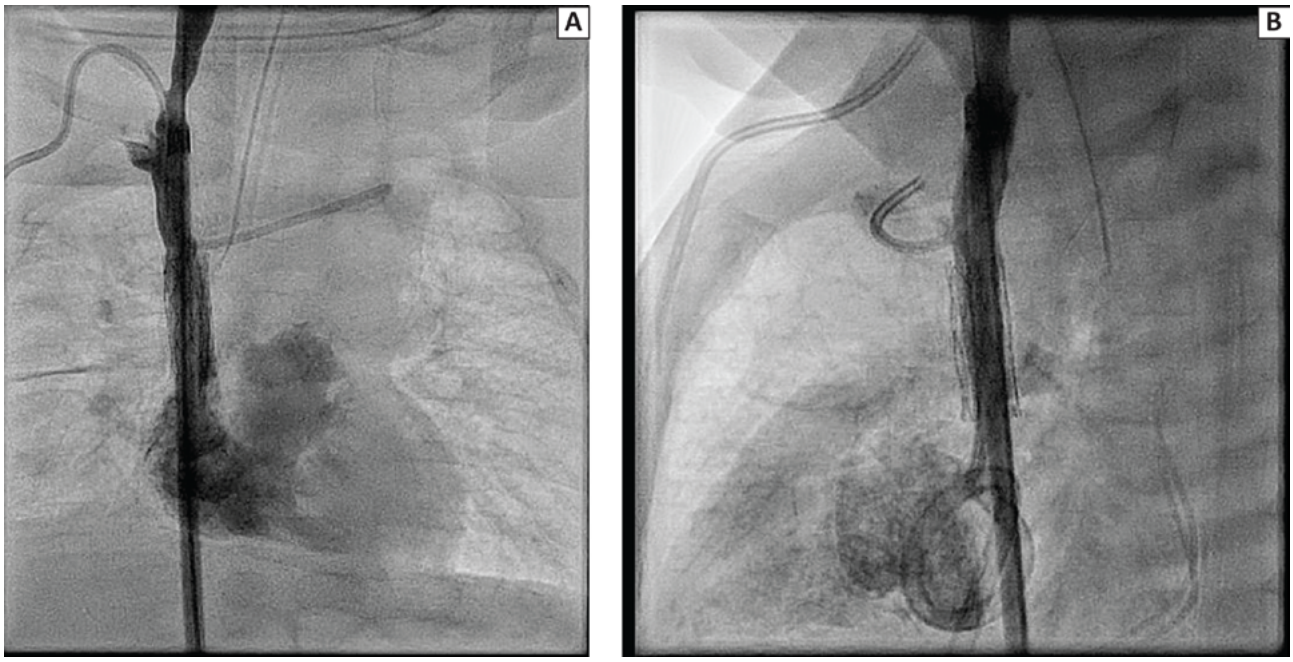

Figure 3. *Panel A.* After implantation of a PG1910XD stent in the superior vena cava and high-pressure dilation to 7 mm, there was increased forward flow across the stent into the right atrium and no retrograde flow in the innominate vein or decompression through collateral veins. Note that the right subclavian central venous line was repositioned in the left innominate vein using a snare catheter from the hemiazygos vein (snaring not pictured). *Panel B.* Lateral view after stent dilation.

