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CASE REPORT

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Rupture of a calcified right ventricle to pulmonary artery homograft by balloon dilation– emergency rescue by venus P-Valve

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Abstract

Background Percutaneous pulmonary valve implantation (PPVI) is a recognized alternative treatment to surgery for patients with dysfunctional right ventricular outflow tracts. Patient selection is essential to avoid serious complications from attempted treatment, such as rupture or dissection, especially of the calcified outflow tracts. We describe a case with an unexpected rupture of a calcified homograft valve and main pulmonary artery, which was treated successfully by emergency implantation of a self-expanding Venus P-Valve (Venus MedTech, Hangzhou, China) without the need for pre-stenting with a covered stent.

Case details A 13-year-old boy had two previous operations of tetralogy of Fallot, one a total repair and the other a homograft valved conduit for pulmonary regurgitation. He presented with dyspnea and severe right ventricular outflow tract obstruction (RVOTO) and had a calcified outflow tract and main pulmonary artery. In the catheter laboratory, a non-compliant balloon dilation resulted in a contained rupture of the conduit. The patient remained hemodynamically stable, and the rupture was treated with a self-expandable Venus P-Valve without the need for a covered stent combined with a balloon-expandable valve or a further surgical procedure.

Discussion Preprocedural evaluation with an inflating balloon is necessary to examine tissue compliance and determine suitability for PPVI. However, this condition is accompanied by a risk of conduit rupture. Risk factors of this complication are calcification and homograft use. These ruptures are mostly controlled with a prophylactic or therapeutic covered stent, with a low rate of requiring surgery. However, there are severe ruptures which lead to hemothorax and death. In the available literature, there was no similar reported case of conduit rupture, which a self-expandable Pulmonary valve stent has managed. It seems that fibrosis and collagen tissue around the heart, formed after open surgeries, can contribute to the control of bleeding in these cases.

Conclusion (clinical Learning Point) The suitability of patients for the PPVI procedure should be examined more carefully, specifically patients with homograft and calcification in their conduit. Furthermore, conduit rupture might be manageable with self-expandable artificial pulmonary valves, specifically in previously operated patients, and the applicability of this hypothesis is worth examining in future research.

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Keywords Congenital heart defect, Tetralogy of Fallot (TOF), Right ventricular outflow tract (RVOT) obstruction, Calcified right ventricular outflow conduit rupture, Venous P-valve

Introduction

Congenital anomalies, which involve the pulmonary valve or right ventricular outflow tract (RVOT), such as tetralogy of Fallot (TOF), truncus arteriosus (TA), and pulmonary atresia (PA), are seen in almost one-fifth of CHD cases [1]. The optimal time for surgical correction of RVOT anomalies, such as patch augmentation of RVOT, inserting bioprosthetic valves, and valved conduits, is usually in the first few months after birth [2]. Many patients then require subsequent repeat operations because of RVOT dysfunction [3]. A large number of these patients are treated by percutaneous pulmonary valve implantation (PPVI) [4], a procedure that has replaced repeat open heart surgery in a significant percentage of patients since its first introduction in 2000 by Bonhoeffer et al. [5]. With the increasing use of different valves, good results have been obtained [4, 6]. However, there is a potential for life-threatening complications such as coronary vessel occlusion or obstruction or rupture of previously implanted conduits, which may have calcified [7].

During the PPVI procedure, assessment is needed to ensure that the coronary arteries are not at risk of compression and subsequent risk of possible myocardial infarction. This requires balloon interrogation of the RVOT with a non-compliant balloon combined with selective left coronary angiography. If the inflated balloon compresses the coronary arteries, PPVI should not be performed [8]. Furthermore, another risk is the possibility of rupture of the calcified conduit. The balloon is inflated in the conduit to test for adequate relief of the obstruction before PPVI. One of the methods that can be used for the evaluation of the suitability of the structure for the PPVI procedure is cardiac imaging by cardiac Computed Tomography (CT) scan or magnetic resonance imaging (CMRI). High levels of conduit calcification have been shown to elevate the likelihood of conduit injury associated with PPVI. Therefore, previous literature has advised carefully considering these factors before performing interventions on patients with pulmonary valve dysfunction [9]. The risk with this approach is irreversible coronary artery compression, which then requires emergency surgery [9, 10]. The advantage is that PPVI can relieve the obstruction and prevent pulmonary regurgitation. Stenosed calcified conduits and homografts are risk factors for conduit rupture [10].

