Indonesian Journal of Cardiology
Indonesian J Cardiol 2026:47
pISSN; 2830-3105 / eISSN: 2964-7304
doi: 10.30701/ijc.1937

Case Reports

A Big Challenge in a Tiny Heart: Right Ventricular Outflow Tract (RVOT) Stenting in a Premature Infant with Pentalogy of Fallot

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Abstract

Background: Pentalogy of Fallot (POF) is a cyanotic congenital heart defect characterized by five anatomical abnormalities: Ventricular Septal Defect (VSD), pulmonary stenosis, Right Ventricular Hypertrophy (RVH), an overriding aorta, and an Atrial Septal Defect (ASD). The presence of the additional ASD alongside the classic features of Tetralogy of Fallot (TOF) can further exacerbate right-to-left shunting and worsen systemic desaturation. Management of POF in premature infants is particularly challenging due to low birth weight, underdeveloped pulmonary vasculature, and increased surgical risks. In high-risk neonates, traditional surgical options such as the modified Blalock–Taussig shunt may not be feasible. Right Ventricular Outflow Tract (RVOT) stenting has emerged as a minimally invasive palliative alternative to improve oxygenation and promote pulmonary artery growth, serving as a bridge to definitive surgical repair. This case highlights the technical considerations of RVOT stenting in high-risk neonates, emphasizing its role as a bridge to definitive surgical repair.

Case Illustration: An 11-day-old premature infant presented with progressive central cyanosis, desaturation episodes (<70%), and shortness of breath. Physical examination revealed respiratory distress, central cyanosis, and an ejection systolic murmur. Imaging showed a boot-shaped heart and oligemia, while echocardiography confirmed POF with severe RVOT obstruction, hypoplastic pulmonary arteries, and reduced Right Ventricular (RV) function. The patient was initially managed for a hypoxic spell but later developed periodic apnea and cardiac arrest, prompting additional diagnosis of apnea of prematurity and admission to the Intensive Care Unit (ICU) for further management. Despite treatment, worsening respiratory effort necessitated intubation. After clinical stabilization and extubation, persistent hypoxia led to RVOT stenting at 32 days of age. After initial difficulty, balloon pre-dilation allowed successful stent deployment, resulting in improved oxygen saturation.

Conclusions: This case demonstrates the feasibility and effectiveness of RVOT stenting as a palliative strategy in a premature infant with POF. The intervention led to immediate improvement in oxygenation and allowed time for clinical stabilization and potential pulmonary artery growth. This highlights that, in selected high-risk neonates where conventional surgical options may be unsuitable, catheter-based palliation can serve as a critical bridge to definitive surgical repair.

(Indonesian J Cardiol, 2026;47)

Keywords: Pentalogy of Fallot, Premature infant, RVOT stenting, Tetralogy of Fallot.

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Introduction

Pentalogy of Fallot (POF) is a rare congenital cyanotic heart defect, with a prevalence of approximately 0.006%, making it rarer than Tetralogy of Fallot (TOF), which has a prevalence of about 0.03%.[1] POF encompasses the four anatomical abnormalities characteristic of TOF—Ventricular Septal Defect (VSD), pulmonary stenosis, Right Ventricular Hypertrophy (RVH), and an overriding aorta—along with an Atrial Septal Defect (ASD).[2] This combination leads to right-to-left intracardiac shunting, resulting in diminished pulmonary blood flow and recurrent cyanotic episodes.[3]

In full-term infants, complete surgical repair is standard, but in premature or low birth weight neonates, this approach carries substantial risk due to tissue fragility and immature organ systems.[4] Thus, palliation is often required before definitive surgery. Although the modified Blalock–Taussig shunt has traditionally been used to increase pulmonary blood flow, its placement in very small or preterm infants is technically demanding and associated with higher morbidity.[5] Right Ventricular Outflow Tract (RVOT) stenting has emerged as a less invasive alternative for neonates unsuitable for early repair, effectively relieving RVOT obstruction, improving oxygenation, and facilitating pulmonary artery growth with acceptable procedural outcomes.[6]

Despite its advantages, RVOT stenting in premature infants remains technically challenging due to small vessel size, fragile myocardium, and increased risk of complications such as stent migration, restenosis, or obstruction of branch pulmonary arteries.[7] The indication for RVOT stenting in this premature patient is debatable, as the observed desaturation may be attributed to respiratory complications associated with prematurity rather than the TOF itself.

