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Case Report

Patent Ductus Arteriosus Recanalization in Tricuspid Atresia with Restrictive Ventricular Septal Defect and Moderate Pulmonary Valve Stenosis: A Minimally Invasive Strategy in a Critical Case

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Abstract

Background: Tricuspid Atresia (TA) is a severe cyanotic Congenital Heart Disease (CHD) characterized by the absence of the tricuspid valve, resulting in Right Ventricular (RV) hypoplasia and dependence on interatrial and extracardiac shunts for pulmonary blood flow. In cases of restrictive Ventricular Septal Defect (VSD) with pulmonary outflow tract obstruction, the pulmonary circulation becomes duct-dependent. Closure of the Patent Ductus Arteriosus (PDA) may therefore lead to life-threatening hypoxemia. Prostaglandin E1 (PGE1) infusion is commonly used to maintain ductal patency; however, in emergency situations where PGE1 is unavailable or ineffective, catheter-based PDA recanalization can serve as a minimally invasive rescue strategy.

Case Illustration: A 4-month-old female with known TA presented with progressive cyanosis and respiratory distress. Echocardiography demonstrated TA, hypoplastic RV, large Atrial Septal Defect (ASD), restrictive VSD, and a ductal tunnel without detectable flow. After initial stabilization, the patient developed recurrent severe desaturation below 70% despite maximal medical and ventilatory support. Urgent cardiac catheterization revealed a functionally closed PDA. Recanalization was successfully performed using balloon dilatation followed by implantation of a 4.0 × 20 mm drug-eluting stent, restoring pulmonary blood flow and improving oxygen saturation from 55% to 91%.

Conclusions: PDA recanalization with stent implantation is a viable and life-saving minimally invasive option in critically ill patients with duct-dependent CHD when pharmacologic support fails and may serve as a bridge to staged surgical palliation.

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Introduction

Tricuspid Atresia (TA) is a severe form of cyanotic Congenital Heart Disease (CHD) characterized by the complete absence of the tricuspid valve, resulting in no direct communication between the right atrium and the Right Ventricle (RV). Consequently, the RV is hypoplastic, and systemic venous return must pass through an Atrial Septal Defect (ASD) to reach the left-sided circulation. Pulmonary blood flow, therefore, depends on alternative pathways, most commonly a Patent Ductus Arteriosus (PDA) or a Ventricular Septal Defect (VSD).[1,8] TA accounts for approximately 1–3% of all congenital heart defects, with an estimated incidence of 1 per 10,000 live births.[2]

In neonates and young infants with pulmonary outflow obstruction, the PDA plays a critical role in maintaining pulmonary circulation. Functional or anatomical closure of the ductus arteriosus may result in profound hypoxemia and hemodynamic collapse, particularly in patients with restrictive VSD or concomitant pulmonary valve stenosis.[1,6] In such conditions, pulmonary blood flow becomes functionally duct-dependent despite the presence of only moderate pulmonary valve obstruction.

Prostaglandin E1 (PGE1) infusion remains the standard initial therapy to maintain ductal patency in duct-dependent CHD. However, its use may be limited by availability, adverse effects, need for intensive monitoring, or reduced efficacy once functional ductal closure has occurred.[4-5] In these circumstances, catheter-based interventions have emerged as valuable rescue strategies. Transcatheter PDA recanalization and stent implantation offer a minimally invasive alternative capable of rapidly restoring pulmonary blood flow in critically ill patients. Although technically challenging and associated with potential complications such as ductal perforation, stent thrombosis, or restenosis, this approach may provide effective stabilization and serve as a bridge to definitive staged surgical palliation, including Bidirectional Cavopulmonary Shunt (BCPS) and Fontan procedure.[4-5,7]

Case Illustration

A 4-month-old female was admitted to the emergency department with progressive shortness of breath over the preceding two weeks, accompanied by productive cough, fever, and generalized edema. She was born at 38 weeks of gestation by cesarean section due to transverse lie, with a birth weight of 2405 g and Apgar scores of 8 and 9. Neonatal history was notable for hyperbilirubinemia requiring phototherapy. There was no maternal history of diabetes, hypertension, cardiac disease, or teratogenic drug exposure. The patient had been previously diagnosed with TA, large secundum ASD, and VSD.

On admission, the patient appeared lethargic and cyanotic. After initial stabilization, peripheral oxygen saturation was approximately 80–82% under mechanical ventilation (Fraction of Inspired Oxygen [FiO_2] 70%, Positive End-Expiratory Pressure (PEEP) 7 cmH_2O, Peak Inspiratory Pressure (PIP) 25 cmH_2O). Cardiac auscultation revealed a grade II/VI systolic murmur at the left parasternal border. Pulmonary examination demonstrated basal rhonchi. Chest radiography showed pneumonia without cardiomegaly. Laboratory evaluation revealed leukocytosis with mixed respiratory and metabolic acidosis.

