PERINATAL JOURNAL

Original Article

Perinatal Journal 2025; 33(2):221-229

https://doi.org/10.57239/prn.25.03320025

Early recognition and stabilization in neonates with critical congenital heart disease: A case series highlighting diagnostic vigilance and timely referral

Ririn Wahyuningtyas[1], Martono Tri Utomo[2]*, I Ketut Alit Utamayasa[2], Risa Etika[2], Dina Angelika[2], Kartika Darma Handayani[2], Indah Musdalifah Chaeruddin[2]

[1]Resident, Department of Child Health, Faculty of Medicine, Universitas Airlangg, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia
[2]Lecturer, Department of Child Health, Faculty of Medicine, Universitas Airlangg, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia

Abstract

Critical Congenital Heart Disease (CCHD) is a life-threatening condition in neonates that often requires early intervention to prevent severe hypoxemia, cardiogenic shock, and death. Timely diagnosis remains a challenge, particularly in ductal-dependent lesions, where the patency of the ductus arteriosus is vital for systemic or pulmonary circulation. Globally, CCHD occurs in approximately 1.8 to 2.5 per 1000 live births, with higher rates reported in resource-limited settings due to delayed diagnosis. We present a case series of three full-term neonates who developed persistent central cyanosis within the first hours of life. Pulse oximetry revealed significant pre- and post-ductal SpO_2 differences (range: 70–80% preductal, 50–60% postductal), raising suspicion of ductal-dependent congenital heart lesions. Bedside echocardiography confirmed the diagnosis in all cases: (1) Ebstein anomaly with pulmonary atresia, (2) pulmonary atresia with intact ventricular septum (PA-IVS), and (3) critical pulmonary stenosis with severe tricuspid regurgitation. Each patient received intravenous prostaglandin E_1 (PGE_1) infusion at 0.05 µg/kg/min, resulting in rapid improvement in oxygenation and hemodynamic stability. Early recognition of CCHD using pulse oximetry screening followed by confirmatory echocardiography is crucial for initiating timely PGE_1 therapy. In all cases, echocardiography enabled detailed assessment of anatomical and hemodynamic abnormalities and guided initial management. This case series highlights the importance of prompt recognition and diagnosis of ductal-dependent CCHD. The combined use of pulse oximetry and echocardiography, followed by timely administration of PGE_1, plays a pivotal role in stabilizing critically ill neonates prior to surgical or catheter-based intervention.

Keywords: Critical congenital heart disease, Ductal-dependent lesion, Prostaglandin E1, Echocardiography, Neonatal cyanosis

Introduction

Critical Congenital Heart Disease (CCHD) refers to structural cardiac anomalies that require intervention within the first days or weeks of life to prevent severe hypoxia, shock, or death (Gomella et al., 2020; Mahle et al., 2009). Globally, CCHD occurs in approximately 1.8 to 2.5 per 1000 live births, with higher rates reported in resource-limited settings due to delayed diagnosis (Hoffman & Kaplan, 2002; Myung et al., 2015). Early detection is essential to initiate timely treatment and reduce morbidity and mortality (Mahle et al., 2009). Delayed diagnosis of Congenital Heart Disease (CHD) remains highly prevalent, especially in cyanotic CHD cases, with overall delayed diagnosis observed in 60.8% of children and up to 86.2% in cyanotic CHD. The most frequent cause of delay was misdiagnosis by healthcare providers, followed by referral system deficiencies, financial barriers, and lack of parental knowledge. The study highlights the urgent need to improve early recognition skills among healthcare workers, strengthen referral systems, and enhance public awareness to reduce delays that contribute to increased morbidity and mortality among children with CHD (Murni et al,, 2021). One of the most effective, non-invasive, and low-cost screening tools is pulse oximetry, particularly using pre- and post-ductal SpO_2 measurements to detect right-to-left shunting or reduced systemic oxygenation (Myung et al., 2015). Performed 24–48 hours after birth, pulse oximetry screening is a non-invasive measurement of the proportion of hemoglobin in blood that is saturated with oxygen. The presence of low blood oxygen saturation (hypoxemia), or a difference between pre-ductal and post-ductal (proximal and distal to the aortic opening of the ductus arteriosus, respectively) saturation, frequently precedes other signs or symptoms in infants with unrecognized CCHD (Kemper & William, 2015). A difference in saturation >3% or SpO_2 <90–95% is considered abnormal and should prompt further evaluation (Mahle et al., 2009). Bedside echocardiography plays a crucial role in confirming the diagnosis, identifying duct-dependent lesions, and evaluating the presence and direction of ductal or intracardiac shunting (Myung et al., 2015; Mansoor et al., 2025). Rapid bedside imaging is especially important when clinical signs are subtle but SpO_2 screening is