Given the limited literature on RVOT stenting in premature infants, this case report details the management of a premature infant with POF, emphasizing the use of RVOT stenting as a palliative bridge to definitive surgical repair.

Case Illustration

An 11-day-old premature infant presented to the emergency room with the chief complaint of cyanosis. The patient has a history of central cyanosis, which has progressively worsened and is accompanied by episodes of shortness of breath. The patient frequently experiences desaturation episodes, with oxygen saturation dropping below 70%, and the highest recorded saturation prior to the time of presentation to the emergency room was only approximately 80%.

The patient was born via cesarean section at 34 weeks and 6 days of gestation (premature birth) due to maternal indications of preeclampsia and ocular stroke. At birth, the baby did not cry immediately and presented with cyanosis. The color of the amniotic fluid at delivery was not known by the mother or the family. The patient received neonatal intensive care for seven days.

The patient has a history of interrupted breastfeeding. There are no reported symptoms of fever, cough, runny nose, or diarrhea. During pregnancy, the mother did not have a history of febrile illness but had controlled hypertension and diabetes mellitus. She regularly took medications for both conditions. The patient's father is a smoker, while the mother is a housewife. There are no pets at home, and there is no known exposure to specific chemicals.

On physical examination, the patient appeared alert but in respiratory distress. Vital signs revealed tachycardia with a Heart Rate (HR) of 175 beats per minute, a Respiratory Rate (RR) of 35 breaths per minute, and a body temperature of 37°C. Oxygen saturation was significantly decreased at 76% on room air. Cardiac examination revealed normal first and second heart sounds, with an ejection systolic murmur grade 3/6 best heard at the upper left sternal border. Examination of the extremities revealed cold acral areas and central cyanosis. Other physical examinations were unremarkable. The Electrocardiogram (ECG) revealed a sinus rhythm with a HR of 175 beats per minute and right axis deviation. It also showed evidence of RVH. (Figure 1)

Laboratory tests revealed significant leukocytosis with a white blood cell count of 23,180/µL and a Neutrophil-to-Lymphocyte ratio (NLR) of 3.50. Serum sodium was 127 mEq/L, and blood glucose was elevated at 179 mg/dL. Platelet count was markedly increased at 536,000/µL. Renal function showed a mildly reduced estimated Glomerular Filtration Rate (eGFR) of 53 mL/min/1.73m^2, with a creatinine level of 0.35 mg/dL and a Blood Urea Nitrogen (BUN) of 17.6 mg/dL.

Arterial blood gas analysis performed in the ER while on nasal cannula oxygen (1 L/min) showed a pH of 7.29, bicarbonate (HCO_3-) of 19.5 mmol/L, and a Base Excess (BE) of –6.1 mmol/L, suggesting metabolic acidosis. The partial pressure of oxygen (pO_2) was critically low at 19.1 mmHg, with an

arterial oxygen saturation (SaO_2) of 24.6%. Serum lactate was markedly elevated at 6.5 mmol/L. Ionized calcium and magnesium levels were also decreased, at 1.18 mmol/L and 0.61 mmol/L, respectively. The Chest X-Ray (CXR) showed evidence of oligemia and a boot-shaped heart. (Figure 2)

An echocardiogram performed in the ER showed a large secundum ASD measuring 5–8 mm with balanced left-to-right shunting, and a subaortic VSD with balanced shunting. Right Ventricular (RV) systolic function was reduced, with a Tricuspid Annular Plane Systolic Excursion (TAPSE) of 7 mm. Moderate to severe infundibular and valvular pulmonary stenosis was present, with a RV to pulmonary artery gradient of 55–60 mmHg. The right and left pulmonary arteries each measured 3 mm, with a Pulmonary Artery to Aortic Diameter Ratio (McGoon ratio) of 1.04. According to the Kirklin table, the expected half-size diameter for an infant of this weight is approximately 4 mm, indicating pulmonary artery hypoplasia. These findings are consistent with POF and severe pulmonary outflow tract obstruction with hypoplastic branch pulmonary arteries. (Figure 3)

