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Balloon Pulmonary Valvuloplasty for Severe Late Residual Pulmonary Valve Stenosis Following Tetralogy of Fallot Repair

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Abstract

Residual valvular pulmonary stenosis late after complete repair of tetralogy of Fallot (TOF) is uncommon, and experience with balloon pulmonary valvuloplasty (BPV) in this setting is limited. A 23-year-old man who had undergone TOF repair at four years of age presented with a one-year history of progressive exertional dyspnoea (New York Heart Association class II) and atypical chest pain. Transthoracic echocardiography showed a peak instantaneous right ventricular (RV)-to-pulmonary artery gradient of 106 mmHg and impaired RV systolic function, with a tricuspid annular plane systolic excursion (TAPSE) of 10 mm. Cardiac catheterization showed an RV systolic pressure of 97 mmHg (RV-to-systemic pressure ratio 0.80), a peak-to-peak gradient of 70 mmHg and a doming pulmonary valve without significant subvalvular obstruction. Single-balloon valvuloplasty with an 18-mm balloon reduced the RV systolic pressure to 60 mmHg (ratio 0.44) and the peak-to-peak gradient to 30 mmHg, with only mild pulmonary regurgitation. Before discharge, the Doppler gradient was 40 mmHg, TAPSE had improved to 14 mm and his symptoms had improved. In selected patients with repaired TOF whose residual obstruction is predominantly valvular, BPV can provide substantial haemodynamic and symptomatic benefit and may defer repeat surgery. Accurate localization of the obstruction and conservative balloon sizing are essential.

Categories: Medical Education, Cardiology, Cardiac/Thoracic/Vascular Surgery
Keywords: adult congenital heart disease, balloon pulmonary valvuloplasty, cardiac catheterization, pulmonary valve stenosis, right ventricular dysfunction, tetralogy of fallot, transcatheter intervention

Introduction

Tetralogy of Fallot (TOF) accounts for approximately 7-10% of congenital heart malformations [1]. Survival after complete repair is excellent, but residual haemodynamic lesions remain an important cause of late morbidity and reintervention, for which lifelong surveillance is required [2-4]. While pulmonary regurgitation is the dominant late lesion, residual or recurrent valvular pulmonary stenosis is less frequent and can persist or progress when the native pulmonary valve is preserved rather than replaced or disrupted by a transannular patch [2-4]. Although often clinically insidious, progressive valvular narrowing imposes a chronic pressure load on an already operated right ventricle, carrying significant risks of progressive right ventricular (RV) hypertrophy, systolic dysfunction, exercise intolerance, arrhythmia, and sudden cardiac death [3,4].

Balloon pulmonary valvuloplasty (BPV), which relieves obstruction by mechanically dilating a narrowed or commissurally fused valve via catheter-guided balloon inflation, has been the treatment of choice for isolated native valvular pulmonary stenosis since 1982 and is well-established in adolescents and adults [5,6]. Conversely, published evidence supporting BPV for late residual valvular stenosis after surgical TOF repair remains sparse, consisting largely of isolated case reports [7]. Unlike isolated native pulmonary stenosis, post-repair cases are complicated by prior RV outflow tract surgery, altered annular geometry, potential multilevel obstruction, and the risk of exacerbating pulmonary regurgitation in a previously operated right ventricle [3,4,7]. We describe a young adult with severe late residual valvular stenosis nearly two decades after TOF repair who was treated successfully with BPV, highlighting the diagnostic imaging criteria and conservative sizing strategy required for safe intervention [7].

Case Presentation

A 23-year-old man had undergone complete surgical repair of TOF at four years of age, with preservation of the native pulmonary valve. Postoperative residual valvular stenosis with a Doppler gradient of approximately 40 mmHg had been managed conservatively, though serial intermediate echocardiographic measurements during adolescence were unavailable prior to his current referral. He presented with progressive exertional dyspnoea (New York Heart Association (NYHA) class II) and intermittent atypical chest pain of one year's duration, for which he was initially evaluated by pulmonary medicine. After respiratory and parenchymal causes were excluded, he was referred to cardiology for evaluation of his repaired congenital heart disease.