We report on a 13-year-old boy with a history of cardiac surgery consisting of repair of TOF and reoperation of RVOT with a homograft conduit. The conduit had become severely calcified and stenosed, and balloon interrogation of the homograft resulted in its rupture. It was treated with a self-expandable Venus P-Valve without complications during or after the procedure.

Case Details

A 13-year-old boy was presented to the Emergency Department (ED) with progressive shortness of breath, which was worse during physical activity. The symptoms had become more severe recently, and he was in NYHA class III. He was presented at the age of 3 months with cyanosis, and tetralogy of Fallot (TOF) was confirmed. Soon afterward, a complete surgical repair of TOF was performed. At the age of 6 years, pulmonary valve replacement (PVR) surgery was performed with a homograft conduit (*Pulmonary homograft 27 mm)*. Cardiac catheterization had been performed six months before his recent presentation and had shown an increased RV systolic pressure of 70 mmHg, with moderately severe RVOT stenosis. On examination, there was a loud ejection systolic murmur at the upper left sternal border radiating to the back. Echocardiography confirmed severe RVOT stenosis (Doppler systolic velocity of more than 4 m/sec) and pulmonary regurgitation (Table 1). He was admitted from the ED for further cardiac catheterization and assessment for possible PPVI.

Cardiac catheterization was performed with conscious sedation, and the right femoral vein and femoral arterial access were obtained. The hemodynamic assessment showed that RV systolic pressure was 75mmHg. Compared with femoral arterial pressure (SBP:100 mmHg / DBP:65 mmHg). The mean PA pressure was 15 mmHg, and the gradient across RVOT was 45 mmHg. The angiography in the RV showed severe stenosis in the mid-main Pulmonary Artery (MPA) with calcification. It was decided to assess for coronary artery compression with a non-compliant balloon before proceeding with PPVI. Based on the angiographic measurements, a balloon-expandable valve of 28 mm diameter was considered an option. A 0.035" Lunderquist guidewire was positioned in the right lower PA, and an Altosa-Gemini balloon of 28 mm diameter and 50 mm length was slowly inflated up to 6 atmospheres, resulting in the abolition of the waist on the balloon. The angiography in the RVOT in the anteroposterior projection appeared satisfactory, but a contained rupture of the calcified homograft was noticed in the lateral projection (Fig. 1).

As the patient was hemodynamically stable and there was no significant pericardial effusion on echocardiography, the team, including the surgical team, decided after discussion to attempt a self-expanding Venus P-Valve.

Table 1 Transthoracic echocardiographic measures of the patient before the procedure:

Chambers sizes and ventricular function	Mild to moderate RAE & RVE, Mild RV dysfunction, TAPSE = 1.3 cm Normal LA & LV size Moderate TR PG = 73mmHg, No MR. Moderate PI PHT = 108msec, Mild PS, PPG = 103mmHg, MPG = 64mmHg. No AI-No AS mild LV systolic dysfunction(LVEF:45%)
Valves	Normally Related Great Arteries, Moderate TR PG = 75mmHg, No MR. Moderate PI PHT = 108msec, PS PPG = 103mmHg, MPG = 64mmHg. No AI-No AS
SVC and IVC size	Normal size SVC & IVC
Pericardial Effusion	There is no PE