The initial working diagnosis was a "tet spell" in

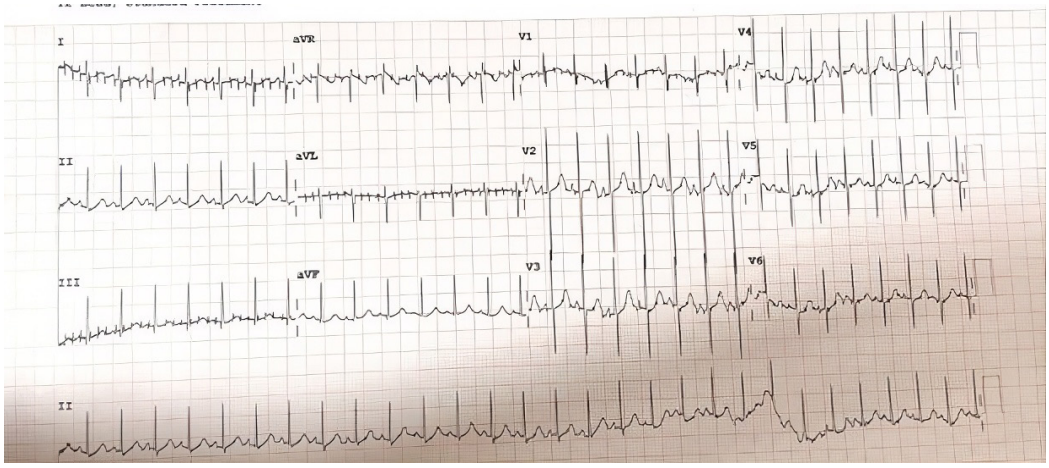

Figure 1. Electrocardiogram on admission demonstrating right axis deviation and right ventricular hypertrophy.

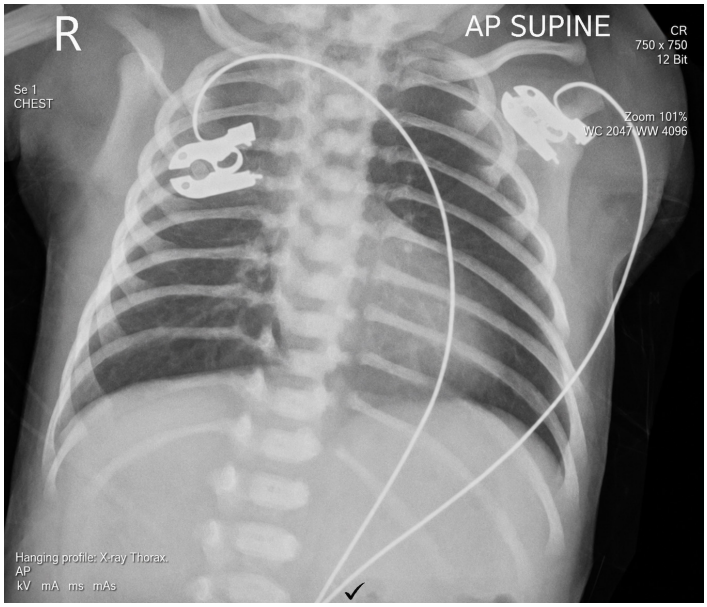

Figure 2. Chest X-ray in the emergency room showing a boot-shaped heart and pulmonary oligemia.