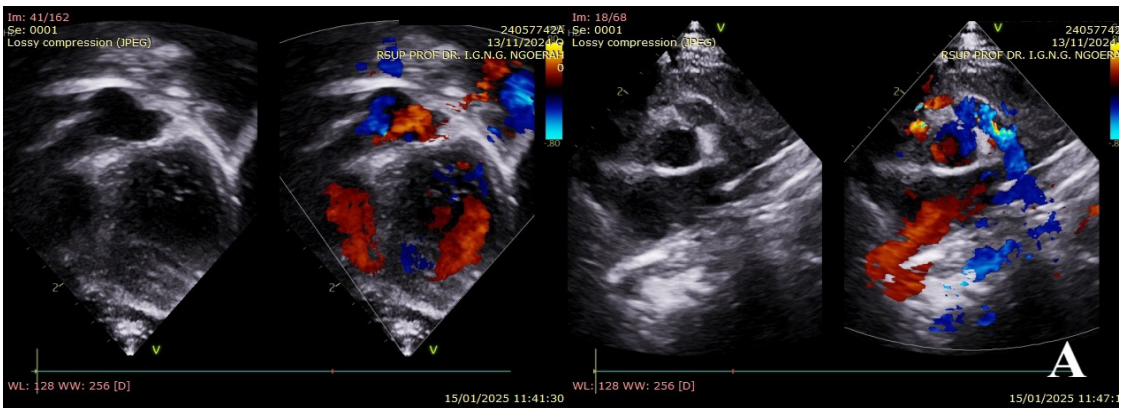

Figure A. Echocardiography examination showing tricuspid atresia with atrioventricular discordance, hypoplastic right ventricle, large ASD with right-to-left shunt, restrictive VSD, and absence of PDA.

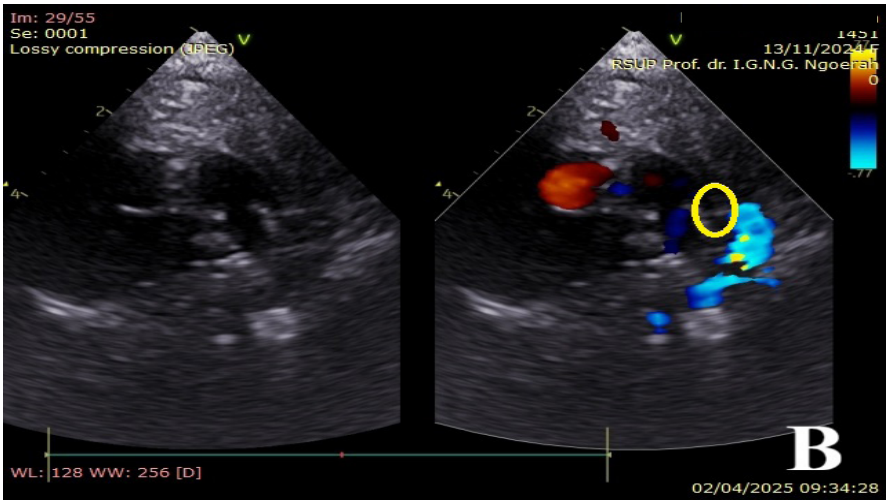

Figure B. Echocardiographic suprasternal view demonstrating a ductal tunnel sign. The yellow circle indicates the closed ductal ampulla without detectable flow prior to intervention.

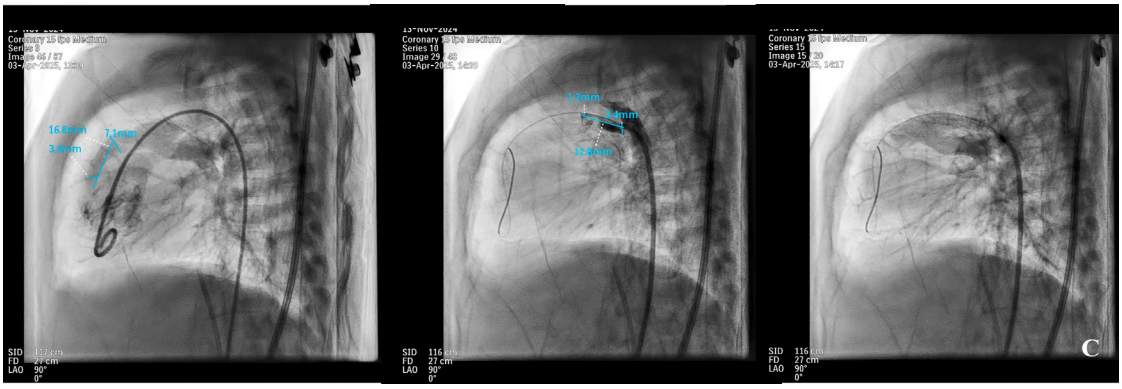

Figure C. PDA Recanalization.