***RAE**: Right atrial enlargement, **RVE**: Right ventricular enlargement, **TAPSE**: Tricuspid annular plane systolic excursion, **LA**: Left atrium, **LV**: Left ventricle, **TR**: Tricuspid regurgitation, **PG**: Pressure gradient, **MR**: Mitral regurgitation, **PI**: Pulmonary insufficiency, **PHT**: Pressure half time, **PS**: Pulmonary stenosis, **PPG**: Peak pressure gradient, **MPG**: Mean pressure gradient, **AI**: Aortic insufficiency, **AS**: Aortic stenosis, **LV**: Left ventricle, **LVEF**: Left ventricle ejection fraction, **SVC**: Superior vena cave, **IVC**: Inferior vena cava, **PE**: Pericardial effusion*

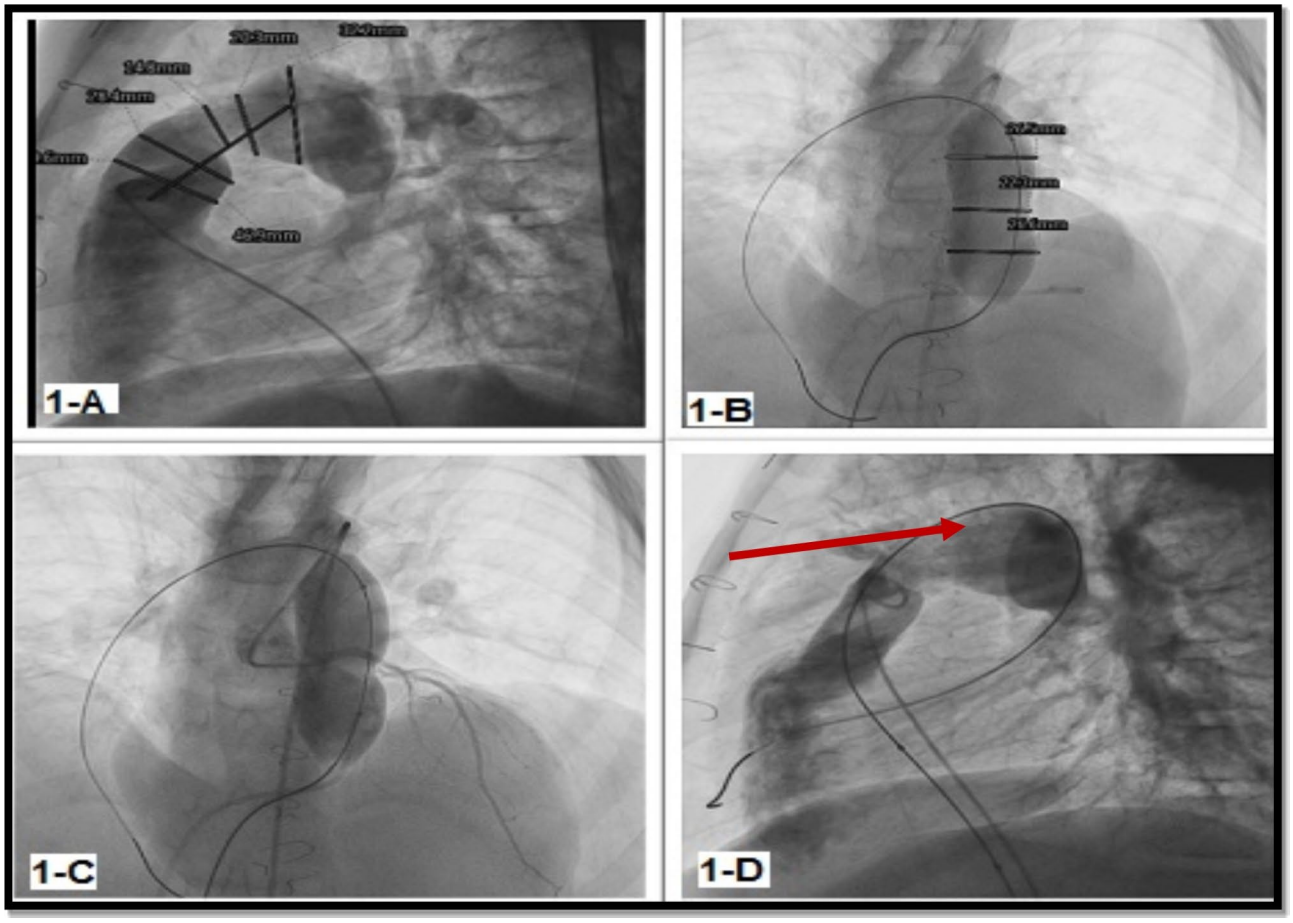

Fig. 1 **A**) Right ventricle angiogram in left lateral projection showing severe stenosis of the mid-main pulmonary artery. **B**) Anteroposterior-cranial frame showing balloon (AltosaSa-XL PTA- Reference number: AL-28-50, LOT number: LN002P100-30) inflation. **C**) The Anteroposterior-cranial frame shows an inflated Altosa-Gemini balloon with a simultaneous selective left coronary angiogram and a patent left coronary system. **D**) main pulmonary artery angiogram in left lateral projection showing a contained rupture of this artery at the level of previous stenosis (red arrow).

This approach was chosen over implanting one or more covered stents to control the rupture, followed by inserting a balloon-expandable THV. Over the Lunderquist guidewire in the RPA, a 26 Fr DrySeal sheath (Gore company) was passed through the RVOT. However, passing the sheath around the RVOT and MPA was difficult despite repeated attempts. At one point, the sheath would not pass beyond mid-MPA, so a check angiogram was performed. This showed that the DrySeal sheath had passed into the contained rupture. The position of the guidewire was changed to the left lower lobe PA, using a 5 Fr multipurpose catheter passed through the sheath. A further attempt was made to pass the sheath, but it was stuck in the rupture again. It was decided to attempt passing a Venus P-valve (34−30) as its carrot may facilitate the passage of the whole system beyond the rupture