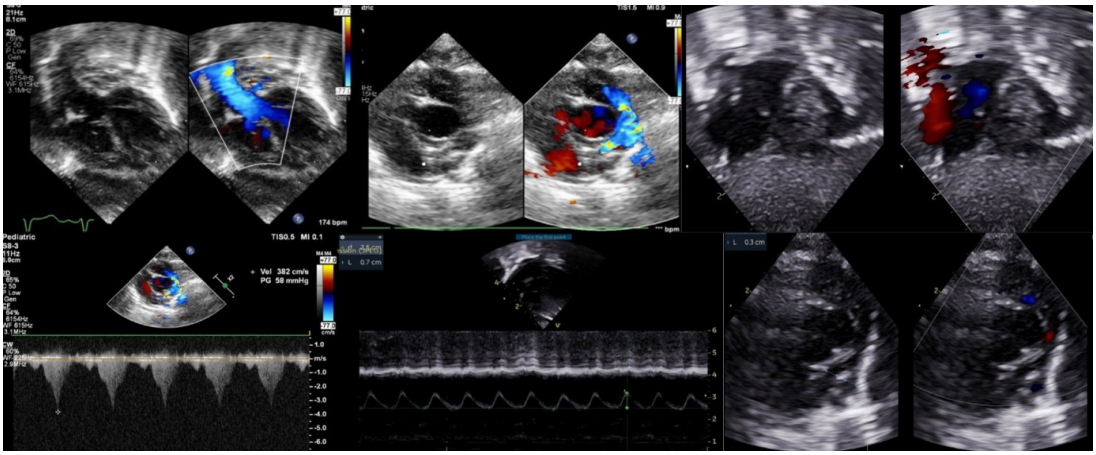

Figure 3. Pre-procedural echocardiography showing a ventricular septal defect with an overriding aorta (top left), infundibular pulmonary stenosis (top center), an atrial septal defect (top right), a pulmonary stenosis gradient of 58 mmHg (bottom left), reduced TAPSE (bottom center), and small pulmonary arteries (bottom right).

the setting of POF, for which the patient received standard management including the knee-chest position, fluid resuscitation, and propranolol administration. However, during several hours of observation in the emergency department, the patient developed recurrent episodes of periodic apnea that culminated in cardiac arrest. Cardiopulmonary resuscitation was initiated and achieved Return of Spontaneous Circulation (ROSC). Consequently, a diagnosis of apnea of prematurity was also made. The patient was then admitted to the Intensive Care Unit (ICU) for further evaluation and management.

The patient was subsequently managed with an aminophylline drip, caffeine citrate, and hydrocortisone; however, no clinical improvement was observed. Due to increased work of breathing, endotracheal intubation was performed. Head ultrasonography revealed minimal Intraventricular Hemorrhage (IVH) in the posterior body of the lateral ventricles and the posterior horns bilaterally. Following intubation, the patient showed clinical improvement and was successfully extubated after improvement in blood gas analysis. However, after careful observation, oxygen saturation decreased again, remaining below 75%, with a nadir of 48%. Worsening acidosis and increasing lactate levels were also noted, indicating inadequate perfusion. Therefore, RVOT stent placement was scheduled after 22 days of hospitalization, making the patient approximately 32 days old at the time.

Femoral vein access was established, and a 5/6F slender sheath was inserted. A baseline angiogram was performed using a 3.5/5F Judkins Right (JR) guiding catheter, revealing a tight RVOT obstruction and small confluent pulmonary arteries (Figure 4). A run-through coronary wire was advanced through the RVOT. Given that the RVOT was not excessively tight, a decision was made to proceed with direct stenting. Based on the patient's weight, the Kirklin table indicates a full-size PA diameter of 6 mm; therefore, we selected a 4.0 × 20 mm Promus Premier coronary stent. However, the stent could not traverse the RVOT. Consequently, lesion preparation was undertaken with several balloon dilatations using a 2.5 × 20 mm Sapphire II coronary balloon, inflated up to 14 atmospheric pressure across six inflations. Following adequate lesion preparation, the stent was positioned within the RVOT and deployed at 16 atmospheric pressure after confirming optimal placement. Post-procedure, peripheral oxygen saturation (SpO_2) improved markedly from 48% to 94%. Echocardiographic evaluation demonstrated that the RVOT stent was appropriately positioned, with a reduced gradient of 40 mmHg compared to the pre-procedural measurement (Figure 5). The patient was subsequently discharged seven days after RVOT stenting in stable condition. Considering the patient's history of intracranial hemorrhage, we discharged the patient on clopidogrel.

