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Original Article

Percutaneous transcatheter closure of prosthetic paravalvular leaks

Kaitao Jian*, Qiang Wang*, Wei Zhang, Jianshi Liu

*Department of Cardiac Surgery, Tianjin Chest Hospital, Taierzhuangnan Road 291#, Jinnan District, Tianjin, China. *Equal contributors.*

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Abstract: Background: Paravalvular leaks (PVLs) are among the most common complications of surgical valve replacement. However, improvements in interventional radiology allow alternative strategies for their treatment. This study evaluated percutaneous transcatheter closure of PVLs in 13 patients (10 male, 3 female). Methods: The location, shape, and size of the PVL were first identified by fluoroscopy, transesophageal echocardiography (TEE), and transthoracic echocardiography (TTE); we then selected the appropriate device. Thirty days after the procedure, heart morphology and function were assessed by TTE. Vital status and adverse events of all patients were regularly assessed. Results: Percutaneous transcatheter closure of prosthetic PVL was a technical success in 12 patients. The only major adverse event occurred in one patient whose sudden death we consider unrelated to the surgery. Left ventricular end-diastolic diameter (LVED) and pulmonary artery pressure (PAP) were decreased significantly compared with pre-operation echocardiographic assessment (from 62.9±16.2 to 59.2±16.1 mm, and from 41.5±10.2 to 34.9±8.9 mmHg). New York Heart Association functional class was improved by at least one grade in five patients. No patient required blood transfusion after the procedure. The patient survival rate after PVL closure was 83%. Another death occurred 13 months after surgery because of progressive heart failure. There was no bioprosthetic or autologous valve erosion, and no hemolysis, thromboembolic events, or cardiac blocking. Conclusions: Our experience suggests that percutaneous transcatheter closure of PVL is feasible and safe with careful anatomic analysis of the PVL and selection of appropriate patients and devices.

Keywords: Paravalvular leak, regurgitation, catheters, Amplatzer occlusion device, device closure

Introduction

Paravalvular leaks (PVLs) are among the most common complications of surgical valve replacement. PVLs in the mitral position are estimated to occur in 12% to 22% of mechanical valve replacements and 3% to 11% of bioprosthetic valve replacements; in the aortic position, PVLs are estimated to occur in 6% to 10% of mechanical valve replacements and 1% to 3% of bioprosthetic valve replacements [1, 2]. The clinical consequences of PVL vary depending on the severity of paravalvular prosthetic regurgitation. Most small PVLs are asymptomatic and have no hemodynamic significance. However, moderate or severe paravalvular prosthetic regurgitation caused by PVLs might cause serious clinical sequelae, such as hemolytic anemia, progressive cardiac function impairment, or heart failure. Due to the widespread use of intraoperative transesophageal echocardiography (TEE), almost all PVLs in the immediate post-operative period can be corrected during the operation; however, PVLs occurring later usually require the valve replacement to be redone.

The technical requirements for redoing valve replacements are not particularly demanding, and in most cardiac centers it has become a routine procedure. Nevertheless, oozing of large wound surfaces caused by separation of adhering tissue in patients taking warfarin, systemic inflammatory response generated by long surgery times, and whether a patient who has already presented with heart failure can recover from the surgery can all be associated with high operative risk and variable results [3, 4].

Interventional therapy is currently prominent in the field of cardiovascular disease, from congenital heart disease, coronary artery disease,