into LPA. Since the assembly could not be relocated, with a maneuver, the Venus P-valve and sheath were pulled back gently and were finally passed behind the rupture, utilizing a wire. After checking, angiography showed that the rupture was still contained and that the valve assembly was distal. The distal flare of the Venus P-Valve was exposed, and gradually, the middle portion and the proximal flare were deployed successfully in a good position. The final check of RV angiography showed complete sealing of the ruptured conduit, and MPA angiography showed a competent Venus P-valve. Final hemodynamics showed an MPA pressure of 15 mmHg and RV systolic pressure of 40 mmHg, with a nonsignificant gradient across the Venus P-Valve. Coronary angiography showed normal coronary arteries without coronary compression (Fig. 2).

The patient was transferred to the Cardiac Care Unit (CCU) and was hemodynamically stable. Cardiac computed tomographic angiography (CCA) was performed 48 h after the procedure and showed no mediastinum leak or other abnormality. The patient was discharged home 72 h after the procedure (Fig. 3). At follow-up one month after discharge, his exercise tolerance and dyspnea had improved considerably. Echocardiography showed trivial pulmonary regurgitation and mild to moderate tricuspid regurgitation with a 25 mmHg peak pressure gradient. The peak pressure gradient for the tricuspid valve before the PPVI was 75 mmHg. The Peak Doppler gradient across the pulmonary valve was 25 mmHg, and there was right atrial and right ventricular enlargement with mild RV dysfunction, similar to before the PPVI (Table 2).