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On examination, His pulse rate was 64 beats/min (reference range: 60-100 beats/min), blood pressure 96/66 mmHg (reference range: 90-120/60-80 mmHg), and oxygen saturation 99% on room air (reference range: 95-100%); height was 162 cm and weight 52 kg. A grade 4/6 ejection systolic murmur was heard at the left upper sternal border. The remainder of the physical examination and routine laboratory investigations were unremarkable. The timeline of clinical events is shown in Table *1*.

Time point	Clinical Event & Management
Age 4 years	Complete surgical repair of tetralogy of Fallot with native valve preservation; residual valvular pulmonary stenosis (Doppler gradient ~40 mmHg) managed conservatively.
Age 22 years	Onset of progressive exertional dyspnoea (NYHA class II) and intermittent atypical chest pain.
Age 23 years, Hospital day 1	Cardiology evaluation after pulmonary causes excluded. ECG (RBBB, RVH with strain), CXR (cardiomegaly), TTE (peak gradient 106 mmHg, leaflet doming, TAPSE 10 mm, RV FAC 11%), and CTPA (mid-MPA 11.4 mm, no subvalvular/branch stenosis).
Hospital day 7	Cardiac catheterization (peak-to-peak gradient 70 mmHg; RV/systemic pressure ratio 0.80) and single-balloon pulmonary valvuloplasty with an 18 × 40 mm balloon (residual gradient 30 mmHg; ratio 0.44).
Before discharge	Pre-discharge TTE: peak gradient 40 mmHg, TAPSE 14 mm, mild PR; symptomatic improvement; discharged home.
Planned follow-up	Outpatient clinical and echocardiographic review at one month (pending at the time of reporting), followed by annual surveillance and cardiac MRI.

TABLE 1: Timeline of clinical events

CTPA: computed tomography pulmonary angiography; CXR: chest radiography; ECG: electrocardiogram; FAC: fractional area change; MPA: main pulmonary artery; NYHA: New York Heart Association; PR: pulmonary regurgitation; RBBB: right bundle branch block; RV: right ventricle; RVH: right ventricular hypertrophy; TAPSE: tricuspid annular plane systolic excursion; TTE: transthoracic echocardiography.

The electrocardiogram showed sinus rhythm, right axis deviation, complete right bundle branch block, and RV hypertrophy with a strain pattern. Chest radiography showed cardiomegaly (cardiothoracic ratio >0.60; reference value: <0.50). Transthoracic echocardiography demonstrated a thickened pulmonary valve with restricted systolic excursion and characteristic systolic leaflet doming, without significant subvalvular narrowing or heavy valvular calcification; the pulmonary annulus measured 16 mm.

Continuous-wave Doppler revealed a peak instantaneous RV-to-pulmonary artery (PA) gradient of 106 mmHg (corresponding to a back-calculated peak velocity of 5.15 m/s). RV systolic function was markedly impaired, with a tricuspid annular plane systolic excursion (TAPSE) of 10 mm (reference value: ⩾17 mm) and an RV fractional area change (FAC) of 11%, accompanied by mild tricuspid regurgitation and absent pulmonary regurgitation. Computed tomography pulmonary angiography (CTPA) confirmed that the narrowing was localized to the valvular level without subvalvular muscular obstruction, supravalvular ridge, or branch PA stenosis, demonstrating a narrow main PA (11.4 mm at its mid-segment) and an incidental persistent left superior vena cava.