Transthoracic echocardiography confirmed TA with hypoplastic RV, a large ASD with right-to-left shunt, a restrictive VSD with left-to-right shunt, moderate pulmonary valve stenosis, and a ductal tunnel sign without detectable flow, suggesting functional closure of the PDA. At this stage, after stabilization of the respiratory infection, elective diagnostic cardiac catheterization was initially planned. However, during hospitalization, the patient developed recurrent episodes of worsening cyanosis with progressive oxygen desaturation below 70% despite optimized ventilatory and medical support. Repeat echocardiographic evaluation continued to demonstrate absent ductal flow. Given the clinical deterioration and suspicion of duct-dependent pulmonary circulation, urgent cardiac catheterization was performed.

Angiography revealed a small ductal ampulla without contrast passage, confluent pulmonary arteries, and moderate pulmonary valve stenosis. Using femoral arterial access, a 0.014-inch guidewire was successfully advanced through the ductal remnant from the aortic side. Balloon dilatation was performed using a 2.5 × 20 mm balloon, followed by implantation of a 4.0 × 20 mm drug-eluting stent across the ductus arteriosus. Post-deployment angiography demonstrated restoration of antegrade flow from the aorta to the pulmonary artery. Peripheral oxygen saturation improved immediately from 55% to 91%. Post-procedurally, the patient was initiated on dual antiplatelet therapy consisting of aspirin at a dose of 5 mg/kg/day and clopidogrel at 0.2 mg/kg/day to reduce the risk of stent thrombosis. Follow-up echocardiography confirmed a patent PDA stent with good forward flow and stable ventricular function. The patient's clinical condition improved, and she was scheduled for reevaluation and further staged surgical palliation, including BCPS.

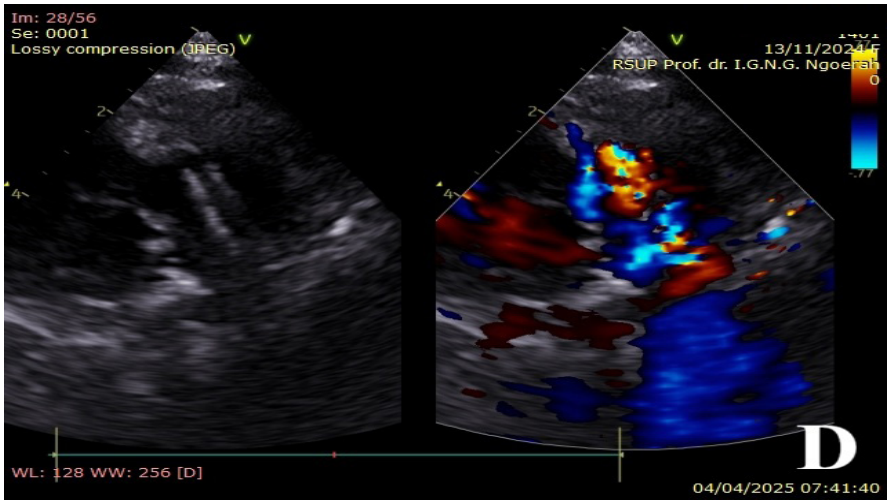

Figure D. Patent Stent at PDA, Flow (+).

Discussion

TA with pulmonary outflow obstruction represents a complex form of cyanotic CHD in which pulmonary blood flow is highly dependent on alternative shunt pathways. In this condition, the degree of systemic hypoxemia is determined not only by the severity of pulmonary valve stenosis but also by the size and functional capacity of intracardiac shunts. In the present case, although pulmonary valve stenosis was classified as moderate, the presence of a restrictive VSD markedly limited antegrade pulmonary blood flow. This combination rendered the patient functionally dependent on the PDA, explaining the profound hypoxemia observed when ductal flow became compromised.[1,6,8] In TA, pulmonary circulation may be supplied through a PDA, a nonrestrictive VSD, or a surgical shunt. When the VSD is restrictive, as in this patient, pulmonary blood flow becomes critically reduced regardless of the degree of pulmonary valve obstruction. Functional or anatomical closure of the ductus arteriosus in this setting can therefore precipitate rapid clinical deterioration, manifested by severe cyanosis and refractory hypoxemia.[1,6] This hemodynamic mechanism underscores the importance of promptly restoring ductal patency in critically ill patients with duct-dependent pulmonary circulation.