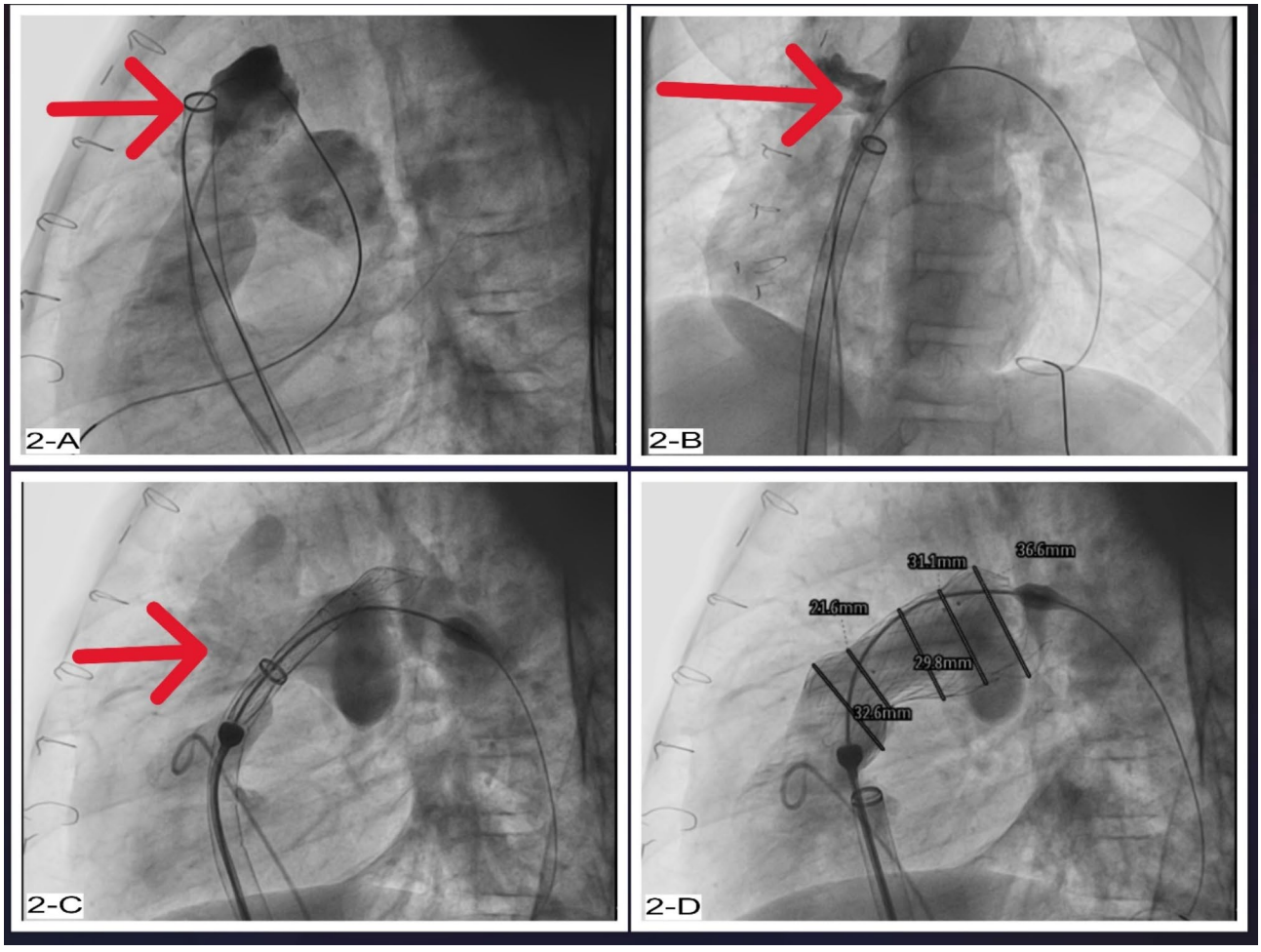

Fig. 2 **A**) Angiogram in left lateral projection through DrySeal sheath in the main pulmonary artery rupture showing contained contrast. **B**) The main pulmonary artery angiogram in the left anterior oblique LAO-cranial projection shows the DrySeal sheath in the main pulmonary artery rupture. LPA stenosis can be seen in this figure. **C**) lateral projection frame showing VenusP-Valve assembly passed through the DrySeal sheath beyond the main pulmonary artery rupture. **D**) The right ventricle angiogram in the left lateral projection shows the satisfactory position of VenusP-Valve and a completely sealed rupture in the main pulmonary artery.