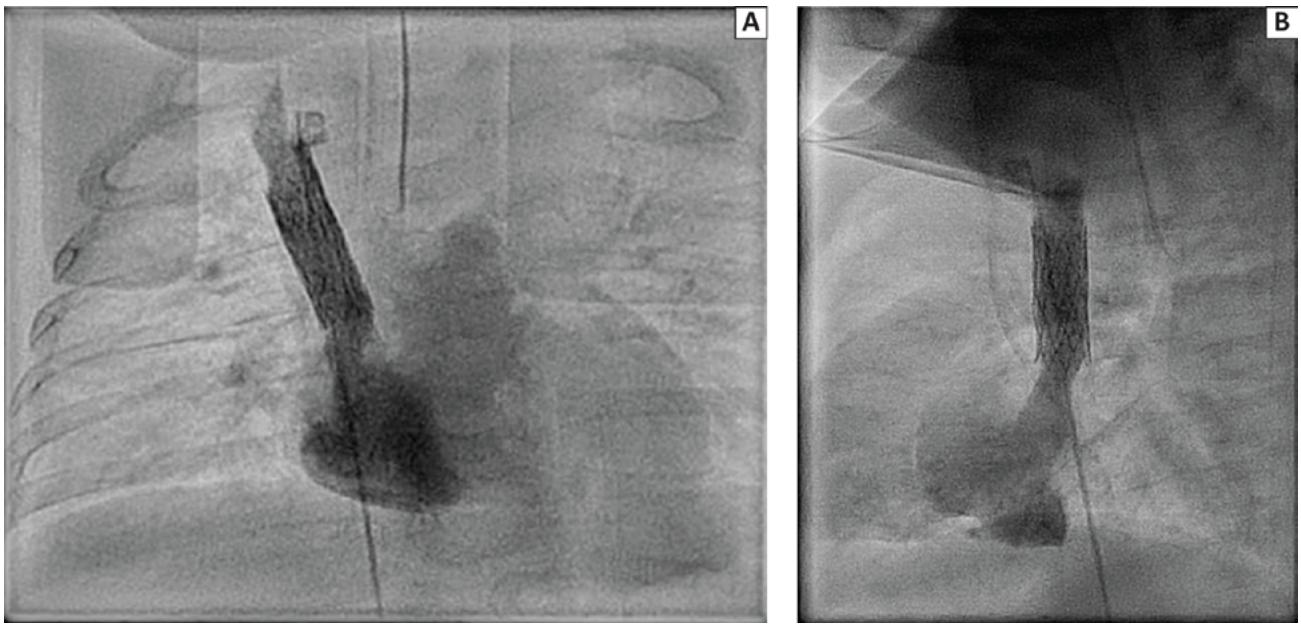

Figure 4. *Panel A.* Two weeks after the initial procedure, there was continued unobstructed flow across the stent and no short-term recurrent stenosis. *Panel B.* Lateral view of 2-week follow-up.

increased pressure in the thoracic duct [5]. The most common site for this type of obstruction is the innominate vein [6], but as demonstrated in this case, the SVC must also be considered as a potential site of vascular compromise. The most common causes of SVC stenosis or obstruction leading to chylothorax in children

are acquired rather than congenital. The three most common etiologies are thrombosis surrounding a port or central line, external compression from tumors or other mass effect, and surgical complications including direct injury of the thoracic duct or postoperative stenosis of the SVC or innominate vein [4, 7, 8, 9, 10].

Congenital—rather than acquired—SVC stenosis as presented in our case is exceedingly rare and thus far has only been reported in the presence of other significant cardiac anomalies. For our patient, the absence of upper body edema or plethora, along with highly developed collateral flow, indicates that this lesion had likely been present since birth. Although the causes of chylothorax are varied, the workup and management are similar across all etiologies. The diagnosis of chylothorax is made based on pleural fluid analysis containing a triglyceride concentration greater than 110 mg/dl, whereas it is excluded by a concentration less than 50 mg/dl. Intermediate values between 50 and 110 mg/dl can be further diagnosed by lipoprotein electrophoresis of the fluid to detect chylomicrons. Alternatively, a ratio of cholesterol-to-triglycerides in pleural fluid of less than 1 is also diagnostic [11]. Chylothorax without a clear underlying cause (e.g., trauma, obstruction, or post-surgical damage) accounts for 5–10% of cases and is typically diagnosed on lymphangiography or lymphoscintigraphy [12].

After initial invasive drainage of the effusion, if indicated for therapeutic or diagnostic purposes, the goal of therapy is to minimize production of chyle medically while treating the offending clot or obstruction, if possible. Medical treatment typically involves a fat-restricted diet supplemented with medium-chain triglycerides, followed by octreotide (0.3–10 mcg/kg/hour titrated to response, with a median dose of 2.8 mcg/kg/hour) and then gut rest and TPN if other measures are unsuccessful. Approximately 80% of pediatric patients respond to medical therapy, although octreotide therapy and TPN carry their own risks [1, 13]. Recommendations vary regarding indications and timing for surgical management with thoracic duct ligation or pleurodesis, but cases are generally considered refractory when effusion persists for more than 2 weeks despite conservative management [14]. Overall, these surgical interventions have a high success rate, and the main risk factor for death or chylothorax recurrence after surgery is thrombosis of the upper body venous vessels [15], thus confirming the importance of relieving such an occlusion whenever possible.

There is only one previous report of isolated congenital SVC stenosis leading to chylous effusion and respiratory failure; in that case, the patient presented in the immediate neonatal period with severe hydrops and upper body edema consistent with SVC syndrome [16]. Here, we report the first case without an obvious acquired etiology presenting outside the immediate perinatal period. Notably, the patient did not present with obvious clinical findings of SVC syndrome and had developed significant venous collateralization, suggesting that the chronicity of his obstruction likely began during fetal life. Ultimately, his upper body central venous pressure was only mildly to moderately elevated due to collateral decompression, which likely allowed for a relatively slow accumulation of chyle in the pleural space until respiratory decompensation.

Furthermore, our patient's hospital course confirms the importance of understanding the various potential underlying causes of chylothorax and early intervention via relief of venous obstruction when possible. Our patient experienced a delay in treatment (i.e., abnormal echocardiogram on day 2, cardiology consulted on day 10, and the procedure itself performed on day 14) due to the standard of care of 2 weeks of medical therapy before declaring a chylothorax refractory and proceeding with an invasive means of treatment. An argument can be made that if a chylothorax is due to a known venous obstruction, intervention could be performed immediately to relieve this obstruction in lieu of medical management. As such, it is essential to perform the necessary imaging studies to evaluate for venous anomalies, thrombosis, or intrathoracic mass affecting both the innominate vein and/or the SVC.