Echocardiography remains the cornerstone for diagnosis and initial assessment, allowing identification of atrioventricular connections, ventricular morphology, shunt pathways, and ductal patency. However, echocardiography may be limited in accurately defining ductal anatomy once functional closure has occurred. In such circumstances, cardiac catheterization provides a definitive anatomical and hemodynamic assessment while simultaneously offering a therapeutic option.[4-5] In the present case, catheterization confirmed the presence of a ductal remnant with no antegrade flow, supporting the decision for urgent intervention.

Maintenance of ductal patency using PGE1 infusion is the standard initial management for duct-dependent CHD. Nevertheless, prostaglandin therapy may be ineffective once the ductus has functionally closed or may be limited by adverse effects, prolonged intensive care requirements, and drug availability.[4-5] Surgical systemic-to-pulmonary shunts, such as the modified Blalock–Taussig (BT) shunt, provide reliable pulmonary blood flow but require thoracotomy and are associated with perioperative morbidity, particularly in hemodynamically unstable or infected patients.[6] In contrast, transcatheter PDA recanalization and stenting offer a minimally invasive alternative capable of rapidly restoring pulmonary blood flow without the need for open surgery.[4-5] In this critically ill infant, transcatheter PDA recanalization resulted in immediate and significant improvement in systemic oxygen saturation, confirming the duct-dependent nature of pulmonary circulation. This approach served as an effective bridge to staged surgical palliation, allowing stabilization before definitive procedures such as BCPS.[7] The increasing availability of catheterization laboratories in resource-limited settings further supports the role of ductal stenting as a feasible rescue strategy when surgical options are not immediately available.

A Drug-Eluting Stent (DES) was selected in this case to reduce the risk of early restenosis in a small-caliber ductus arteriosus and to ensure short-term patency until surgical palliation could be performed.[4-5]

Although concerns exist regarding the use of DES in ductal stenting due to potential challenges during subsequent surgical removal, the priority in this scenario was rapid and sustained restoration of pulmonary blood flow in a life-threatening condition. Careful postoperative management with dual antiplatelet therapy was therefore essential to minimize the risk of stent thrombosis.[4-5] Despite its advantages, ductal stenting is not without limitations. Technical challenges include difficulty in crossing a closed ductus, risk of ductal perforation, stent migration, and restenosis. Furthermore, close follow-up is required to monitor stent patency and pulmonary artery growth.[4-5] Nonetheless, in selected patients with duct-dependent pulmonary circulation, PDA recanalization offers a valuable and potentially life-saving option within a comprehensive, staged treatment strategy.[7]

Conclusion

TA with restrictive VSD and pulmonary outflow obstruction represents a complex CHD in which pulmonary blood flow becomes critically dependent on ductal patency. Functional closure of the PDA may result in rapid clinical deterioration and life-threatening hypoxemia. In such situations, transcatheter PDA recanalization with stent implantation offers a feasible, life-saving, minimally invasive strategy when pharmacologic ductal support is ineffective or unavailable. This approach provides effective hemodynamic stabilization and serves as an important bridge to staged surgical palliation, highlighting the value of timely intervention and multidisciplinary management in complex CHD.

List of Abbreviations

ASD	Atrial Septal Defect
BCPS	Bidirectional Cavopulmonary Shunt
BT	Blalock–Taussig
CHD	Congenital Heart Disease
FiO_2	Fraction of Inspired Oxygen
PDA	Patent Ductus Arteriosus
PEEP	Positive End-Expiratory Pressure
PGE1	Prostaglandin 1
PIP	Peak Inspiratory Pressure
RV	Right Ventricle / Right Ventricular
TA	Tricuspid Atresia
VSD	Ventricular Septal Defect

Ethical Clearance

Not applicable.

Publication Approval

All authors consent to the publication of this manuscript.

Authors' Contributions

G.D.M collected the clinical, laboratory, and imaging data. Contributed to the literature review, case analysis, discussion, and assisted with manuscript formatting and editing. All authors have read and approved the final manuscript.

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Conflict of Interest

The authors declare no conflict of interest.

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Authors acknowledge that artificial intelligence (AI) tools were only used to assist in language editing and did not generate or alter the scientific content, analyses, or conclusions presented in this manuscript.

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