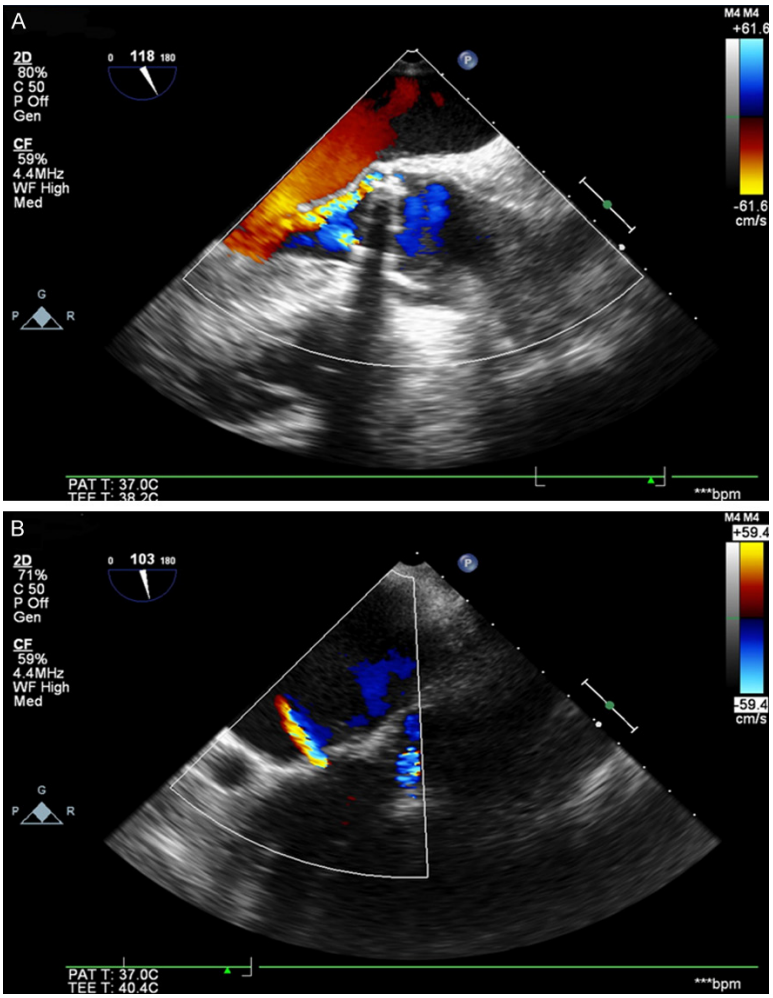

Figure 1. Echocardiography of paravalvular prosthetic regurgitation. A. Transesophageal echocardiogram showing para mitral valve regurgitation. B. Transesophageal echocardiogram showing para aortic valve regurgitation.

and even to valve disease. The advent and development of interventional radiology provides an alternative strategy for the treatment of PVLs. Although percutaneous transcatheter closure of PVLs has achieved a certain degree of success [5, 6], complications such as prosthetic impingement, bioprosthetic leaflet erosion, and device embolization have been reported. Prosthetic valve malfunction after closure of the paravalvular leak is one of the most common complications [7]. Rogers et al. reported a case of bioprosthetic leaflet erosion [8] and Kennedy et al. reported a case of symptomatic mitral stenosis [9] after a mitral paravalvular leak closure. Furthermore, late embolization is also a risk after paravalvular leak closure [10]. Optimal visualization of PVLs is the essential task of the procedure; however, a profound understanding of the anatomic and spatial characteristics of PVLs, the properties of different closure devices, and types and positions of previous prosthetic valves is the key to success. Therefore, in this study, we share our experience of percutaneous closure of PVLs. To assess results and outcomes, we focus on anatomic details, properties of devices, and types and positions of previous prosthetic valves.

Methods

Patients

Between January 2008 and December 2012, 17 patients were clinically diagnosed with PVLs by Doppler echocardiography (**Figure 1**). Two patients with additional cardiac pathology were excluded (one with severe tricuspid regurgitation and the other with coronary artery disease requiring coronary artery bypass surgery) because open heart surgery was determined to be the more appropriate treatment. Another two patients were excluded because of active infective endocarditis. The remaining 13 patients were eligible for percutaneous closure of PVL if they met the following selection criteria that developed on the basis of our experience. All patients needed to meet the first two criteria and one of the latter three:

(1) The prosthetic valve was fixed on the annulus.

(2) There was no neoplasm or thrombus on the prosthetic valve, and no thromboembolic events had occurred in the previous month.

(3) The patients suffered congestive heart failure caused by PVL, whose symptoms and signs could not be improved by medical treatment. Risk assessment for redoing valve replacement was high.

Table 1. Patient characteristics (*n* = 13)

Characteristic	Value
Age, y (mean ± SD)	51.6±11.4
Male/Female	10/3
BMI (mean ± SD)	24.9±2.9
Treated prosthesis type	
Aortic/Mitral	4/9
Bioprosthesis/Mechanical prosthesis	6/7
Interval since last surgery, months (mean ± SD)	76.2±74.6
Range, months	6-240
No. of previous operations (mean ± SD)	1.2±0.6
Presenting symptoms	
Heart failure	10
Hemolytic anemia	3
History of IE	2
Original disease	
Congenital disease	3
Rheumatic heart disease	9
Degenerative valvular disease	1
Medical history	
Pulmonary hypertension	11
Systemic hypertension	5
Atrial fibrillation	5
Coronary artery disease	1
Prior stroke	1
Permanent pacemaker	0
Predicted reoperative mortality (%)	7.5±4.5