Following multidisciplinary heart team discussion - and having excluded pulmonary, coronary, and multilevel outflow causes of his symptoms - cardiac catheterization with an intent to perform BPV was undertaken on hospital day seven under conscious sedation via the right femoral artery (6F) and right femoral vein (7F), with intravenous heparin (100 U/kg). Selective coronary angiography confirmed normal coronary artery origins and absence of obstructive disease or anomalous coronary course across the right ventricular outflow tract (RVOT). Baseline haemodynamic pressures obtained via catheter pullback were as follows: RV 97/1 mmHg (end-diastolic 11 mmHg; Figure *1A*), main PA 27/9 mmHg (mean 18 mmHg; Figure *1B*), and aorta 121/65 mmHg (mean 85 mmHg), yielding a peak-to-peak RV-PA systolic gradient of 70 mmHg (97 − 27 mmHg) and an RV-to-systemic systolic pressure ratio of 0.80.

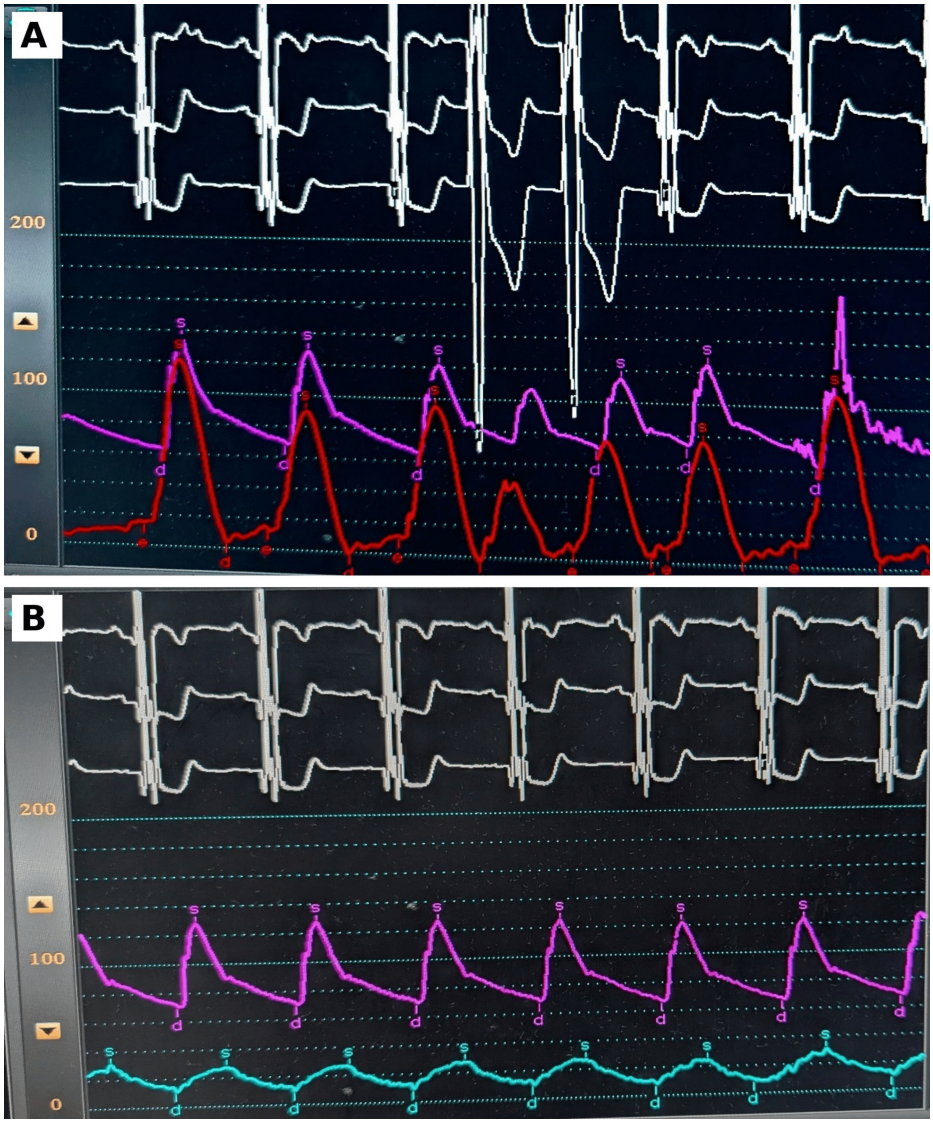


FIGURE 1: Haemodynamic recordings before balloon pulmonary valvuloplasty

(A) Simultaneous aortic (magenta) and right ventricular (red) pressure tracings demonstrating near-systemic right ventricular systolic pressure (97 mmHg vs. aortic 121/65 mmHg). Wide-complex beats represent catheter-induced ventricular ectopy. (B) Simultaneous aortic (magenta) and main pulmonary artery (cyan; 27/9 mmHg) pressure tracings confirming a 70 mmHg peak-to-peak transvalvular gradient.