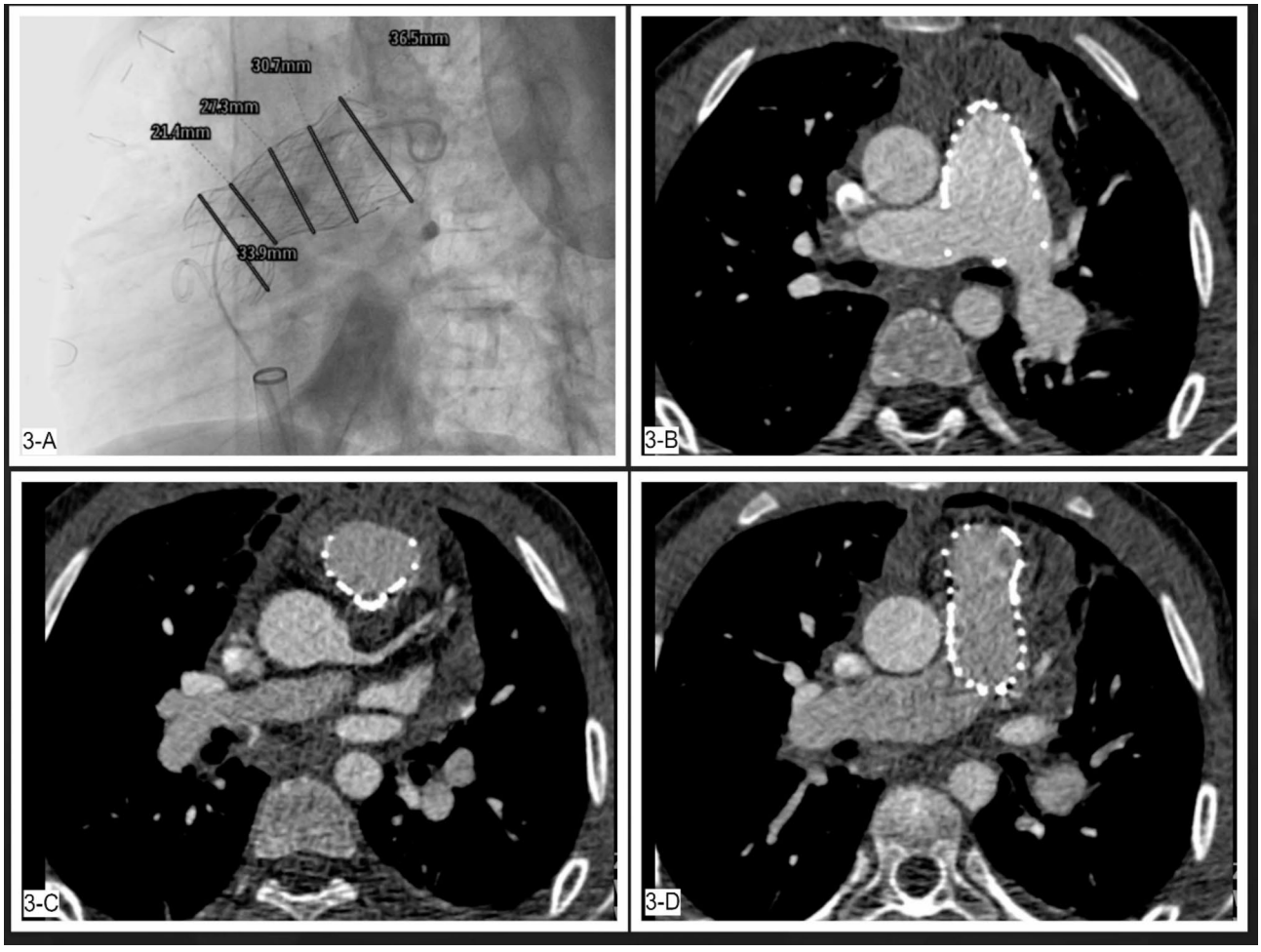

Fig. 3 **A)** The frame still shows measurements of different levels of VenusP-Valve, **B**: Left pulmonary artery stenosis is shown in this figure, **C**, **D)** Cardiac computed tomography angiography shows soft tissue density with a cystic component. Air is seen in the anterior mediastinum without enhancement in favor of post-operation changes (suggestive of hematoma)