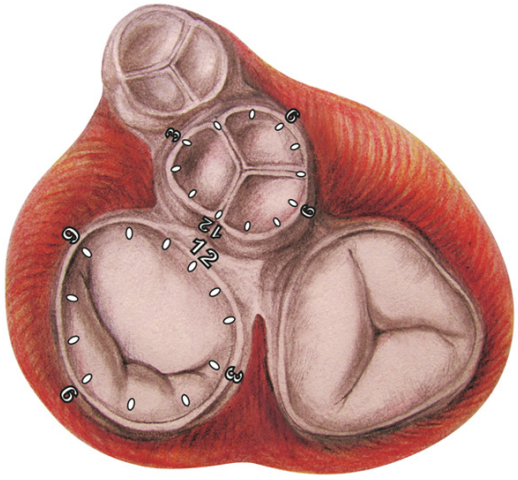

Figure 2. Clock-face designation of the mitral and aortic valves. The mitral-aortic fibrous continuity corresponds to 12 o'clock in both clocks. For the aortic valve, the noncoronary cusp is between 8 o'clock and 12 o'clock, the left coronary cusp is between 12 o'clock and 4 o'clock, and the right coronary cusp is between 4 o'clock and 8 o'clock.

(4) Mild paravalvular prosthetic regurgitation caused by PVL complicated with infective endocarditis.

(5) Progressive severe hemolysis anemia caused by PVL, and reoperation was deemed dangerous.

The general characteristics of patients are summarized in **Table 1**.

Clinically hemolytic anemia was defined as anemia (hemoglobin ≤ 11 g/dL in women and ≤ 12 g/dL in men; lactate dehydrogenase ≥ 600 mg/dl) requiring blood transfusions and/or erythropoietin injections to maintain hemoglobin ≥ 10 g/dl, excluding diagnosis of any other source of blood loss. The definition of heart failure was New York Heart Association (NYHA) functional class III or IV. Surgical risk was assessed by EuroSCORE [11]. Anatomic location of the PVLs adopted the clock-face format which is a widely accepted surgical nomenclature [12, 13] (**Figure 2**). Technical success was defined as resolution of paravalvular prosthetic regurgitation by deployment of an occlusive device across the PVL without any mechanical interference with the valve prosthesis or surrounding structures, as checked by intra-operative TEE, and no procedure-related outcome events occurring during the procedure and within the subsequent 24 h; such events include cardiovascular death, thromboembolic events (i.e. myocardial infarction, stroke and other organ embolism), cardiac tamponade, atrioventricular block, and emergency conversion to surgery.

The study conformed to the ethical guidelines of the Declaration of Helsinki and was approved by the institutional review board of our hospital. All patients were advised of the procedural risks and the limited data on clinical efficacy of the procedure as well as the off-label use of all closure devices. A written informed consent was obtained from each patient.

Technical approach to transcatheter PVL closure

For mitral PVL, the procedure was performed in either the anterograde or retrograde style. In the anterograde pattern, under general anesthesia, a pigtail catheter was inserted via a

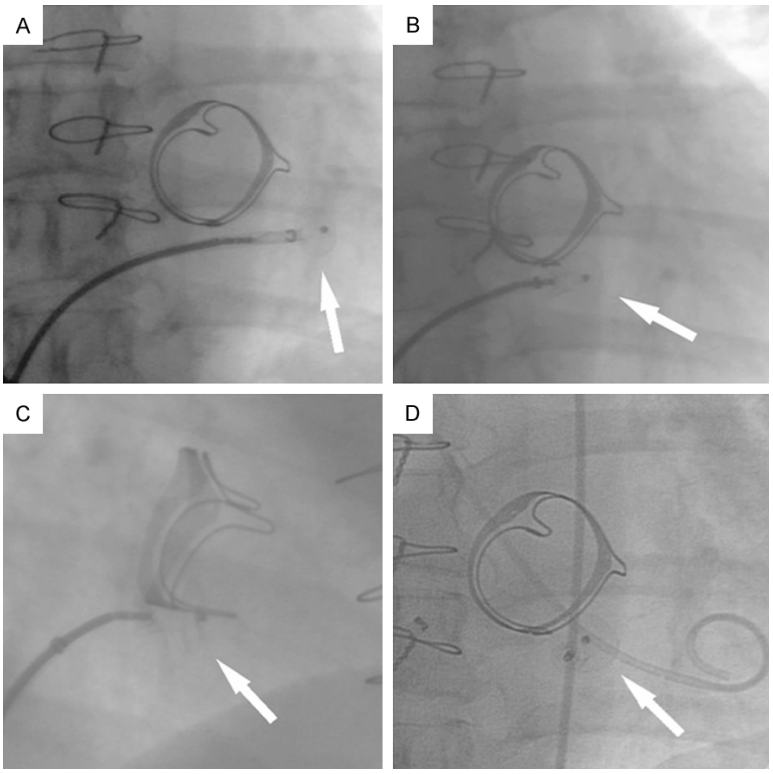

Figure 3. Transcatheter closure of mitral bioprosthetic paravaluvar leakage. A. The disk of an Amplatzer Duct Occluder is open (arrow). B. The retention skirt is well seated at the edge of the valve (arrow). C. The Duct Occluder is fully deployed and pulled firmly against the orifice of leakage (arrow). D. The position of the open Duct Occluder is confirmed by fluoroscopy (arrow).