Pressure scale: 0-200 mmHg (with 100 and 200 mmHg grid markers labeled and the 0 mmHg baseline at the bottom grid line); sweep speed: 25 mm/s; s: peak systolic pressure; d: early/lowest diastolic pressure; e: end-diastolic pressure immediately prior to systolic upstroke.

Right ventriculography in the right anterior oblique 15°/cranial 15° projection (Figure *2A*) and left anterior oblique 18°/cranial 18° projection (Figure *2B*) confirmed a thickened, doming pulmonary valve with restricted leaflet excursion and no significant dynamic or fixed subvalvular obstruction, establishing that the stenosis was predominantly valvular.

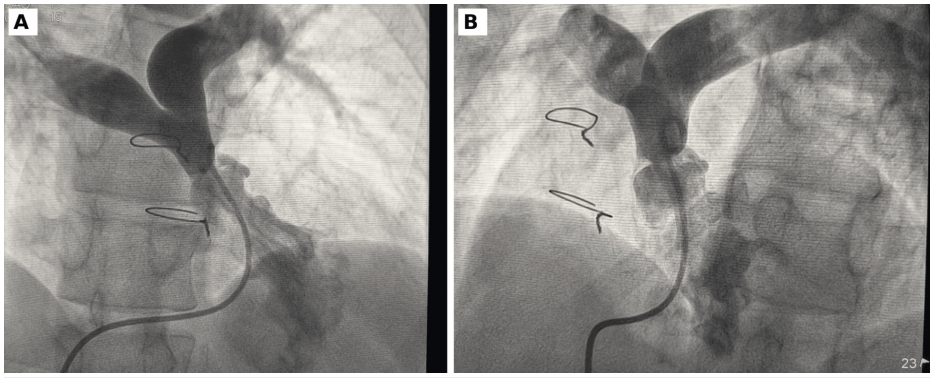


FIGURE 2: Angiography of the right ventricular outflow tract and pulmonary arteries

(A) Right anterior oblique 15°/cranial 15° and (B) left anterior oblique 18°/cranial 18° projections showing a thickened, doming pulmonary valve with restricted leaflet excursion (center of frame at the sinotubular junction) and preserved infundibular dimensions below the valve without subvalvular obstruction. Metallic sternal wires from the prior median sternotomy are visible anteriorly.

The pulmonary valve was crossed using a 6F Judkins Right catheter over a 0.035-inch hydrophilic guidewire (Terumo Corporation, Tokyo, Japan), which was exchanged for a 0.035-inch Amplatz Super Stiff guidewire (Boston Scientific, Marlborough, MA, USA) positioned in a distal branch PA (Figure *3*).