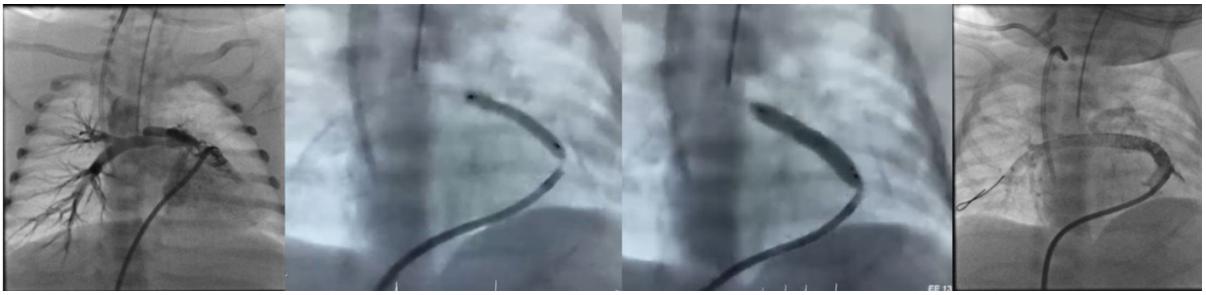

Figure 4. Angiographic procedure of the patient. From left to right: baseline angiogram showing severe RVOT obstruction with small confluent pulmonary arteries, balloon dilatation of the RVOT, stent deployment, and post-procedural result.

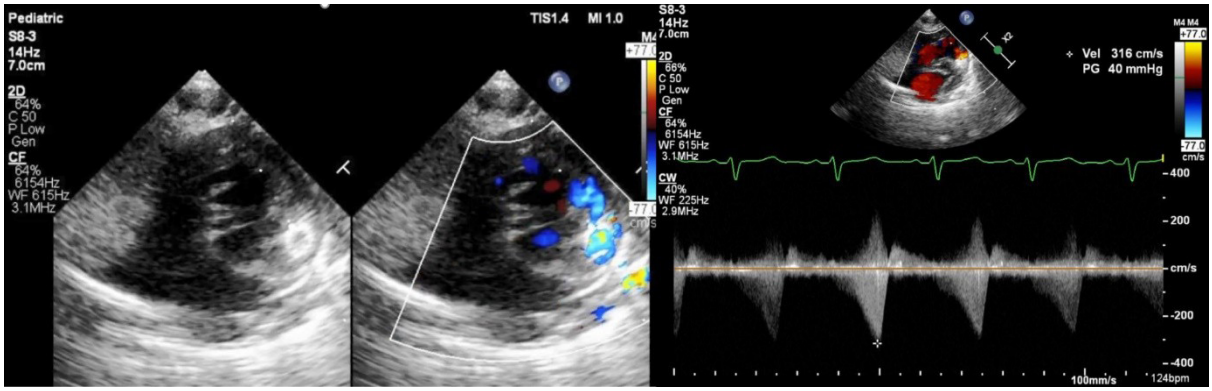

Figure 5. Post-procedural echocardiography showing the device well positioned in place with a reduced pulmonary stenosis gradient.

Discussion

POF is characterized by antero-cephalad deviation of the infundibular septum, resulting in malalignment and a nonrestrictive perimembranous VSD, an overriding aorta, RVH, and RVOT obstruction caused by infundibular pulmonary stenosis. Additionally, an ASD occurs.[8] These structural abnormalities underlie the characteristic hemodynamic disturbances of POF. Compared with classic TOF, the presence of an ASD intensifies the hemodynamic burden by promoting additional right-to-left intracardiac shunting, which further reduces pulmonary blood flow and exacerbates systemic hypoxemia.[9] In our case, the patient demonstrated all cardinal features of POF, including a large secundum ASD, consistent with the diagnostic criteria. Infants with POF typically present with cyanosis that worsens with crying or feeding ("tet spells"), failure to thrive, and hypercyanotic episodes due to dynamic RVOT obstruction.[9] Similarly, our patient presented to the emergency department with marked cyanosis and desaturation, consistent with the typical clinical presentation of POF.

