Case Report

Transcatheter Pulmonary Valve Implantation in a Repaired Tetralogy of Fallot Patient with Prosthetic Valve Stenosis

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Abstract

Tetralogy of Fallot (TOF) is the most common cyanotic congenital heart disease, and many patients require reintervention after surgical repair because of right ventricular outflow tract (RVOT) or pulmonary valve (PV) dysfunction. We report the case of a 34-year-old male with repaired TOF and prior prosthetic PV replacement who presented with dyspnea and palpitations. Imaging revealed severe prosthetic PV stenosis with preserved left ventricular function and a suitable RVOT anatomy. The patient underwent successful valve-in-valve transcatheter PV implantation (TPVI) using a 24.5 mm MyVal™ prosthesis through the Python XL sheath. Balloon testing excluded coronary compression, and the gradient decreased from approximately 70-16 mmHg. Postprocedure, the patient showed rapid symptomatic relief and sustained valve performance at 6 weeks. This case underscores the safety and minimally invasive nature of TPVI as an alternative to repeat surgery in selected patients with TOF.

Keywords: MyVal transcatheter valve, prosthetic pulmonary valve stenosis, Python XL sheath, tetralogy of Fallot, transcatheter pulmonary valve implantation, valve-in-valve

Introduction

Tetralogy of Fallot (TOF) is the most common cyanotic congenital heart disease, occurring in approximately 3 of 10,000 live births. It is characterized by the four features: ventricular septal defect, overriding aorta, right ventricular hypertrophy, and right ventricular outflow tract (RVOT) obstruction.[1] Standard repairs included VSD patch closure and RVOT relief. Transcatheter pulmonary valve implantation (TPVI) has recently emerged as a minimally invasive alternative to surgical pulmonary valve replacement (PVR) in patients with dysfunctional RVOT.[1] TPVI is increasingly preferred in selected patients,[1,2] although only a small proportion of patients undergo this intervention. Currently, only 20%–25% of patients requiring RVOT reconstruction and PVR are suitable for TPVI.[3] This case underscores TPVI as a feasible and less invasive option for managing RVOT dysfunction in patients with TOF repair.

Case Report

A 34-year-old male with Type II diabetes mellitus presented with progressive fatigue, exertional dyspnea (New York Heart Association class II), and palpitations. He had a history of congenital heart disease, specifically TOF with absent pulmonary valve (PV) syndrome, and underwent intracardiac repair in childhood with PVR using a bioprosthetic valve in 2009. Upon examination, his blood pressure was 138/90 mmHg, pulse rate was 132/min, respiratory rate was 22/min, and oxygen saturation was 98% on room air. The systemic examination results were also unremarkable. Electrocardiography revealed normal sinus rhythm with right ventricular hypertrophy and strain pattern, complete right bundle branch block, right axis deviation, and right atrial enlargement. Echocardiography showed severe prosthetic PV stenosis (peak gradient ~70 mmHg and Vma × 4.2 m/s) with right ventricular dilatation and preserved left ventricular (LV) ejection fraction (EF) (~60%). Computed tomography (CT) angiography revealed severe prosthetic PV stenosis with

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restricted leaflet mobility and calcification. Multiplanar reconstruction confirmed a reduced effective orifice area and elevated gradients across the prosthesis, consistent with severe prosthetic valve stenosis, and adequate landing zone measurements (minimum diameter, 21–23 mm; maximum diameter, 27–30 mm; area, ~380–450 mm²; and perimeter, ~72–78 mm). These findings confirmed adequate annular sizing to accommodate a 24 mm MyVal transcatheter valve. The clinical and imaging findings fulfilled the criteria for intervention in view of severe stenosis, worsening symptoms, and risk of progressive right ventricular remodeling [Figure 1].

As shown in Figure 1, fluoroscopic images demonstrating the sequential steps of TPVI in a 34-year-old male with repaired TOF and prosthetic PV stenosis.