Our patient's presentation and rapid recovery following acute decompression of the upper body central veins with effective intravascular stent implantation demonstrates that significant chylous effusion can occur from severe congenital vascular obstruction alone, in the absence of surgical complications or other factors. When chylothorax is caused by vascular obstruction, this phenom-

enon is most commonly observed at the site of the innominate vein. However, our case provides an important reminder to consider the SVC as another potential site of stenosis or obstruction, even in the absence of other intracardiac lesions. Anatomic narrowing causing obstruction to lymphatic drainage should be considered in all patients with chylothorax, even in the absence of other signs of venous congestion such as upper body edema. Early catheter-based interventions should be considered because they are effective in relieving vascular obstructions and resolving chylous effusions.

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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Percutaneous Tricuspid Valve Replacement

Two Cases of Valve-in-Valve and Valve-in-Ring

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Abstract

Tricuspid valve (TV) replacement via transcatheter techniques is feasible but not yet widely utilized. The two cases reported here describe transcatheter replacement of a failing bioprosthetic TV and a failing native valve that previously underwent surgical annuloplasty repair. The first case was a 26-year-old male with Ebstein's anomaly who underwent surgical TV replacement with a 33-mm Hancock bioprosthesis 15 years prior to transcatheter intervention. The valve had become stenotic and regurgitant and was successfully replaced with a Melody valve mounted on a 22-mm Ensemble balloon-in-balloon system. The second case was a 59-year-old female with rheumatic heart disease who had undergone prior surgical TV repair with a 30-mm Edwards incomplete annuloplasty ring. Her repaired valve eventually became severely regurgitant, and she underwent successful transcatheter valve replacement using a 29-mm Sapien XT valve. She exhibited paravalvular regurgitation necessitating vascular plug implantation. These two cases highlight the potential utility and limitations of commercially available transcatheter valves in the tricuspid position. Moreover, this report compares and contrasts the procedural nuances of TV replacement within a complete bioprosthetic valve ring versus an open annuloplasty ring.

Key Words:

Tricuspid valve stenosis • Tricuspid valve insufficiency • Ebstein's anomaly • Transcatheter valve replacement • Right-sided heart failure • Cardiac valve annuloplasty • Heart valve prosthesis

Introduction

Native tricuspid valve (TV) dysfunction is associated with both congenital and acquired heart conditions. Conditions such as severe pulmonary hypertension or right ventricular (RV) outflow obstruction can lead to elevated RV systolic pressure, commonly causing RV and TV annular dilation and subsequent tricuspid regurgitation (TR). Patients with congenital RV outflow dysfunction, such as repaired Tetralogy of Fallot with pulmonic stenosis and/or regurgitation, commonly develop secondary TR. Other congenital conditions associated with primary TV dysfunction include Ebstein's anomaly, with regurgitation occurring due to dysplastic tricuspid leaflets. Although TR may be well tolerated for decades, eventual clinical sequelae usually emerge. These most commonly involve progressive RV dysfunction, the development of ventricular and supraventricular arrhythmias, and elevated central venous pressure leading to multi--organ congestion.

TV repair techniques include the placement of annular rings as well as more complex operations to relocate or augment TV leaflets in patients with Ebstein's anomaly. Repair is usually successful in the short and intermediate term, but up to 25% of

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patients develop moderate or severe regurgitation within 5 years [1, 2]. In patients with congenital forms of TR, surgical replacement may be necessary at a young age; longer-lasting mechanical valves would require placing young patients on anticoagulants, and yet bioprosthetic valves commonly require replacement within 10–15 years [3, 4]. Use of biological valves early in life most certainly predicts multiple reoperations as they begin to fail, and repeat surgery is associated with increased morbidity and mortality [5].

Percutaneous TV replacement may be a viable alternative to surgical reoperation. This procedure has been well documented in the aortic and pulmonary valve positions, and there is a growing body of evidence for the use of both the Melody (Medtronic Inc.) and Sapien (Edwards Lifesciences) valves in failing TV rings and bioprostheses [6]. Although the procedural success rate is high with immediate hemodynamic benefits, there is paucity of long-term data. In terms of recurrent TR post annuloplasty, successful utilization of transcatheter valves is limited to isolated case reports and small case series [7, 8, 9]. The present report details two cases of transcatheter TV replacement—one within a failing bioprosthesis and the other in a regurgitant native valve with an incomplete annuloplasty ring—and seeks to highlight the differences in technique, device selection, and procedural outcomes.