The management of POF depends on the severity of symptoms, patient comorbidities, and anatomical characteristics. Treatment strategies generally aim to alleviate cyanosis, promote pulmonary artery growth, and minimize perioperative risk. For stable infants with adequate pulmonary artery size, primary complete repair between 3 and 6 months of age is preferred because it provides excellent long-term outcomes.[10] However, symptomatic neonates, particularly those who are premature or have low birth weight, frequently require initial palliation to augment pulmonary blood flow before definitive repair.[11] The decision between early complete repair and staged palliation is multifactorial, guided by patient-specific and anatomic considerations. Several criteria have been proposed to guide the selection of a two-stage approach, including active cyanosis, low body weight, and underdeveloped pulmonary arteries, with the latter often assessed using the McGoon ratio. A McGoon ratio of less than 1.2 has been associated with suboptimal outcomes after early complete repair and therefore serves as a threshold for recommending initial palliation.[12]

Historically, primary repair in neonates with hypoplastic pulmonary arteries has been associated with unfavorable outcomes. Early experiences revealed high mortality rates and significant postoperative complications, such as pericardial effusion, chylothorax, prolonged mechanical ventilation exceeding one week, and extended ICU stays.[13] These challenges are particularly relevant to our patient, who presented as a premature infant with profound cyanosis and hypoplastic pulmonary arteries, yielding a McGoon ratio of 1.04. Premature infants pose additional challenges because of their small size, immature organ systems, and frequent comorbidities such as respiratory distress syndrome and IVH.14 In our patient, prematurity at 34 weeks of gestation, accompanied by respiratory distress and IVH, significantly increased the surgical risk. Given these high-risk features, initial palliation was deemed the most appropriate strategy.

The next key consideration involves selecting the most suitable palliative technique. Traditional surgical options include the modified Blalock–Taussig shunt, which has been widely utilized to enhance pulmonary blood flow. In recent years, however, catheter-based techniques such as RVOT stenting have emerged as effective and less invasive alternatives. The decision among early primary repair, initial palliation, and Blalock–Taussig shunt versus RVOT stenting depends on several factors, including pulmonary artery anatomy, presence of comorbidities, and institutional expertise.[15]

RVOT stenting has demonstrated superior and more symmetrical pulmonary artery growth compared with the Blalock–Taussig shunt. In comparative analyses, stented patients experienced a greater increase in the Nakata index (from 104.2 to 208.6 mm^2/m^2) than those who received a Blalock–Taussig shunt (107.3 to 169.4 mm^2/m^2), indicating enhanced pulmonary arterial development. Furthermore, RVOT stenting avoids several complications associated with surgical shunts, including pulmonary overcirculation, distortion of the pulmonary artery branches, and shunt thrombosis.[16]

In terms of short-term outcomes, RVOT stenting offers comparable or even superior results. Compared to Blalock–Taussig shunts, it is associated with a similar 30-day mortality rate (1.7% vs 4.9%), but with significantly shorter ICU stays (22% vs 100%), reduced overall hospital stay (median 7 vs 14 days), and a shorter interval to definitive repair (median 232 vs 428 days)—benefits that likely result from more rapid pulmonary artery growth.[17] Further supporting evidence comes from Sandoval et al., who investigated 180 infants with TOF across varying risk profiles. In their cohort, 42 high-risk infants underwent RVOT stenting due to small pulmonary arteries (median Nakata index 79 mm^2/m^2), low body weight, and multiple comorbidities. Stenting successfully alleviated cyanosis, raising median oxygen saturation from 75% to 94%, and allowed for somatic and pulmonary arterial growth (post-stenting Nakata index 147 mm^2/m^2). This facilitated complete repair at a median age of 6 months and weight of 6.3 kg, comparable to lower-risk infants undergoing elective repair. Importantly, 95% of stents were successfully retrieved during surgery, and postoperative outcomes—including ICU stay and Extracorporeal Membrane Oxygenation (ECMO) requirement—were similar or even more favorable than those following early primary repair.[18]

Collectively, these studies underscore the safety and efficacy of RVOT stenting as a bridge to definitive repair in infants with poor anatomy and significant comorbidities. In light of this evidence, and considering our patient's prematurity, low weight, and pulmonary artery hypoplasia, RVOT stenting was selected as the preferred palliative strategy to safely delay complete surgical repair.

From a procedural standpoint, the technique of RVOT stenting requires careful planning to ensure optimal hemodynamic benefit and to preserve the integrity of cardiac anatomy for future repair. When necessary, lesion preparation with balloon dilatation can facilitate stent passage and expansion.[17] In our case, the stent could not initially cross the RVOT via a direct approach; therefore, sequential balloon dilations were performed prior to successful stent deployment.