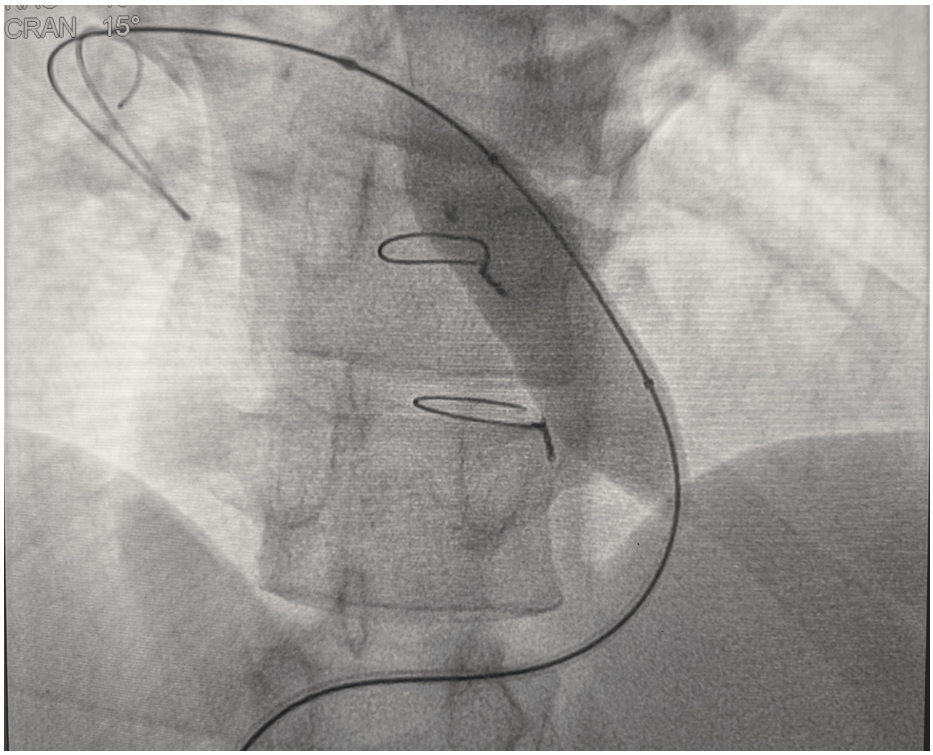


FIGURE 3: Guidewire position across the pulmonary valve before balloon dilatation

Right anterior oblique 15°/cranial 15° projection showing the 0.035-inch Amplatz Super Stiff guidewire coursing from the right atrium and right ventricle across the pulmonary valve, with its curved distal tip anchored in a distal branch pulmonary artery (upper-left lung field) to provide stable rail support for balloon delivery.

CRAN: cranial.

Given the 16 mm pulmonary valve annulus and narrow main PA, an 18 × 40 mm Atlas Gold balloon (Bard Peripheral Vascular, Tempe, AZ, USA) was selected. It yielded a conservative balloon-to-annulus ratio of

1.12-and inflated across the valve until the waist disappeared. Immediately post-dilatation, RV systolic pressure fell to 60 mmHg and main PA pressure was 30/3 mmHg, reducing the peak-to-peak systolic gradient to 30 mmHg (60-30 mmHg); against a simultaneous aortic pressure of 135/76 mmHg, the RV-to-systemic systolic pressure ratio dropped to 0.44. Repeat angiography demonstrated improved leaflet excursion without infundibular spasm, PA injury, or tricuspid valve damage, and only mild pulmonary regurgitation. Haemostasis was achieved by manual compression and a figure-of-eight subcutaneous suture without complications.

Pre-discharge transthoracic echocardiography (performed prior to discharge following post-procedural monitoring) showed a peak transvalvular velocity of 3.17 m/s (displayed as 317 cm/s on the Doppler readout), corresponding to a peak instantaneous gradient of 40 mmHg (Figure *4*), mild pulmonary regurgitation on colour-flow and continuous-wave Doppler assessment, and an early improvement in TAPSE from 10 mm to 14 mm.