Table 2 Echocardiographic features of the patients one month after the procedure

Chambers sizes and ventricular function	Mild RAE & RVE, Mild RV dysfunction, TAPSE = 1.6 cm, LV systolic dysfunction (LVEF:45%),
Valves	Normally Related Great Arteries, Normal motion Bioprosthetic pulmonary valve, NO PS, Trivial PI, Mild to moderate TR, PPG = 25mmHg MPG = 14mmHg
SVC and IVC size	Normal size SVC & IVC.
Pericardial Effusion	Minimal pericardial effusion around LV apex = 4 mm,

RAE: *Right atrial enlargement*, ***RVE***: *Right ventricular enlargement*, ***TAPSE***: *Tricuspid annular plane systolic excursion*, ***LA***: *Left atrium*, ***LV***: *Left ventricle*, ***TR***: *Tricuspid regurgitation*, ***PG***: *Pressure gradient*, ***MR***: *Mitral regurgitation*, ***PI***: *Pulmonary insufficiency*, ***PHT***: *Pressure half time*, ***PS***: *Pulmonary stenosis*, ***PPG***: *Peak pressure gradient*, ***MPG***: *Mean pressure gradient*, ***AI***: *Aortic insufficiency*, ***AS***: *Aortic stenosis*, ***LV***: *Left ventricle*, ***LVEF***: *Left ventricle ejection fraction*, ***SVC***: *Superior vena cave*, ***IVC***: *Inferior vena cava*, ***PE***: *Pericardial effusion*

Discussion

In patients with previously repaired tetralogy of Fallot and conduits, RVOT stenosis is not uncommon, while in patched RVOTs, PR may be the dominant lesion [3, 4]. Residual RVOT obstruction may be amenable to balloon dilation or stent implantation, which can lead to PR in many patients. This may then need PPVI or surgery; the latter may be associated with higher risks because of multiple operations previously as well as in the future because of valve degeneration or calcification of implanted valves [11, 12]. RVOT and pulmonary valve dysfunction (both stenosis and regurgitation) may result in RV and RA enlargement and tricuspid valve regurgitation, leading to RV dysfunction. Untreated, this can progress to LV dysfunction, heart failure, and an increased rate of sudden cardiac death [11, 12]. Therefore, early treatment of abnormal anatomy and hemodynamics is important to prevent irreversible progression. Two

available therapeutic options include conventional reoperation and percutaneous pulmonary valve implantation (PPVI). The latter has been shown to be an effective and safe therapeutic option over the last two decades in selected patients [10]. To make a decision between surgical treatment or PPVI, some determining factors include the safety and appropriate anatomy for PPVI, such as pulmonary valve annulus size, suitability of RVOT and PA anatomy, and risk of coronary artery compression caused by high-pressure RV-PA conduit expansion [13–15]. Moreover, one of the probable concomitant pathologies with this condition is pulmonary artery stenosis, which can be symptomatic (dyspnea, activity restriction); they should be addressed as well, considering the risk of complications such as sudden cardiac death [16].