In infants weighing less than 3 kg, coronary stents are preferred due to their smaller profile and ability to be delivered through low-profile sheaths. Accordingly, a coronary stent was selected for our patient. Venous access is typically achieved via the right femoral vein, using 4–6 F sheaths. In our procedure, a slender 5/6 F sheath was employed through the right femoral vein. A 3.5/5 F JR guiding catheter and a coronary wire were used to enhance support and deliverability.[17] The use of a long sheath or guiding catheter provides greater procedural stability and allows repeated contrast injections to accurately assess stent position and pulmonary artery filling—an approach we also adopted.

To ensure compatibility with future surgical repair, stent placement should aim to maintain anatomic relationships and avoid distortion that

may complicate later reconstruction.[17] In this case, the stent was intentionally positioned to maintain anatomic integrity and facilitate future surgical retrieval, as the patient is scheduled for definitive repair in the long term. For small infants (~3 kg), RVOT palliation commonly employs coronary stents measuring approximately 3.5–4.0 mm, as these devices can be delivered through low-profile sheaths.[19] Several contemporary RVOT stent series report the frequent use of 4-mm stents in infants weighing approximately 3 kg.[15,20] At our center, stent selection also incorporates the principle of using approximately 60–70% of the Kirklin full size as a practical guide.

In our case, a non–valve-sparing RVOT stenting approach was selected. For newborns with markedly hypoplastic pulmonary arteries, recent evidence supports the use of a transpulmonary (across-the-valve) stenting strategy. In high-risk infants with severely small pulmonary arteries, this approach has been shown to markedly improve systemic oxygenation and promote substantial bilateral pulmonary artery growth, enabling later surgical repair with successful stent removal and transvalvular widening.[20] Likewise, larger cohort data indicate that transvalvular stenting is frequently required in patients with hypoplastic pulmonary arteries and is consistently associated with meaningful arterial enlargement, whereas valve preservation is feasible in only a minority of such cases.[21]

Post-RVOT stenting in infants with congenital heart disease is typically followed by antiplatelet therapy—most commonly low-dose aspirin, with or without clopidogrel—to reduce the risk of stent thrombosis.[15] In the setting of recent intracranial hemorrhage, however, decisions regarding the initiation or continuation of antiplatelet therapy require a careful balance between thrombotic and bleeding risks and should be individualized.[22] In our patient, we decided to continue clopidogrel as a pragmatic compromise between thromboprophylaxis and hemorrhagic risk, even in the presence of intracranial hemorrhage, given that the bleed was small, although evidence in this specific context remains limited and warrants further investigation.

After RVOT stenting, one of the primary considerations is the resulting hemodynamic changes. The hemodynamic effects of RVOT stenting in POF differ markedly from those in classic TOF because of dual-level shunting. By relieving RVOT obstruction, stenting reduces right-to-left shunting across the VSD and increases pulmonary blood flow, thereby improving systemic oxygenation.[23] However, in the presence of a large ASD, atrial-level right-to-left shunting may persist or even intensify. After stenting, RV and right-atrial pressures fall, while increased pulmonary venous return raises left-atrial pressure, creating a gradient that favors continued atrial-level shunting.[20] As a result, oxygenation may improve only modestly, and the sudden rise in pulmonary flow can occasionally produce left-ventricular volume overload or pulmonary edema. Pulmonary arterial growth may also be asymmetric due to preferential flow, and ASD closure is often required for optimal physiology.[1]

In contrast, a modified Blalock–Taussig shunt increases pulmonary blood flow and systemic saturation in POF but also raises left-heart preload. With an open ASD, oxygenated blood may shunt left-to-right at the atrial level, increasing RV volume, reducing LV filling through septal shift, and lowering systemic cardiac output.[24] Although oxygenation may initially improve, atrial-level recirculation decreases systemic delivery efficiency, and excessive shunt or atrial shunting can precipitate pulmonary edema or heart failure. Careful hemodynamic monitoring is therefore essential to balance oxygenation with pulmonary vascular load.