Case 1: Failing Bioprosthesis

A 23-year-old male born with Ebstein's anomaly and ostium secundum atrial septal defect underwent surgical TV repair at 3 years of age. Complications included prolonged intensive care unit stay and a sternal wound infection. At 14 years of age, he underwent surgical TV replacement with a 33-mm Hancock bioprosthesis due to progressive TR and recurrent atrial arrhythmias. Complications included ventricular fibrillation and bleeding requiring resuscitation and exploration, again resulting in a prolonged hospital course. He did well thereafter and was not cyanotic, therefore the atrial septal defect was percutaneously closed. A decade later, he developed progressive symptoms and signs of RV failure and volume overload and was noted to have severe stenosis and moderate regurgitation of the bioprosthetic TV. Due to multiple prior sternotomies and surgical complications, the patient and surgeon preference was to avoid further surgery. He was therefore referred for transcatheter valve replacement.

The patient underwent uncomplicated percutaneous tricuspid valve-in-valve (TVIV) replacement with a 22-mm Melody valve under general anesthesia with transesophageal echocardiography (TEE) guidance. The right femoral vein was utilized for right heart catheterization and TV intervention. Pre-interventional mean inflow gradient was 6 mmHg with a heart rate of 55 beats per minute (bpm; Figure 1A).

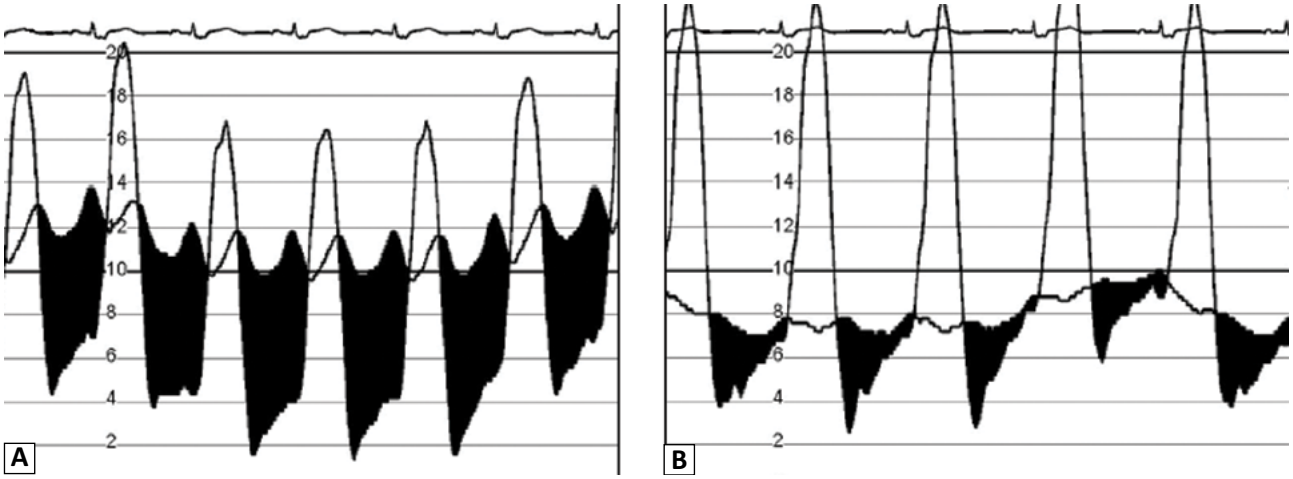

Figure 1. *Panel A*. Pre-deployment mean RV-pulmonary artery gradient measurement of 6 mmHg, Fick cardiac output of 4.45 L/min, and estimated TV area by the Gorlin formula of 0.97 cm^2. *Panel B*. Post-deployment mean RV-pulmonary artery gradient measurement of 2 mmHg.

Following diagnostic catheterization, an Amplatzer super-stiff (St. Jude Inc.) was placed in the right pulmonary artery, and a PTS-X 30-mm balloon (Numed Inc.) was inflated across the tricuspid bioprosthesis with a 21-mm waist noted (Figure 2A). A Melody valve was then mounted on a 22-mm Ensemble balloon-in-balloon delivery system and deployed within the Hancock II bioprosthesis (Figure 2B). Subsequent hemodynamic assessment showed a reduction in the mean inflow diastolic gradient to 2 mmHg (Figure 1B) without evidence of intra- or paravalvular leak (PVL). The patient was extubated in the catheterization laboratory and discharged to home the following day on daily 325 mg aspirin therapy. A post-operative echocardiogram revealed a mean inflow gradient of 3.5 mmHg and trace TR. The patient reported increased physical stamina and denied dyspnea on moderate exertion.

However, 8 months after the valve implantation, a routine echocardiogram demonstrated an increase in the diastolic transvalvular mean Doppler gradient to 11 mmHg, suggestive of valve stenosis. The valve was not well visualized, but there was clinical suspicion of a possible thrombotic etiology. The patient was initiated on anticoagulation with rivaroxaban 20 mg daily with a reduction in the mean diastolic gradient by Doppler to 4 mmHg within 3 weeks, and the valve has continued to function well 14 months post-implantation.