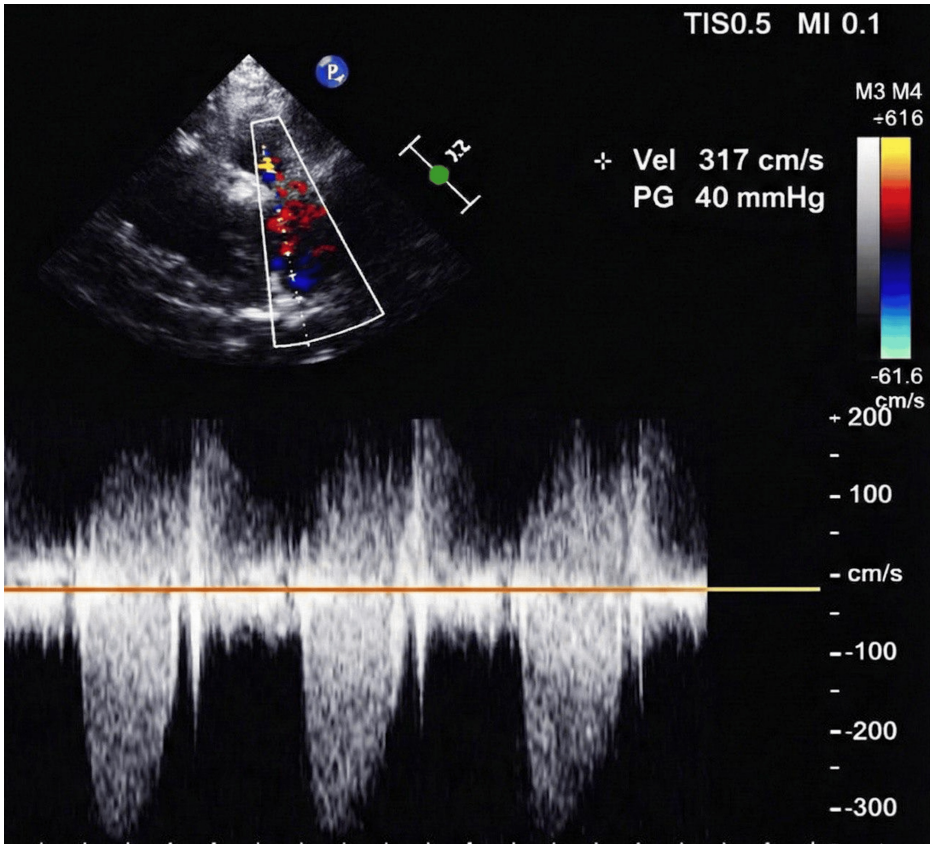


FIGURE 4: Continuous-wave Doppler across the pulmonary valve before discharge

Pre-discharge transthoracic echocardiography demonstrating a reduction in peak transvalvular velocity to 3.17 m/s (317 cm/s; peak instantaneous gradient 40 mmHg, down from 106 mmHg pre-procedure). Note that the peak negative spectral Doppler envelope extends slightly below the −300 cm/s display tick mark on the vertical axis, corresponding to the automated peak velocity readout of 317 cm/s.

Vel: velocity; PG: pressure gradient.

The patient reported marked improvement in exertional tolerance and was discharged in stable condition, with a further review planned at one month (not yet completed at the time of submission) and annually thereafter. Pre- and post-procedural haemodynamic and echocardiographic parameters are summarized in Table *2*.

Modality	Parameter	Pre-BPV (Baseline)	Post-BPV (Timing Specified)
Cardiac Catheterization	RV systolic pressure (mmHg)	97	60 (immediate post-BPV)
Cardiac Catheterization	PA systolic pressure (mmHg)	27	30 (immediate post-BPV)
Cardiac Catheterization	Peak-to-peak RV–PA gradient (mmHg)	70	30 (immediate post-BPV)
Cardiac Catheterization	RV-to-systemic systolic pressure ratio	0.8	0.44 (immediate post-BPV)
Transthoracic Echocardiography	Peak transvalvular velocity (m/s)	5.15*	3.17 (pre-discharge)
Transthoracic Echocardiography	Peak instantaneous gradient (mmHg)	106	40 (pre-discharge)
Transthoracic Echocardiography	TAPSE (mm)	10	14 (pre-discharge)
Transthoracic Echocardiography	RV fractional area change (%)	11	Not re-measured pre-discharge
Transthoracic Echocardiography / Angiography	Pulmonary regurgitation severity	Absent	Mild (immediate & pre-discharge)