femoral artery puncture. Left ventriculography was performed in combination with TEE and transthoracic echocardiography (TTE) to figure out the position and size of the PVL. The active clotting time was maintained at 300 s by heparinization. Through the inferior vena cava, a Schwarz catheter was crossed into the left atrium via a septal puncture. Along the Schwarz catheter, a cobra catheter was advanced into the left atrium. Reference contrast with an appropriate projection view was established. A guide wire was advanced through the PVL into the left ventricle and then ascended to the aorta to establish a loop. The guide wire was exchanged with a stiff supporting wire. A delivery sheath was inserted over the supporting wire and an occlusion device (Amplatzer) was deployed. Left ventriculography and TTE examination was conducted to confirm the disappearance of the PVL and restoration of normal valve function (**Figure 3**).

In the retrograde approach, under general anesthesia, a pigtail catheter was inserted through a femoral artery puncture. Left ventriculography was performed in combination with TEE and TTE to figure out the position and size of the PVL. The active clotting time was maintained at 300 s by heparinization. Then a super-slip guide wire was advanced through the femoral artery puncture to the ascending aorta to the left ventricle (LV) and then through the PVL to the left atrium (LA). If imaging data indicated a PVL of appropriate size and position, a femoral vein puncture could be selected. If the PVL was close to the neighboring atrial septum, a jugular vein pathway was selected and septal puncture was conducted to advance the snare to the LA. A loop was established and the PVL was closed by following the above-mentioned procedure. Left ventriculography and TTE examination was conducted to confirm the disappearance of the PVL and restoration of normal valve function.

For aortic PVLs, the procedure was performed in retrograde fashion. Under general anesthesia, a pigtail catheter was inserted through a femoral artery puncture. Ascending aortography in combination with TEE was performed to figure out the position and size of the PVL. The active clotting time was maintained at 300 s by heparinization. A multipurpose catheter was placed above the aortic valve via the femoral artery. Reference contrast with appropriate projection view was established. A guide wire was advanced into the LV through the PVL then exchanged with a super-stiff supporting wire (**Figure 4**). The delivery system was inserted and the occluder was properly deployed (**Figure 5**). Aortography and TEE was conducted to confirm absence of residual shunt and that valve function was restored. The technical details are listed in **Table 2**.

Perioperative management

In general, all patients were periprocedurally administered intravenously injected unfractionated heparin (85 IU/kg), aiming for an active

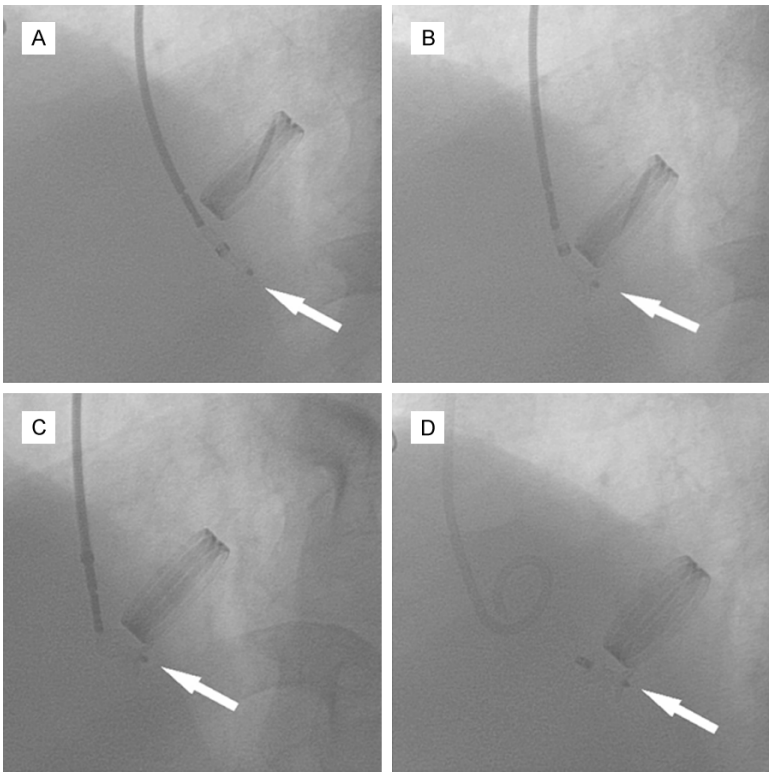

Figure 4. Transcatheter closure of aortic mechanical paravaluvar leakage. A. The distal disk of a Muscular VSD Occluder is open (arrow). B. The retention skirt is confirmed to be well-seated at the edge of the valve, and the waist of the device is deployed in the leak (arrow). C. The proximal disk of the Muscular VSD Occluder is deployed and pulled firmly against the orifice of the leak (arrow). D. The position of the open Muscular VSD Occluder is confirmed by fluoroscopy (arrow).