After obtaining informed consent from the high-risk patient and multidisciplinary heart team approval, TPVI was performed under fluoroscopic guidance via right femoral venous access. A Python XL sheath was advanced into the main pulmonary artery (PA) for stable valve delivery purposes. A Lunderquist wire was positioned in the left PA for additional support because of the prior implantation of a 23 mm bioprosthetic valve (internal diameter 21 mm). Existing Biocor Stents (St. Jude Medical Inc.) was fractured using a 22-mm Z-Med balloon to allow the expansion of the new prosthesis. A 24.5-mm MyVal™ valve (Meril Life Sciences, India) was deployed across the dysfunctional prosthetic valve, followed by postdilatation with a 26-mm balloon. Coronary protection and occlusion tests confirmed unobstructed left coronary flow. Final angiography demonstrated an optimal valve position, no paravalvular leak, and preserved coronary perfusion. Immediate postprocedure echocardiography showed a significant gradient reduction (mean 16 mmHg; Vma × 2.5 m/s). The patient received preprocedural antibiotic prophylaxis with intravenous ceftriaxone, followed by the administration of unfractionated heparin during the procedure to maintain adequate anticoagulation. After implantation, aspirin 150 mg daily was initiated as antiplatelet therapy to facilitate transcatheter valve endothelialization and reduce the thrombotic risk.

The patient experienced rapid symptomatic improvement and was discharged the next day. At the 6-week follow-up, echocardiography confirmed stable valve performance with velocities of 2.0–2.5 m/s, mean gradients of 11–16 mmHg, preserved LV systolic function (EF ~60%), and no chamber dilatation. Mild tricuspid regurgitation persisted, with structurally normal mitral and aortic valves and no effusion, thrombi, or vegetation.

Discussion

TPVI was first described by Bonhoeffer *et al*. in 2000, establishing a minimally invasive alternative to surgical reintervention for RVOT dysfunction.[4] This pioneering work culminated in the development of the Melody Transcatheter PV (Medtronic Inc., Minneapolis, MN, USA), which subsequently received U.S. Food and Drug Administration Humanitarian Device Exemption approval in 2010 and full Premarket Approval in 2015 for use in dysfunctional RVOT conduits4. Since then, TPVI has evolved into an established therapeutic option for selected patients with conduit or prosthetic PV dysfunction, offering the advantages of earlier intervention, reduced perioperative morbidity, and expedited recovery compared with open heart surgery.