TABLE 2: Pre- and post-procedural haemodynamic and echocardiographic parameters

*Pre-BPV peak transvalvular velocity (5.15 m/s, rounded from 5.148 m/s) was back-calculated from the directly measured pre-procedural peak instantaneous Doppler gradient (106 mmHg) using the simplified Bernoulli equation, whereas post-BPV peak velocity (3.17 m/s, displayed as 317 cm/s on the echocardiographic readout) was directly measured and corresponds to a rounded peak gradient of 40 mmHg (40.2 mmHg unrounded).

BPV: balloon pulmonary valvuloplasty; PA: pulmonary artery; RV: right ventricle; TAPSE: tricuspid annular plane systolic excursion.

Discussion

Improved survival after surgical TOF repair has produced a growing population of adults requiring lifelong specialized follow-up [2,8]. Current international guidelines recommend periodic multimodality assessment so that significant residual lesions are identified and treated before irreversible RV dysfunction ensues [3,4]. In our patient, a mild-to-moderate residual gradient tolerated for nearly two decades progressed to a degree of severe obstruction accompanied by exertional symptoms and marked RV systolic impairment (TAPSE 10 mm, RV FAC 11%), underscoring the necessity of continuous surveillance even for apparently stable residual gradients [3,4].

Residual RV outflow tract obstruction after TOF repair is frequently multilevel, involving subvalvular infundibular muscle, the pulmonary valve annulus or leaflets, the supravalvular main PA, or the branch PAs [3,9]. Because BPV relieves only the valvular component, establishing the predominant anatomical level of obstruction through multimodality imaging is critical prior to intervention [3,9]. In our clinical decision-making workflow, transthoracic echocardiography first identified systolic leaflet doming with restricted excursion, while CTPA excluded fixed subvalvular, supravalvular, or branch PA stenosis and defined the narrow mid-main PA diameter (11.4 mm). Diagnostic cardiac catheterization and biplane right ventriculography subsequently confirmed isolated commissural fusion without dynamic infundibular obstruction and excluded anomalous coronary anatomy. Given the severe gradient, symptoms, and depressed RV systolic function, a clear indication for intervention was warranted [3,4]. Bhyravavajhala et al. reported a similarly favourable immediate response to BPV in two adult patients with isolated residual valvular stenosis following TOF repair [7].

The baseline Doppler peak instantaneous gradient (106 mmHg) substantially exceeded the catheter peak-to-peak gradient (70 mmHg), an expected physiological distinction: continuous-wave Doppler measures the maximal instantaneous pressure drop during early systole (and may be further augmented by pressure recovery phenomena across a narrow main PA), whereas catheterization compares non-simultaneous peak systolic pressures in the RV and PA [10]. Following BPV, achieving a residual peak-to-peak gradient of 30 mmHg (pre-discharge Doppler gradient 40 mmHg) and reducing the RV-to-systemic pressure ratio from 0.80 to 0.44 implied an adequate procedural result [10,11]. Clinically, a residual peak-to-peak gradient ⩽30 mmHg or RV-to-systemic pressure ratio <0.50 substantially unloads the pressure-overloaded RV and relieves symptoms without necessitating surgical conduit or valve replacement [10,11]. Moreover, part of the immediate residual gradient frequently reflects reactive infundibular muscular hypertrophy, which typically regresses over months following successful relief of valvular stenosis, leading to further gradient reduction on serial follow-up [12].