clotting time of > 300 s. In all patients, the immediate results were evaluated by fluoroscopy and TEE just after the devices were deployed. For patients with a mechanical valve, warfarin was administered for anticoagulation for life. Patients with bioprosthetics were administered warfarin for 6 months. International Normalized Ratio (INR) was tested once a week and anticoagulant dose adjusted as needed to keep the value between 1.5 and 2.5.

Follow-up

All patients were routinely evaluated 30 days after the procedure with TTE to assess cardiac hemodynamic and morphological parameters, including LA diameter, left ventricular end-diastolic diameter (LVED), right atrium (RA) diameter, right ventricular end-diastolic diameter (RVED), pulmonary artery (PA) inner diameter, pulmonary artery pressure (PAP), and ejection fraction (EF). Vital status and adverse events of all patients were determined by regular telephone contact and clinical visits.

Statistical analysis

Continuous variables are expressed as mean ± standard deviation. Paired Student's *t*-test was used for comparison of hemodynamic and morphological parameters. Statistical analyses were performed using the SPSS 20.0 software package. Statistical significance was inferred at $P < 0.05$.

Results

Of the 13 procedures performed, six addressed defects in biological valve prostheses (46%) and seven in mechanical valve prostheses (54%). Nine defects involved the mitral valve (69%) and four (31%) defects were periaortic. According to the clock-face diagram in **Figure 2**, 33% of the leaks treated were located between 2 o'clock and 10 o'clock clockwise, and 67% were between 10 o'clock and 2 o'clock. A total of 12 Amplatzer devices were used, including 5 Muscular VSD Occluders (42%), 6 Vascular Plug II Occluders (50%), and 1 Duct Occluder (8%). Average fluoroscopy time was 75.9±8.4 mins, average amount of contrast agent used was 65.2±9.0 mL. Average length of stay after procedure was 7.1±3.1 days. The predicted reoperative mortality was 7.5±4.5%, according to the EuroSCORE. One patient needed blood transfusions before the procedure.

Immediate results

We attempted closure of PVLs in 13 patients, with permanent device placement in 12 defects. Overall, technical success was achieved in 12 patients (92%).

The one failure to deploy the occlusive device was because we were not able to navigate across the defect with the delivery system. This