Case 2: Failing Native Valve with Incomplete Annuloplasty Ring

A 59-year-old female with a history of atrial fibrillation and rheumatic mitral valve (MV) stenosis underwent bioprosthetic MV replacement and the Maze procedure at 46 years of age. At 57 years of age, she received a redo MV replacement for prosthetic valve stenosis and TV repair with a 30-mm Edwards incomplete annuloplasty ring for severe TR. She developed high-grade atrioventricular block and also received a transvenous permanent dual chamber pacemaker with an atrial lead and a ventricular lead placed in a lateral cardiac vein via the coronary sinus to avoid crossing the TV. Within 1 year, she developed heart failure symptoms and required up-titration of diuretic therapy and home intravenous dobutamine. She had progressive renal failure and severe recurrent TR requiring hospitalization and initiation of multiple inotropic medications and continuous veno-venous dialysis. She was also diagnosed with congestive hepatic cirrhosis. She was evaluated for surgical valve re-

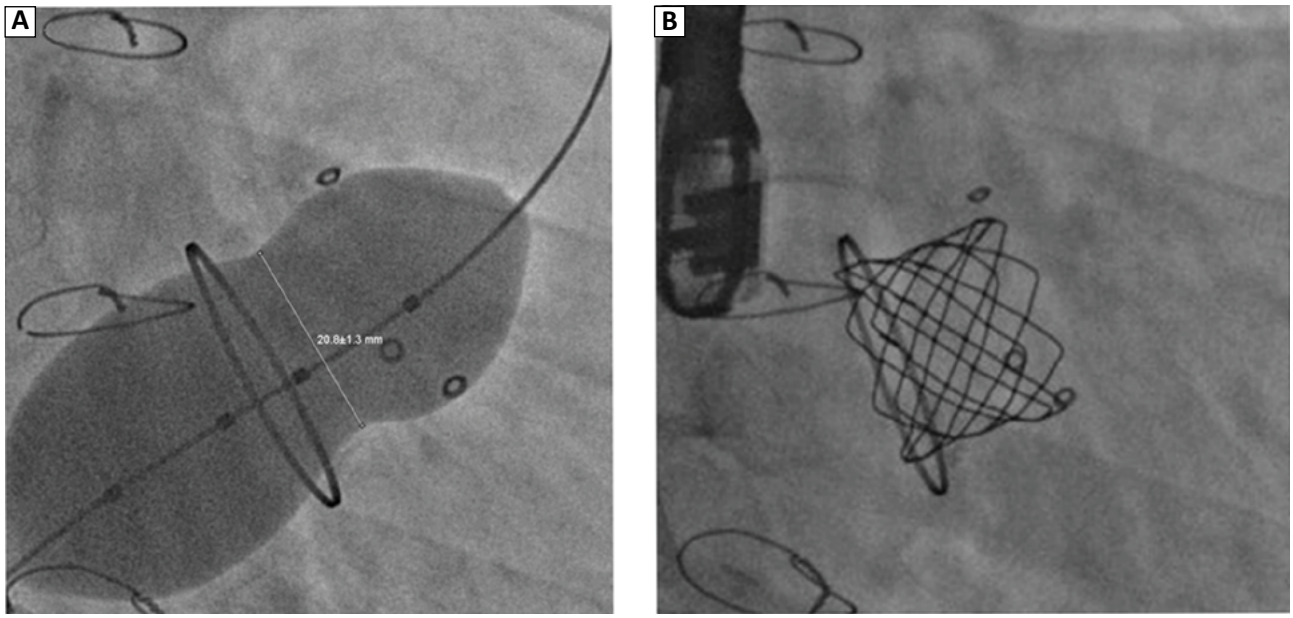

Figure 2. *Panel A.* Balloon inflation across the stenotic prosthesis indicated a 21-mm waist. *Panel B.* Post-placement of a 22-mm Melody valve.

placement and multi-organ transplantation but was felt to not be a candidate for either. Percutaneous TV replacement was considered as a potential palliation.

The patient underwent transcatheter TV implantation using a 29-mm Edwards Sapien XT valve without pre-stenting of the ring. The procedure was guided by TEE and fluoroscopy and performed under general anesthesia (Figure 3A). Via a femoral venous approach, a 30-mm × 4-cm PTS-X balloon (Numed) was inflated across the annuloplasty ring to reveal a 27–28 mm waist (Figure 4A) with evidence of mild to moderate residual regurgitation within the medial "open" portion of the ring (Figure 3B). Right coronary angiography showed no evidence of compression. A 29-mm Edwards Sapien XT valve and delivery system were introduced into the tricuspid position. The valve was deployed with rapid pacing at 160 bpm using the existing permanent pacemaker (Figure 4B). Following valve deployment, TEE imaging demonstrated moderate medial PVL and mild lateral PVL (Figure 3C). The medial PVL was successfully crossed with a guide wire and catheter, and two Amplatzer vascular plugs (AVP II, St. Jude Medical) were successfully placed, reducing the PVL to mild (Figure 3D).