While a balloon-to-annulus ratio of 1.2-1.4 is generally recommended for native isolated pulmonary valve stenosis to achieve effective commissural splitting while minimizing severe regurgitation and ventricular outflow tract injury [10,11], balloon sizing after TOF repair requires particular caution. In repaired TOF, chronic severe pulmonary regurgitation is a major driver of progressive RV dilatation, arrhythmia, and eventual need for pulmonary valve replacement [3,4]. Furthermore, because CTPA demonstrated a narrow main PA (11.4 mm), the chosen balloon size (18 mm; balloon-to-annulus ratio 1.12) was deliberately conservative to prevent main PA dissection or rupture while avoiding severe iatrogenic pulmonary regurgitation [10,11].

Regarding procedural durability, published series on native adolescent and adult BPV demonstrate sustained long-term gradient relief with freedom from reintervention exceeding 80-85% at 10-15 years [6,10]. However, mid- to long-term outcome data specifically for BPV in post-TOF repair residual valvular stenosis remain limited to small retrospective cohorts and case reports, which suggest that while immediate gradient reduction is consistently achieved in predominantly valvular lesions, restenosis or progressive pulmonary regurgitation can still occur over mid-term follow-up, warranting structured surveillance [3,4,7]. Accordingly, serial follow-up with echocardiography and cardiac magnetic resonance imaging is planned to quantify RV volumes, ventricular mechanics, and regurgitant fraction over time [3,4].

This report has important limitations. First, it describes a single case with outcomes limited by the short follow-up period, as the patient had not yet returned for his scheduled one-month post-discharge assessment; consequently, the mid- and long-term durability of gradient relief cannot be confirmed. Second, although baseline RV dysfunction was corroborated by both TAPSE (10 mm) and RV FAC (11%), post-procedural RV assessment relied primarily on TAPSE (which improved from 10 mm to 14 mm prior to discharge). Because TAPSE reflects longitudinal annular excursion and is load-dependent, acute afterload reduction following BPV can produce an immediate rise in TAPSE that may overestimate true recovery of global intrinsic RV contractility in a previously operated ventricle. Advanced multi-parametric evaluation - including serial RV FAC, tricuspid annular systolic velocity (S'), free-wall longitudinal strain, and cardiac MRI - will be necessary during follow-up to confirm sustained reverse remodelling [3,4].

Conclusions

Severe residual valvular pulmonary stenosis can emerge nearly two decades after an apparently successful repair of TOF, underscoring the importance of lifelong clinical and echocardiographic surveillance of even mild residual lesions. When multimodality imaging and haemodynamic assessment confirm that obstruction is predominantly valvular - while recognizing that mild dynamic infundibular components may coexist and regress over time - BPV offers a safe, minimally invasive alternative to repeat surgical intervention that can achieve substantial immediate haemodynamic unloading and symptomatic relief. Conservative balloon sizing is crucial to avoid main PA injury and limit iatrogenic pulmonary regurgitation in a previously operated right ventricle. Nevertheless, given the single-case nature of this report and the absence of post-discharge longitudinal follow-up to date, continued clinical and imaging surveillance - alongside larger, longer-term studies - is required to establish the mid- and long-term durability of this strategy.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Dipesh Soni, Ajay U. Mahajan, Arjun Chakra Gaur, Dhiraj Kumar

Acquisition, analysis, or interpretation of data: Dipesh Soni, Arjun Chakra Gaur, Dhiraj Kumar

Drafting of the manuscript: Dipesh Soni, Arjun Chakra Gaur, Dhiraj Kumar

Critical review of the manuscript for important intellectual content: Dipesh Soni, Ajay U. Mahajan, Arjun Chakra Gaur, Dhiraj Kumar

Supervision: Dhiraj Kumar

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **AI use statement:** The authors used Claude Opus 5.5 as follows: language polishing, structure or formatting, drafting summaries in Introduction, Case presentation, References. We verified the final manuscript and made necessary corrections in the references and re-checked facts mentioned in the introduction of the report. The authors confirm that they have reviewed and verified all AI-assisted content and take full responsibility for the work. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have

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