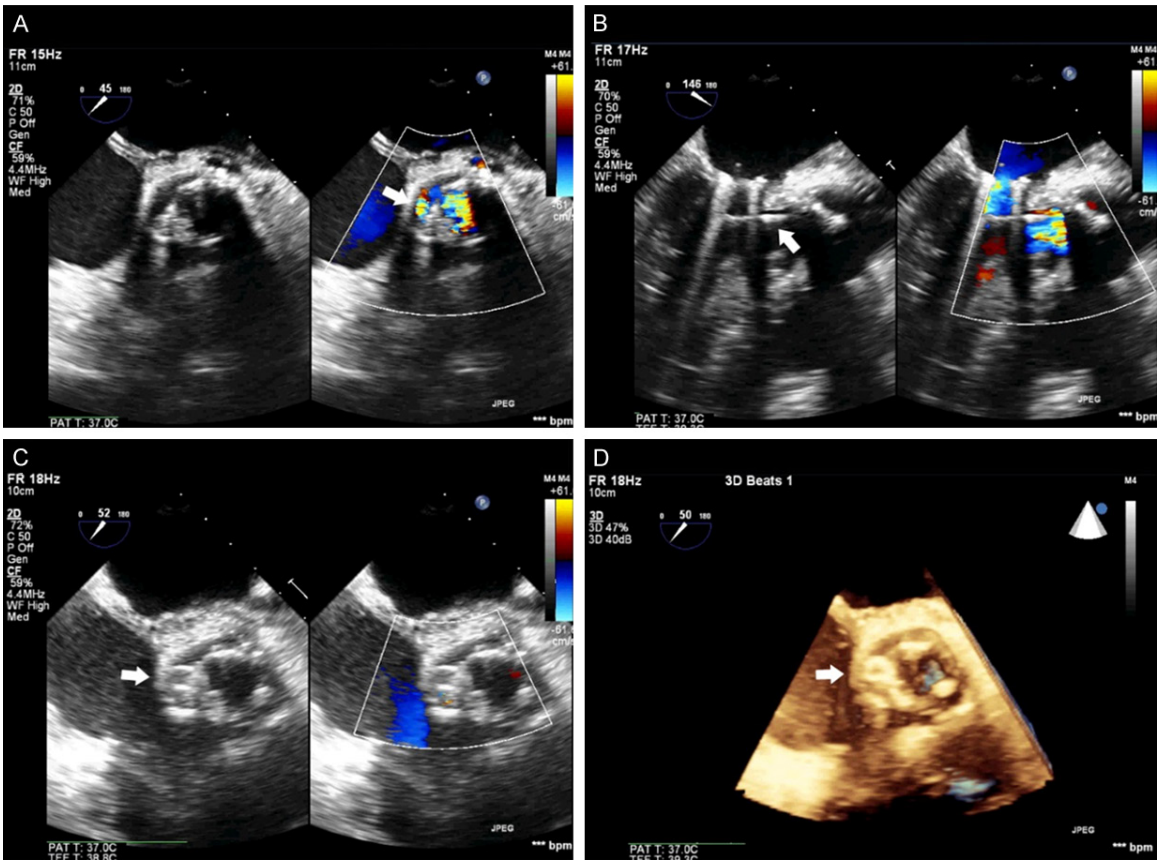

Figure 5. Transesophageal echocardiography (TEE) guidance of percutaneous aortic paravalvular leak (PVL) closure. A. TEE shows the aortic paravalvular leak (arrow). B. Delivery of catheter across PVL (arrow). C. Amplatzer Duct Occluder in place (arrow). D. Real-time 3D TEE shows the occluder in place (arrow).

patient had a crescent-shaped defect located between 2 o'clock and 4 o'clock around a mechanical mitral valve combined with atrial fibrillation. Pre-operational TTE showed that the LA diameter was 87 mm and LVED was 70 mm. The patient refused to have valve replacement redone, and received medical therapy. The heart function of this patient deteriorated progressively, and the patient was hospitalized several times due to repeated heart failure during 2 years' follow-up.

Another patient had two periaortic defects around a mechanical valve. One was located between 10 o'clock and 2 o'clock, with a longest axis of 3 mm; the other was located between 4 o'clock and 8 o'clock, with a longest axis of 2 mm. The former was closed by retrograde transaortic placement of a 4-mm Muscular VSD Occluder. Post-procedural TEE revealed that residual paravalvular regurgitation was mild and was of no hemodynamic significance. The second procedure was deemed dispensable. Regular follow-up showed this patient to have satisfactory heart function.

There was no prosthetic leaflet impingement during the procedure in any of the patients. There were no thromboembolic events, atrioventricular block, or emergency conversion to surgery within the subsequent 24 h.

Follow-up

The overall major adverse event rate at 30 days was 8% (1/12). One patient, who was complicated with prior stroke and coronary artery disease, died suddenly in hospital 7 days after successful percutaneous treatment of a defect associated with a 29-mm bioprosthetic mitral valve. The implanted device was an 8-mm Vascular Plug II. In other patients, no severe complications (hemolytic or thromboembolic events) or bioprosthetic or autologous valve erosion occurred at 30 days. LVED and PAP were decreased significantly compared with

Table 2. Technical details

Characteristic	Value
Perivalvular defects	
No. of closures attempted	13
Number of patients with mitral PVL	4
Number of patients with aortic PVL	9
Number of patients with aortic PVL and mitral PVL	0
Size of defect (longest axis), mm	5.5±1.4
PVL locations	
2 o'clock-6 o'clock-10 o'clock	8
10 o'clock-2 o'clock	4
No. with device implanted	12
Total no. of devices implanted	12
Patients with multiple defects closed	0
Amplatzer occluder device used	
Vascular Plug II	6
Duct Occluder	1
Muscular VSD Occluder	5
Contrast agent used, mL (mean ± SD)	65.2±9.0
Fluoroscopy time, min (mean ± SD)	75.9±8.4
Procedure success	12
Length of stay after procedure, days (mean ± SD)	7.1±3.1

pre-operation echocardiographic assessment (from 62.9±16.2 to 59.2±16.1 mm, and from 41.145±10.2 to 34.9±8.9 mmHg). There was no statistical difference between post-operation and pre-operation values in LA, RA diameter, RVED, PA inner diameter, and EF (**Figure 6**).

