



Case Report

Reconstruction of the Pulmonary Trunk With A Homograft In Patients With Previous Tetralogy of Fallot Repair: A Case Report

Roman N. Komarov, Maxim I. Tkachev, Georgi A. Varlamov, Ilyas D. Gailaev, Muhanad Al Atrach

University Clinical Hospital N°1 of SECHENOV MEDICAL UNIVERSITY- Cardiovascular Surgery department

Abstract

Background: Tetralogy of Fallot (TOF) is a common congenital heart defect often requiring pulmonary valve replacement due to complications like pulmonary regurgitation. We present a case of a TOF patient with prior valve replacement admitted for prosthetic valve dysfunction.

Case presentation: At Sechenov University, the patient underwent successful reconstruction of the right ventricular outflow tract and pulmonary trunk using cryopreserved homografts. Postoperative recovery was uneventful, with discharge on day 14.

Conclusion: Pulmonary homografts provide favorable outcomes and reduced reinterventions, though degeneration remains a challenge. Future research should focus on factors affecting implant durability, such as age and size, to optimize long-term outcomes.

KEYWORDS

Tetralogy of Fallot;
Pulmonary homograft;
Bioprosthesis;
Pulmonary valve replacement; Right ventricular outflow tract reconstruction;
Cryopreserved homograft

Article History

Submitted: 17 Jan 2025
Revised: 15 Feb 2025
Accepted: 9 Mar 2025
Published: 1 Nov 2025

Introduction

Tetralogy of Fallot (TOF) represents the most common form of cyanogenic congenital heart disease, occurring in 4-5 per 10,000 newborns [1]. Modern advances in early diagnosis and surgical treatment have contributed to significant improvements in survival rates, which exceed 95% into adulthood. However, despite advances in corrective interventions, patients with corrected TOF often develop long-term right ventricular (RV) overload due to pulmonary regurgitation (PR), which can lead to progression of RV dysfunction, heart failure, sustained ventricular tachycardia, and sudden death. These complications lead to a significant increase in morbidity and mortality

starting in the third decade of life in patients with corrected TOF [2].

Due to the progression of these complications, pulmonary valve replacement is performed using various types of right ventricular outflow tract conduits, including bioprostheses and homografts. Positive effects such as reduction in right ventricular volumes, reduction in PR and improvement in NYHA functional class have been reported after correction using these conduits. However, one of the main problems encountered postoperatively is the need for repeated interventions due to bioprosthesis dysfunction.



The average life expectancy of right ventricular conduits (when these products are used in the correction of a malformation) is estimated to be 10-15 years [3]. Although numerous studies focus on the time to reoperation, the presence of homograft dysfunction is a more sensitive marker of its service life [4].

This article presents Sechenov University's experience in treating TOF patients with right ventricular outflow tract and pulmonary valve reconstruction using cryopreserved homografts. Our data align with other studies, showing that homograft implants reduce right ventricular volumes, decrease pulmonary regurgitation, and improve functional status. However, homograft durability remains a challenge, necessitating further research to optimize long-term outcomes and minimize reoperations. A single clinical case was highlighted for its representativeness, demonstrating key aspects of the study. This focused approach allows for a detailed analysis of the intervention technique, effectiveness, and avoids overgeneralization.

Description of the clinical case

Clinical picture

Patient A., 26 years old, was admitted with complaints of marked fatigue and dyspnoea during physical exertion. She had a history of radical TOF correction at age 2 (2000) with a successful outcome. In 2009, she underwent pulmonary valve replacement with a biological prosthesis. Throughout childhood, her condition remained stable, and regular examinations showed satisfactory prosthesis function and normal indices. However, in 2021, dysfunction of the pulmonary artery prosthesis was detected, with an echocardiography showing a pulmonic gradient of over 30 mm Hg, indicating significant narrowing of the prosthesis. There was also increased diastolic load on the right ventricle and marked pulmonary hypertension, along with slight dilation of the right atrium and mild enlargement of the right ventricle. Despite the absence of pronounced complaints, dynamic follow-up was recommended. In 2023, the patient became pregnant, and her condition worsened, with increased dyspnoea and decreased exercise tolerance. These symptoms gradually progressed

despite treatment, and physical activity intolerance was noted even during routine activities. On physical examination, the patient reported mild chest pain, which intensified with physical activity. Dyspnoea occurred not only during significant exertion but also during everyday activities.