One part of the Venus-P valve structure is the nitinol stent. These parts lead to the advantageous feature of being physically adaptable and being able to adapt to the pulmonary arterial trunk (PAT) without compressing neighboring structures [17]. The available sizes include a range between 28 and 36 mm in diameter with 2 mm increases. One of the most serious complications of homograft implantation is conduit rupture, which might be seen in up to one-tenth of the patients. Therefore, after each balloon inflation, it is crucial to look for RVOT injuries to find and treat the injury that has occurred [18]. Nowadays, PPVI can be performed with either self-expandable or balloon-expandable valves in catheterization laboratories, under general anesthesia or sedation. Although the safety and efficacy of PPVI have been shown, they are associated with known complications, which can be life-threatening in some cases [4, 19]. Preprocedural balloon inflation is needed in PPVI for two reasons: firstly, to check the distance of the coronary arteries from the PPVI location and, therefore, assess the risk of compression, and secondly, to identify the most suitable location and size of the PPVI [20]. On the other hand, balloon interrogation may result in severe complications such as conduit rupture and fracture of the stent if one is already implanted [10, 21]. One of the methods that can be used to evaluate the structure's suitability for the PPVI procedure is cardiac imaging by cardiac CT scan or magnetic resonance imaging (CMRI). High levels of conduit calcification have been shown to elevate the likelihood of conduit injury associated with PPVI. Therefore, previous literature has advised carefully considering these factors before performing interventions on patients with pulmonary valve dysfunction [9]. With PPVI, there is an acceptable risk of requiring surgical reintervention in the future, most commonly due to high post-procedure RVOT gradient and post-deployment stent recoil or compression or conduit rupture. However, pre-stenting of RVOT before the PPVI may be associated with a lower risk of the need for reintervention [22–24].

With regard to complications of PPVI, in a systematic review by Sohaib A. et al., conduit rupture was the most common procedural complication of PPVI, with an incidence of 2.6%. This is a potentially life-threatening complication, as it can lead to massive hemothorax and need emergency surgical intervention to save the patient's life [25]. In another review, Ansari M. et al. estimated that the incidence of RVOT rupture was as high as 9% in some studies [1]. However, Jeffrey W. et al., in a study of 616 patients who had undergone transcatheter pulmonary valve replacement (TPVR), concluded that conduit rupture was uncommon, and most of them were minor and could be controlled with covered stents and did not require surgery. However, the progression of the ruptures is not predictable, and some patients may be hemodynamically unstable and need emergency surgery [1]. They noted that the highest risk of conduit injury was in stenotic homografts [26]. It has been demonstrated that calcified and stenotic homografts are specific risk factors for this type of rupture. The preprocedural evaluation should be performed carefully in patients with these risk factors, and interrogating the RVOT by implanting a covered stent as a prophylactic measure may be protective against this potentially catastrophic complication [10, 27].

Most cases of conduit ruptures are managed with covered stents or open heart surgery [28]. However, rarely reported, some cases of minor flaps or dissections can also be managed conservatively [27]. When dealing with such complex patients, it may be worth considering the implantation of a covered stent and gradual inflation with increasing balloon diameters for interrogation until such diameters are reached, at which point a balloon-expandable valve can be implanted. Such an approach requires frequent selective coronary angiography to ensure patency at each diameter and repeated RV or PA angiograms to exclude any rupture.

Conclusion

To our knowledge, none of the cases of conduit rupture reported previously have been managed with self-expandable valves. Usually, covered stent implantation is performed, followed by implantation of balloon-expandable valves. Sometimes, more than one covered stent may be required as the rupture can extend. Our patient had had two previous open-heart operations. Hence, a significant amount of fibrosis and scarring around the heart is likely to have resulted in controlled bleeding and prevention of hemopericardium, tamponade, or hemothorax. Different lengths and diameters of Venus P-Valve are available, so ruptures can likely be contained more easily with such a valve. Therefore, it is worth considering this valve to deal with such a scenario.

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None.

Author contributions
PE contributed to data curation, supervision, and project administration. HM and MA contributed to data collection, analysis, supervision, and manuscript review. AF and PD contributed to data collection, analysis, and data curation. SQ and PB contributed to data analysis, writing the initial draft, and revising the final manuscript. All authors read and approved the final version of the manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethical approval
The study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments. The study was approved by the Ethics Committee of Rajaei Hospital of Tehran. Considering not including any revealed information about the patient and the breach of confidentiality, the committee waived the requirement for an ethics code.

Consent for publication
Written informed consent was obtained from the patient's legal guardian for publication of this study and accompanying images.

Competing interests
The authors declare no competing interests.

Consent to participate
Written informed consent was obtained from the patient's legal guardian.

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