During long-term follow-up, 1 patient died at 13 months due to progressive heart failure. This patient was a 49-year old male complicated with hypertension. Pre-procedure TTE showed that LVED was 102 mm and EF was 15%; NYHA functional class was IV. He received successful percutaneous treatment of a defect associated with a 25-mm mechanical aortic valve. The implanted device was a 7-mm Muscular VSD Occluder. TTE at 30 days after the procedure showed that LVED was decreased to 99 mm and EF was increased to 20%.

An improvement in NYHA functional class of at least one grade was noted in five patients (42%). No patient required blood transfusion after the procedure. The survival rate for patients after closure of PVL was 83%.

Discussion

Our investigation indicated that percutaneous transcatheter closure of PVLs can be applied with a relatively high rate of technical success and an acceptable incidence of complications in carefully selected patients. The procedure was conducted as a therapeutic strategy for patients who were deemed to be at high risk for surgery. We achieved a technical success rate of 92% in our investigation.

The procedure failed in one patient who had a large LA diameter (87 mm) and large LVED (70 mm). In a completely interventional method, it is very difficult in such a large atrial or ventricular space for the guide wire to establish a support passing through the PVL, no matter whether an anterograde or retrograde style is attempted. Another possible option might be a transapical approach, where the point of entry into the left ventricle is carefully planned, in a hybrid cardiac surgical procedure, if it is possible. Therefore, the percutaneous repair strategy chosen in each case depends on the unique anatomy; location, shape, and size of the PVL; position of the valve; and type of prosthesis.

PVL of the aortic valve makes the blood return to the left ventricle in diastole causing an increase in LVED and reducing the effective or forward stroke volume. This leads to an increase in LV end-diastolic pressure (LVEDP), and then results in increased LA pressure and PAP. PVL of the mitral valve results in high LA pressure, which can also lead to pulmonary edema and high PAP. The left ventricle adapts to the substantial regurgitant volume by increasing LV end-diastolic volume which eventually leads to LV dilatation and shape change. The closure of PVL, no matter whether associated with the mitral or aortic valve, reduced preloading of LV end-diastole volume and LA pressure which resulted in decreased LVED and PAP after the procedure. TTE at 30 days after the procedure demonstrated significant improvement in both morphology and hemodynamic parameters, and none of the above-mentioned complications were observed to occur.

Careful identification of the anatomy of the defect, including its relationship with surrounding structures, with the assistance of fluorogra-

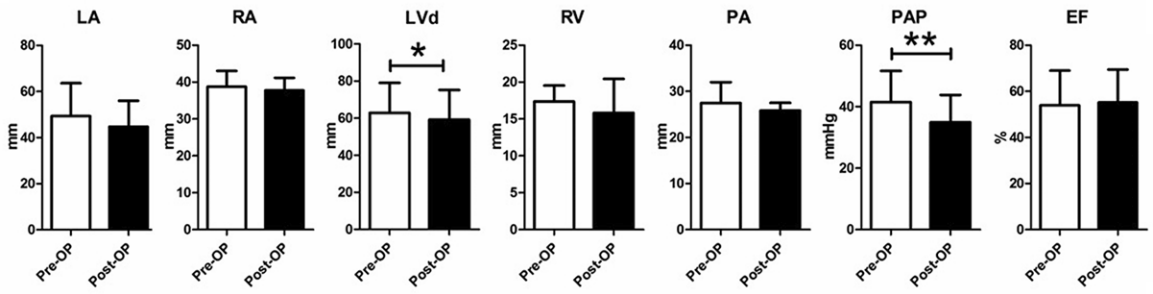

Figure 6. Short-term follow-up by transthoracic echocardiography compared with pre-operative echocardiography. Pre-OP: Pre-operation; Post-OP: 30 days post-operation; LA: Left atrial diameter; RA: Right atrial diameter; LVd: Left ventricular end-diastolic diameter; RV: Right ventricular diameter; PA: Pulmonary arterial inner diameter; PAP: Pulmonary artery pressure; EF: Ejection fraction; $*P < 0.05$; $**P < 0.01$.

phy and TEE, especially real-time 3D TEE [14], is essential. Analysis of patient risk factors and individualized selection of the device are also necessary. Because one part of a PVL is the annulus of the prosthetic valve, PVLs are usually of a crescent or irregular shape, and few are round.