Diagnostic tests

ECG, echocardiography, and MSCT-coronary angiography were performed to assess the heart condition. ECG showed sinus rhythm, heart rate of 60 beats/min, leftward electrical axis deviation, and right bundle branch block. Transthoracic echocardiography revealed an enlarged left atrium (64 ml, 4.1 cm) and impaired left ventricular diastolic function ($E/A = 1.9$, type 3 dysfunction). Left ventricular systolic function was preserved with EF of 65%. The right atrium was enlarged (75 ml), and right ventricular EF was 40%. Pulmonary hypertension was grade 3 (86 mmHg), with a bioclamp pressure gradient of 63 mmHg (peak) and 38 mmHg (mean). The pulmonary valve bioprosthesis showed grade 1-2 regurgitation and restricted cusp mobility. MSCT coronary angiography revealed normal pulmonary artery diameters (24 mm - trunk, 24 mm - right, 27 mm - left) and no coronary stenosis. Myocardial bridging of the anterior descending artery was noted. Based on the patient's symptoms and echocardiographic findings, the decision was made to replace the pulmonary artery with a cryopreserved homograft.

Surgical technique

A median sternotomy was performed, followed by cardiolysis and standard cardiopulmonary bypass with left ventricle vent through the superior right pulmonary vein. Anterograde non-selective cardioplegia with Del Nido solution arrested the heart. The pulmonary artery trunk was opened, revealing calcinosis and degradation of the bioprosthesis, which was explanted. The right ventricular outflow tract was mobilized, and an anastomosis between the tract and a 30 mm cryopreserved homograft was performed using continuous Prolene 5/0 sutures. The distal anastomosis between the homograft and pulmonary artery was also formed. Intraoperative transesophageal echocardiography

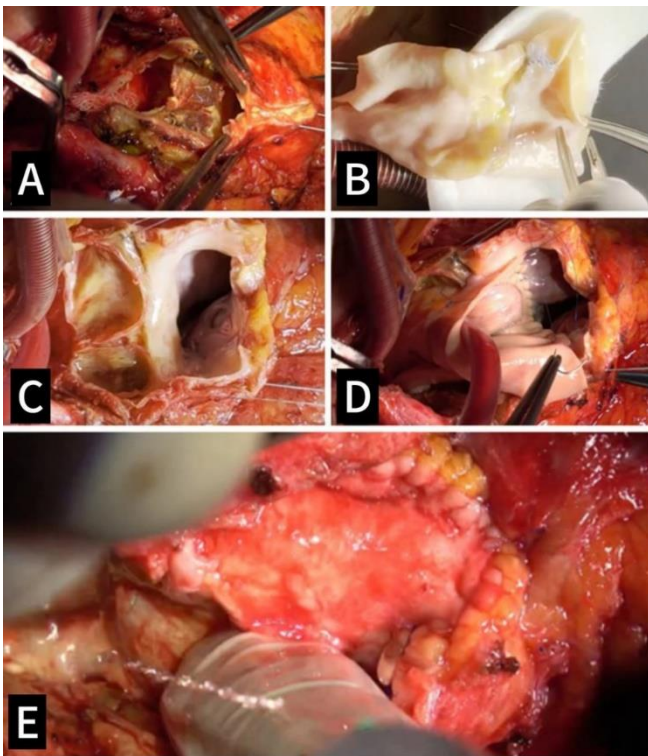


Figure 1: Pulmonary artery replacement with cryopreserved pulmonary homograft. A. Opened pulmonary artery showing dysfunctional prosthetic pulmonary valve B. Cryopreserved pulmonary homograft C. View right ventricular outflow tract after removal of the biological prosthesis D. Anastomosis of homograft to right ventricular outflow tract E. Completed pulmonary artery replacement

showed satisfactory left ventricular ejection fraction and properly functioning pulmonary artery prosthesis. The operation was completed in a standard fashion (Figure 1).

Results

Histological examination of valve samples showed pronounced sclerotic deformation of the pulmonary valve flaps without signs of inflammation. Postoperative echocardiography revealed Grade 1 central regurgitation of the pulmonary homograft, satisfactory cusp movement, and a peak pressure gradient of 10 mmHg. The cardiopulmonary bypass and myocardial ischemia durations were 85 and 56 minutes, respectively, with 500 ml blood loss. The patient was extubated 7 hours later and received dobutamine (3.5 mcg/kg/min) for inotropic support. She was transferred to the ward 16 hours post-surgery in stable condition. Antibacterial (cefuroxime), anticoagulant, and diuretic therapy were administered. Postoperative chest X-ray showed minor left pleural fluid accumulation,

managed with diuretics. No major complications occurred, and the patient was discharged on day 14, reporting increased physical tolerance and reduced dyspnea.

Discussion

Pulmonary artery replacement after TOF correction remains a critical issue in cardiac surgery, as conduit longevity is a significant limitation. While options like homografts, bovine jugular vein conduits (Contegra™), and porcine valves are available, all face risks of failure over time, potentially requiring further interventions. Homografts are advantageous due to their low antibody-forming activity, better body compatibility, and biological nature, making them less prone to calcification and degeneration compared to xenografts and other biological prostheses, although challenges with long-term durability remain.

Since the first use of homografts for right ventricular outflow tract reconstruction as by D. Ross and J. Somerville in 1966, their application in the treatment of congenital heart disease has become widespread [5]. In the Meijer et al. study, 89% of 26 patients did not need repeat valve replacement after 17 years, with an overall freedom from complications of 61.5% [6].