The patient tolerated the procedure well and was weaned from all pressor support within 1 week, remaining hemodynamically stable thereafter. Her renal function improved dramatically, and she was able to discontinue dialysis 1 month post-intervention. Follow-up transthoracic echocardiograms showed mild TR with no stenosis 8 months post-intervention. Her functional capacity improved from New York Heart

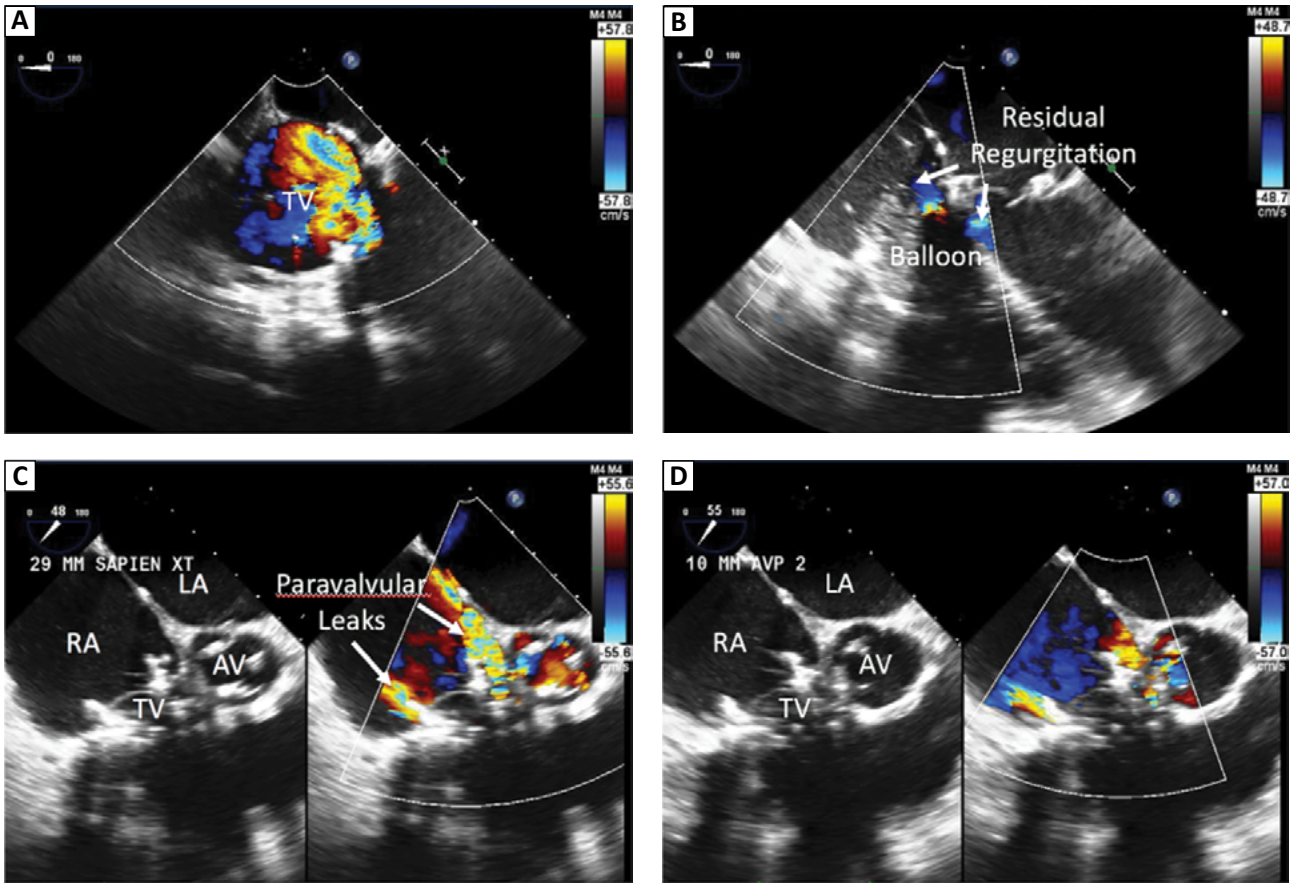

Figure 3. Transesophageal Doppler echocardiographic images. *Panel A*. Tricuspid valve color Doppler pre-deployment demonstrating severe regurgitation. *Panel B*. Balloon inflation across the native valve demonstrated residual medial regurgitation in the open part of the annuloplasty ring. *Panel C*. Post-deployment of a 29-mm Sapien XT valve showed moderate medial and mild lateral paravalvular leak (PVL). *Panel D*. Post-deployment of Amplatzer vascular plugs medially resulted in a reduction of PVL. No central regurgitation was noted. RA = right atrium; LA = left atrium; TV = tricuspid valve; AV = aortic valve.

Association (NYHA) class IV prior to the procedure to NYHA class II within 3 months. However, increasing severity of the lateral PVL precipitated worsening heart failure symptoms and volume overload. Therefore, she underwent repeat catheterization 10 months after valve replacement with successful occlusion of the lateral PVL under intracardiac echo guidance (Figure 5A). Follow-up echocardiography at 3 and 6 months showed stable valve function and mild lateral and medial PVL (Figure 5B).