When mitral or aortic PVLs are located in the area between 10 o'clock and 2 o'clock, in the vicinity of the central fibrous body and the left fibrous trigone, careful attention should be paid during the procedure. First of all, deployment of the closure device near the central fibrous body may press on the membranous part of the interventricular septum which could lead to atrioventricular block or left bundle branch block. Secondly, this area contains the anterior cusp annulus of the original mitral valve and is adjacent to the left coronary sinus and non-cusp coronary sinus of the aortic valve. Therefore, we need to be particularly careful to make sure that the closure device does not affect prosthetic valve movement and neighboring leaflets. Mild interruption of neighboring leaflets is difficult to detect during the operation because it scarcely interferes with hemodynamics. However, with valve opening and closing during the cardiac cycle, the autologous valve might contact the closure device, which may lead to potential perforation or even damage, and patients would have to undergo redo surgery in the end. Valve perforation or damage may occur anywhere during the interventional closure of bioprosthetic valve PVL due to the above-mentioned causes, ultimately giving rise to long-term treatment failure [8, 9]. Interruption of mechanical valve movement may lead to severe disturbance of hemodynamics, but this can always be found immediately after deployment of the closure device and be corrected in time [7]. As to large PVLs, redo surgical valve replacement is still the best management. A large PVL could result in a rocking valve, and in theory, the PVL would become larger and larger under the action of the shearing force resulting from the rocking valve [15, 16].

In the current study, the Amplatzer Vascular Plug II and Amplatzer Muscular VSD Occluder were the most commonly used devices because of their suitable characteristics. At present, no given devices are specifically designed for PVLs. Devices that have already been applied for closure of PVLs include the Rashkind double-umbrella [17], Gianturco coils [18], Gianturco-Grifka vascular occlusion device [19], Amplatzer Duct Occluder [20], Amplatzer Vascular Plug [21], Amplatzer coils [22], Amplatzer Septal Occluder [23], and Amplatzer Muscular VSD Occluder [24]. Among them, Amplatzer devices are widely used because they can be retrieved into the sheath and redeployed.

The Amplatzer Septal Occluder has a short connecting waist and large disk/waist ratio. The center point of the device can shift easily within the defect. The larger disk is supposed to be in the left atrium and it could easily interfere with prosthetic valve movement if it is deployed in the left ventricle [1, 8]. For these reasons, it is rarely applied for PVLs. The Amplatzer Duct Occluder is a cone-shaped device with a retention skirt on the aortic side. It has a severe limitation in that the retention skirt must be placed in a high-pressure chamber in order to prevent the device falling off, which restricts its use in PVL closure. The Amplatzer Muscular VSD

Occluder is a symmetrical double-disk device whose connecting waist is 7 mm long and ranges in diameter from 4 to 18 mm. The model with the 4-mm waist diameter has a disk diameter 5 mm larger than the waist (i.e., 9 mm); in all other models, the disk diameter is 8 mm larger than the waist. This means that, with these devices, the rim attaching to the adjacent valve struts or leaflets is 2.5 mm minimum or 4 mm maximum, which reduces the chances of impinging on the surrounding structure or prosthetic leaflets. The Amplatzer Vascular Plug II has been used frequently in the past 5 years. It has oval shaped disks and waist [21] with a 2-mm rim for attaching. Although not designed specifically for PVL, taking into consideration its features overall, it seems preferable for PVL closure, especially in situations where the space between the PVL and surrounding structures is small. For relatively large or crescent-shaped defects, the simultaneous or sequential deployment of any combination of two or more smaller devices is better than a single large device because the risk of impingement on the prosthetic leaflets or surrounding structure is minimized [24, 25].

In this series, only 13 patients were involved. Thus, further study of a broader group of patients is required to add weight to the outcomes of percutaneous repair of PVLs. Continued long-term follow-up is also needed to establish a comprehensive therapy strategy for PVLs integrating interventional, surgical, and hybrid methods.

There are some limitations to our study. First, all of the patients in this study received valve replacement and percutaneous transcatheter closure of PVLs in our center; therefore, this study is the experience of a single cardio center. The indications and contraindications summarized from our experiences can be of reference for the broader application of interventional closure of PVL, but the guidelines remain in need of further investigation through large and multicenter case analyses. Second, we only used Amplatzer devices for PVL closure. How these outcomes compare with those of devices from different manufacturers for interventional closure of PVL is not known. Third, a larger group of patients and longer term outcomes of interventional closure of PVL remains essential, especially for closure of bioprosthetic valve leakage.

Conclusions

In our study, a relatively high rate of technical success and an acceptable incidence of complications were achieved. The hemodynamics and morphology of the heart were improved. Our experience suggests that percutaneous transcatheter closure of PVL is feasible and safe with careful PVL anatomic analysis and appropriate selection of patients and devices. Nevertheless, studies involving a larger group of patients and longer term follow-up are required.

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Disclosure of conflict of interest

None.

Address correspondence to: Dr. Jianshi Liu, Department of Cardiac Surgery, Tianjin Chest Hospital, Taierzhuangnan Road 291#, Jinnan District, Tianjin 300222, China. Tel: +86-022-88185074; Fax: +86-022-88185074; E-mail: Jianshi_Liu@hotmail.com

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