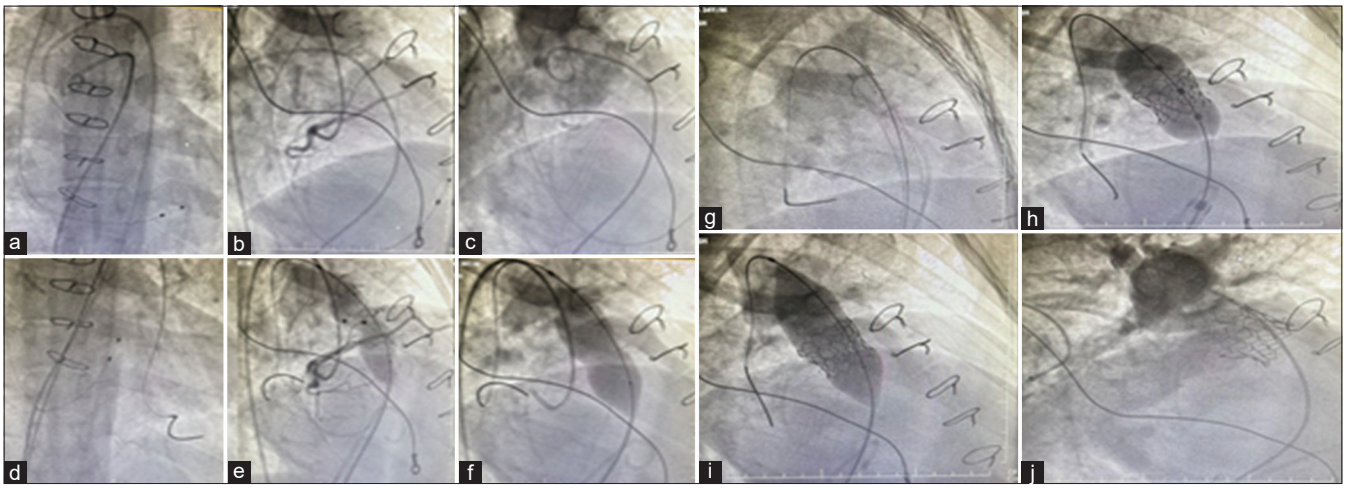

Figure 1: Fluoroscopic images demonstrating sequential steps of transcatheter pulmonary valve implantation (TPVI) in a 34-year-old male with repaired Tetralogy of Fallot and prosthetic pulmonary valve stenosis. (a) Initial fluoroscopy showing previously implanted prosthetic pulmonary valve and vascular access sheath *in situ.* (b) Coronary angiogram performed to assess risk of coronary compression before valve intervention. (c) Wire positioning into the left pulmonary artery for procedural stability. (d) Advancement of Python XL sheath into the main pulmonary artery. (e) Multiple guidewire stabilization including Wanderlust wire across the dysfunctional prosthesis. (f) Balloon sizing and coronary protection catheter in place to exclude coronary compression. (g) Positioning of the crimped 24.5 mm MyVal transcatheter valve across the stenotic prosthetic pulmonary valve. (h) Deployment of the MyVal valve under fluoroscopic guidance. (i) Post-dilatation of the valve using a 26-mm balloon to optimize expansion and reduce gradients. (j) Final fluoroscopic image showing well-seated valve with optimal expansion and unobstructed coronary flow

According to the 2020 European Society of Cardiology guidelines, PVR either surgical or transcatheter is indicated in symptomatic patients following TOF repair who exhibit severe pulmonary regurgitation and/or at least moderate RVOT obstruction (Class I, Level C). When anatomical conditions permit, a transcatheter PV approach is the preferred option in patients without a native RVOT (Class I) and is likewise recommended in those with symptomatic right ventricle–to–PA conduit stenosis defined by a right ventricular systolic pressure greater than 60 mmHg or lower if accompanied by impaired flow and/or severe pulmonary regurgitation (Class I, Level C).[5,6]

The optimal outcomes depend on stringent patient selection and thorough procedural planning, incorporating multimodal imaging, such as transthoracic echocardiography and cardiac CT, along with an invasive hemodynamic evaluation. The TPVI procedure generally includes establishing venous access, progressive dilation of the conduit, coronary compression testing using balloon inflation, and placement of a covered stent to provide a secure landing zone and reduce the risk of conduit rupture before valve deployment. Despite these standardized steps, the procedure is not universally applicable; anatomical complexities may predispose patients to adverse events, including coronary artery compression, conduit rupture, stent fracture, or paravalvular leak, underscoring the importance of individualized anatomical and clinical assessment before TPVI is undertaken.

The clinical results observed in this patient demonstrated the efficacy and safety of TPVI for prosthetic PV stenosis. The marked reduction in the transvalvular gradient and immediate improvement in RVOT hemodynamics was consistent with international experience. In the Melody TPV IDE trial, involving 171 patients, freedom from reintervention and explantation at 5 years was 76% ± 4% and 92% ± 3%, respectively, while another cohort of 148 patients followed for a median of 4.5 years confirmed durable valve performance with low complication rates.[7] These outcomes validate TPVI as a reliable alternative to surgical reoperation in appropriately selected patients.

Historically, patients with recurrent PV stenosis or regurgitation following TOF repair underwent repeat open-heart surgery, a treatment pathway associated with cumulative morbidity, increased mortality risk, extended hospitalization, and prolonged convalescence.[8] In contrast, TPVI offers a less invasive catheter-based solution with shorter hospital stays, reduced procedural complications, and rapid symptomatic improvement, including relief from fatigue, dyspnea, and palpitations, which may be sustained over time.[9] These advantages are particularly relevant for patients with multiple prior sternotomies, in whom the surgical risk is markedly increased.

In parallel, ongoing technological innovations have further optimized the procedural outcomes. Dedicated tools, such as the Python XL sheath, used in this case, enhance procedural safety by providing robust multilevel wire support and stable valve delivery, even in anatomically complex RVOT settings.[10] Moreover, the expanding array of valve designs compatible with diverse RVOT anatomies continues to increase operator flexibility and long-term procedural success.[11]

Overall, TPVI is reshaping the management paradigm for survivors of congenital heart disease who require PV re-interventions. As device technology, imaging capabilities, and procedural expertise continue to advance, TPVI is expected to play an increasingly prominent role in reducing the need for repeat surgeries and improving long-term patient outcomes. The current case reinforces these trends and underscores the growing applicability and regional adoption of TPVI in contemporary practices.

Conclusion

This case highlights TPVI as a safe, minimally invasive alternative to repeat surgical replacement in patients with repaired TOF and prosthetic valve dysfunction. The procedure achieved excellent hemodynamic outcomes, marked gradient reduction, rapid symptom relief, and sustained valve performance. With ongoing advancements in valve technology, imaging, and delivery systems, TPVI is emerging as the preferred option for selected patients requiring PV reintervention, offering a better quality of life and lower procedural risk than surgery.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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