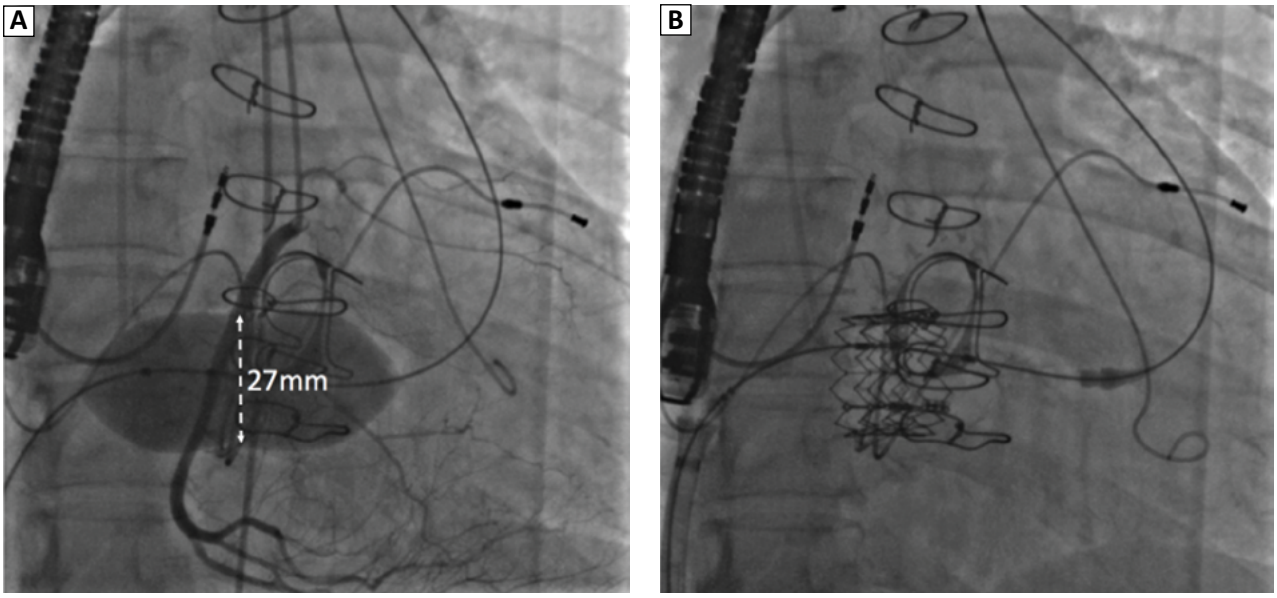

Figure 4. *Panel A*. Balloon inflation across the annuloplasty ring indicated a 27-mm waist. *Panel B*. Post-placement of a 29-mm Sapien XT valve.

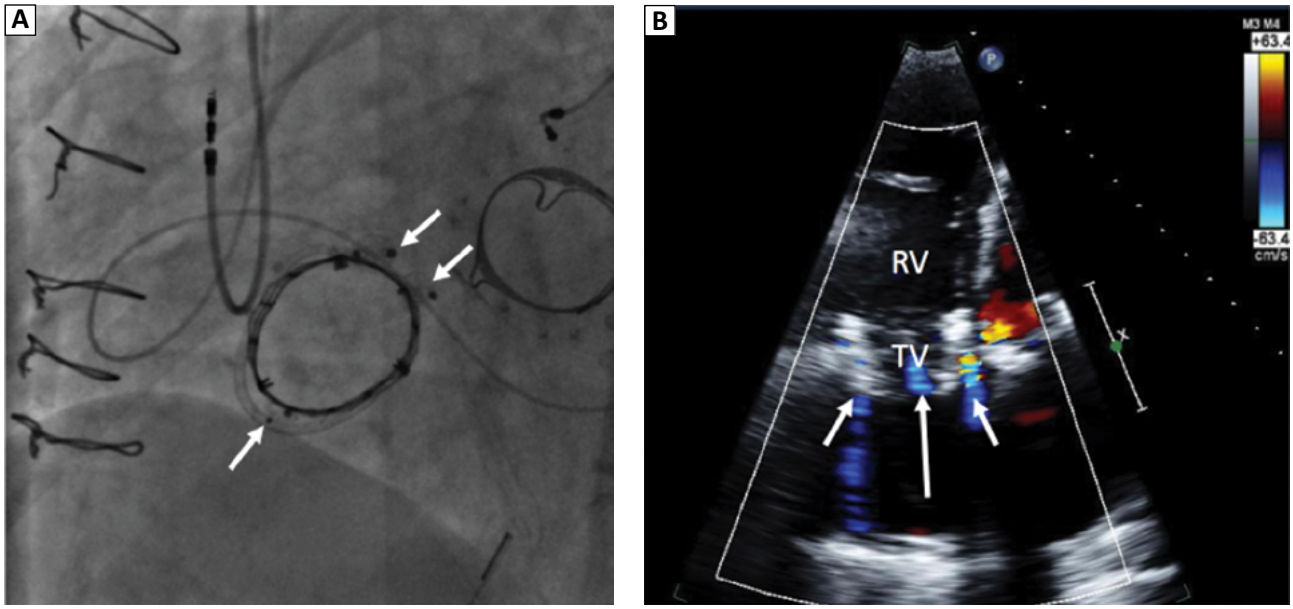

Figure 5. *Panel A*. Post-deployment of additional Amplatzer vascular plugs to treat worsening lateral paravalvular leak (PVL) in addition to medial plugs from initial catheterization. *Panel B*. Transthoracic echocardiogram with Doppler post-deployment of additional vascular plugs showed reduced PVL. Trace central regurgitation was noted. RV = right ventricular; TV = tricuspid valve.