ASD closure is typically delayed until definitive repair and guided by cardiac catheterization and balloon occlusion testing to ensure that right atrial pressure remains stable after closure. ASD closure is considered once pulmonary vascular resistance decreases, the RV is adequately conditioned, and systemic oxygenation is stable. If balloon occlusion testing reveals a significant rise in right atrial pressure, closure is postponed. Therefore, the decision is individualized, taking into account each patient's hemodynamic profile and postoperative adaptability.[25-26]

Regarding the timing of definitive repair, the 2022 American Association for Thoracic Surgery (AATS) Expert Consensus recommends an ideal window between 3 and 6 months of age, while other contemporary reviews note that repair may be safely performed up to approximately 11 months in selected infants.[27-28] Importantly, the decision is not determined by age alone; adequate pulmonary artery growth and sufficient body weight and physiological maturity are essential prerequisites before proceeding to complete repair, ensuring that the cardiopulmonary system can accommodate the hemodynamic changes following intracardiac reconstruction.[29] In our case, the patient has not yet undergone definitive repair. Complete correction

is planned once the child reaches an appropriate age and demonstrates adequate growth of the pulmonary arteries.

Conclusion

In conclusion, this case illustrates that RVOT stenting is a feasible and effective palliative strategy for premature infants with POF, particularly in the context of severe RVOT obstruction, hypoplastic pulmonary arteries, and high surgical risk. The procedure provided immediate improvement in systemic oxygenation, stabilized the patient clinically, and allowed for potential somatic and pulmonary arterial growth, serving as a critical bridge to definitive surgical repair. Careful procedural planning, including balloon pre-dilation and use of appropriately sized coronary stents, is essential to ensure safe deployment while preserving anatomical integrity for future surgery. Moreover, the presence of a large ASD requires careful hemodynamic monitoring, as atrial-level shunting may persist or complicate oxygenation. Overall, RVOT stenting represents a valuable, minimally invasive alternative to traditional surgical palliation in selected high-risk neonates, enabling staged management and optimizing outcomes prior to complete repair.

List of Abbreviations

ASD	Atrial Septal Defect
BE	Base Excess
BUN	Blood Urea Nitrogen
CXR	Chest X-Ray
ECMO	Extracorporeal Membrane Oxygenation
eGFR	Estimated Glomerular Filtration Rate
ECG	Electrocardiography/ Electrocardiogram
HCO_3-	Bicarbonate
HR	Heart Rate
ICU	Intensive Care Unit
IVH	Intraventricular Hemorrhage
JR catheter	Judkins Right Catheter
McGoon ratio	Pulmonary Artery to Aortic Diameter Ratio
NLR	Neutrophil-to-Lymphocyte Ratio
pO_2	Partial Pressure of Oxygen
POF	Pentalogy of Fallot
ROSC	Return of Spontaneous Circulation
RR	Respiratory Rate
RV	Right Ventricle
RVH	Right Ventricular Hypertrophy
RVOT	Right Ventricular Outflow Tract
SaO_2	Arterial Oxygen Saturation
SpO^2	Peripheral Oxygen Saturation
TAPSE	Tricuspid Annular Plane Systolic Excursion
TOF	Tetralogy of Fallot
VSD	Ventricular Septal Defect

Ethical Clearance

Not applicable.

Publication Approval

All authors consent to the publication of this manuscript.

Author Contributions

All authors contributed to the preparation and writing of this case report. Material preparation, data collection, and data analysis were performed by AR, RP, and AAS. The first draft of the manuscript was written by AR. RP and AAS provided critical supervision, intellectual input, and constructive recommendations to improve the manuscript throughout the writing process. All authors reviewed, revised, and approved the final version of the manuscript.

Acknowledgments

The authors would like to express their gratitude towards the Department of Cardiology and Vascular Medicine, National Cardiovascular Center Harapan Kita, Universitas Indonesia for their unwavering support in completing this work.

Conflict of Interest

None.

Availability of Data and Materials

Not applicable.

Funding

This paper received no specific grant from any funding agency, commercial or not-for-profit sectors.

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Not applicable.

Generative AI and AI-Assisted Technologies in the Writing Process

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

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