A study by Boethig et al. analyzed 188 homografts over 20 years. Ten-year freedom from re-intervention was 82% for homografts >19 mm in diameter, versus 45% for smaller ones. The Ross procedure showed 68% freedom from graft degeneration at 10 years, compared to 25% with other pulmonary artery reconstruction methods. Patients older than 10 years had 83% freedom from degeneration, while younger patients had 51% [7]. These data highlight the importance of conduit selection, particularly in pediatric practice, where implant longevity and function are key for long-term success.

Pulmonary homografts show higher freedom from re-intervention compared to bovine venous conduits and porcine valves. At 5 years, re-intervention freedom was 92%, dropping to 80% at 10 years. The incidence of infective endocarditis

was low (1%), confirming homografts' long-term safety and reliability [8].

Homografts provide superior haemodynamic performance and lower risks of reoperation or degeneration compared to bioprostheses, making them preferable in select cases [9]. Despite their positive outcomes, pulmonary homografts face durability concerns due to valve degeneration, highlighting the need for developing alternative valve conduits.

Homograft longevity is influenced by factors such as younger patient age, prolonged donor warm ischemic time, smaller graft size, and using aortic homografts in elderly patients. These contribute to accelerated degeneration, requiring careful evaluation when selecting implants [8].

In a study by M.M. Winter et al., key risk factors for pulmonary homograft dysfunction included postoperative pulmonary stenosis (pressure ≥ 20 mmHg), regurgitation (grade ≥ 1), and valve replacement before age 18. Patients with no risk factors had a 91% 10-year freedom from dysfunction, while those with two or more risk factors showed only 25% [10].

After tetralogy of Fallot repair, pulmonary valve dysfunction often necessitates replacement. Homografts offer high survival and low reoperation rates but remain prone to degeneration. Improving durability and performance requires further research. Optimising outcomes depends on factors like patient age, implant size, and early postoperative evaluation, which are essential for enhancing long-term results.

Conclusion

After tetralogy of Fallot correction, many patients require pulmonary valve replacement due to dysfunction. Homograft valves offer effective alternatives with high survival and low reoperation rates. However, homograft degeneration remains a challenge, necessitating research to enhance durability and performance. Surgical planning should consider factors like patient age and implant size to optimise

outcomes. Early postoperative evaluation is crucial for improving long-term results.

Funding: Self-funded

Conflict of interest: Authors declare no conflict of interest.

References

1. Van Der Bom T, Bouma BJ, Meijboom FJ, Zwinderman AH, Mulder BJM. [The prevalence of adult congenital heart disease, results from a systematic review and evidence based calculation](#). American Heart Journal. 2012; 164(4): 568-575.
2. Nollert G, Fischlein T, Bouterwek S, Böhmer C, Klinner W, Reichart B. [Long-Term Survival in Patients With Repair of Tetralogy of Fallot: 36-Year Follow-Up of 490 Survivors of the First Year After Surgical Repair](#). Journal of the American College of Cardiology. 1997;30(5): 1374-1383.
3. Geva T. [Indications for pulmonary valve replacement in repaired tetralogy of fallot: the quest continues](#). Circulation. 2013; 128(17): 1855-1857.
4. Oosterhof T, Van Straten A, Vliegen HW, et al. [Preoperative Thresholds for Pulmonary Valve Replacement in Patients With Corrected Tetralogy of Fallot Using Cardiovascular Magnetic Resonance](#). Circulation. 2007; 116(5): 545-551.
5. Ross DN, Somerville J. [Correction of pulmonary atresia with a homograft aortic valve](#). Lancet. 1966; 2(7479): 1446-1447.
6. Meijer FMM, Kies P, Jongbloed MRM, et al. [Excellent durability of homografts in pulmonary position analysed in a predefined adult group with tetralogy of Fallot](#). Interact Cardiovasc Thorac Surg. 2019; 28(2): 279-283.
7. Boethig D, Goerler H, Westhoff-Bleck M, et al. [Evaluation of 188 consecutive homografts implanted in pulmonary position after 20 years](#). European Journal of Cardio-Thoracic Surgery. 2007;32(1):133-142.
8. Marathe SP, Hussein N, Wallace FRO, et al. [Comparison of homografts and bovine jugular vein conduits in the pulmonary position in patients <20 years of age](#). J Thorac Cardiovasc Surg. 2022;164(3):752-762.e8.

9. Cocomello L, Meloni M, Rapetto F, et al. [Long-Term Comparison Between Pulmonary Homograft Versus Bioprosthesis for Pulmonary Valve Replacement in Tetralogy of Fallot](#). J Am Heart Assoc. 2019;8(24):e013654.
10. Bokma JP, Winter MM, Oosterhof T, et al. [Individualised prediction of pulmonary homograft durability in tetralogy of Fallot](#). Heart. 2015;101(21):1717-1723.