Discussion

Although there is an increasing number of reports of successful transcatheter deployment of biological valves in the tricuspid position, this is not yet standard of care. In 2011, a study of 15 patients from eight medical centers reported successful deployment in 12 patients without complication [10]. A more recent study of 10 patients, including five with Ebstein's anomaly, reported only one complication of valve migration post-deployment [11]. The largest experience to date is a multi-center database of TVIV implants detailing the outcomes and 1-year follow-up of 156 patients with TVIV, which demonstrated no procedural mortality and excellent short- to medium-term outcomes [6]. Our two cases involved specific complexities of the procedure and post-implantation outcome, demonstrating our approach to their management.

Among these patients, it is important to identify the primary diagnosis because Ebstein's anomaly is associated with complex hemodynamics. Patients with Ebstein's anomaly often suffer from RV dysfunction and restrictive diastolic function and may have undergone numerous surgical repairs and replacements. Moreover, they are at increased risk for both atrial and ventricular arrhythmias [12]. As the morbidity and mortality risk associated with surgical TV replacement is often high [5], the excellent short- and intermediate-term outcomes of trans-catheter valve replacement make it a viable alternative.

Surgical TV replacement is most often performed using bioprosthetic valves. There is limited experience with mechanical valves in this position, and there is a concern for a higher risk of valve thrombosis in the tricuspid position [13]. It has traditionally been recognized that the primary mode of bioprosthetic TV failure is structural deterioration with resultant calcification and progressive stenosis and regurgitation. However, recent studies show that bioprosthetic valve thrombosis (BPVT) is common and a more underdiagnosed mode of failure than originally thought [14, 15]. The estimated prevalence of BPVT in the tricuspid position is 12%, resulting in early failure of the valve usually occurring within 5 years of implantation. BPVT of a tricuspid bioprosthesis is usually insidious in presentation and results from an elevation in central venous pressure, which symptomatically is much less dramatic than left-sided valve thrombosis with a resultant elevation in left atrial pressure and pulmonary edema [13, 16]. Pathognomonic signs of BPVT include an acute rise in transvalvular gradient of more than 50% over baseline in the short- to medium-term, paroxysmal atrial fibrillation, increased cusp thickness, decreased cusp mobility, and sub-therapeutic international normalized ratio [16]. BPVT was highly likely in Case 1, in which there was a sharp increase in valve gradient (>50%) within 8 months after Melody valve placement. The initiation of anticoagulation resulted in a dramatic improvement in valve gradient, which provides additional support for the assertion that this was early BPVT. The use of non-vitamin K antagonists for anticoagulation following bioprosthetic valve replacement has not been well studied but in this case proved effective.

Percutaneous valve replacement of native TVs presents with additional challenges due to the lack of secure circumferential scaffolding provided by surgical bioprostheses. The native annulus is highly distensible and is not a stable landing zone for balloon-dilated transcatheter valves. In patients with annuloplasty bands or rings of the TV, a transcatheter valve-in-ring approach is feasible. Case 2 demonstrates that such an approach is possible in patients with incomplete annuloplasty rings from previous TV repair.

It should be noted that an incomplete ring annuloplasty does not supply the same level of support as a previously placed bioprosthetic valve. PVL is common and should be expected, especially in the medial open part of the ring. However, this can be readily treated with transcatheter vascular plug/occluder deployment at the time of valve implantation. PVL can be progressive, as in Case 2, and may require additional vascular plug placement during follow-up.

Additionally, it is important to recognize that the reduced radial support along the open portion of the existing ring could ostensibly lead to deformation of the bioprosthetic valve. Sufficient loss of circular shape could prevent the leaflets from coapting as originally designed, potentially resulting in regurgitation. However, despite the loss of the circular shape in Case 2, there was minimal intravalvular regurgitation.

Paravalvular regurgitation is expected and was adequately treated with vascular plugs.

Conclusion

In addition to exhibiting immediate post-interventional improvements and relief of symptoms without complication, both patients have remained stable in the intermediate term. One patient likely developed Melody valve thrombosis, which was effectively treated with oral anticoagulation. Pending additional long-term follow-up, these cases provide evidence of substantial and versatile benefits of a transcatheter approach to TV replacement, both in bioprosthetic and native TV post-surgical annuloplasty.

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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