Correspondence: Martono Tri Utomo, Lecturer, Department of Child Health, Faculty of Medicine, Universitas Airlangg, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia, **e-mail:** mrmartono73@gmail.com, **Received:** July 16, 2025 **Accepted:** September 5, 2025

abnormal. When a duct-dependent circulation is identified—such as in pulmonary atresia, transposition of the great arteries, or severe coarctation— prostaglandin E1 (PGE1) must be promptly administered to maintain or reopen the ductus arteriosus and ensure adequate pulmonary or systemic perfusion (Hoffman & Kaplan, 2002). Early initiation of PGE1 improves hemodynamic stability and allows time for definitive intervention, such as cardiac catheterization or surgery (Mahle et al., 2009). This case series highlights the importance of a structured approach using SpO_2 screening, echocardiographic confirmation, and pharmacologic ductal support in neonates with suspected CCHD.

Case report

Case report 1 – Ebstein anomaly with cyanosis in a term neonate

A female neonate was born via cesarean section at 38 weeks of gestation with a birth weight of 2900 grams. The infant was referred to our unit on May 25, 2025, at 9 days of age with complaints of persistent central cyanosis and feeding difficulty. On physical examination, oxygen saturation was decreased, with SpO_2 measured at 80% pre-ductal and 65% post-ductal, indicating significant right-to-left shunting and possible duct-dependent circulation.

The baby appeared mildly cyanotic, with no murmur, good cry, and mild respiratory distress. Chest X-ray showed cardiomegaly with right atrial and right ventricular enlargement with decreased pulmonal vascularity.

Echocardiography revealed Ebstein anomaly, pulmonary atresia with small PDA. Laboratory findings showed stable hemoglobin and hematocrit, with mild leukocytosis and thrombocytosis.

The working diagnosis was congenital heart disease with features suggestive of Ebstein anomaly, based on the enlarged right atrium and right ventricle seen on radiographic imaging and Echocardiography. The infant was stabilized with supportive oxygen therapy, monitoring, and feeding support. Prostaglandin E1 (PGE1) was considered to maintain ductal patency, and the baby was referred to a pediatric cardiology center for definitive echocardiographic evaluation and potential intervention.

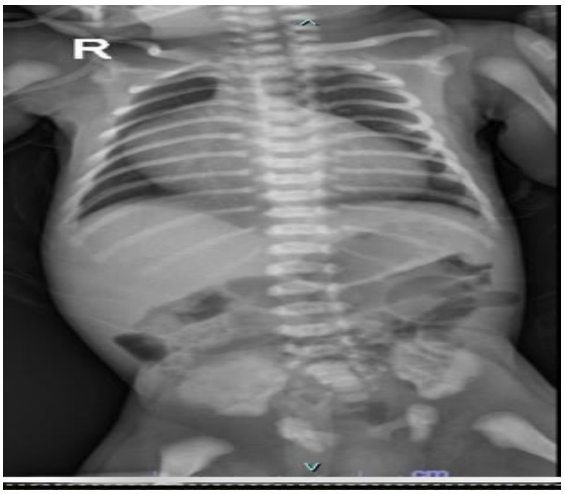

Figure 1. Postnatal anterior-posterior x-ray of chest showed cardiomegaly with right atrial and right ventricular enlargement, consistent with suspected ebstein anomaly

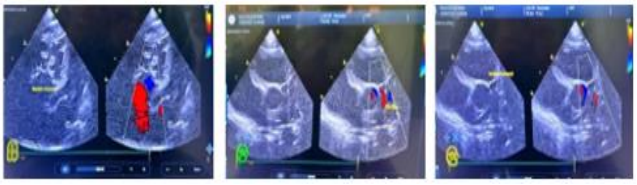

Figure 2. Fetal echocardiography

1. 4 chamber view showed atrialisasi RV (ebsstein anomaly)
2. PSAX view showed atresia pulmonal
3. PSAX view showed PDA flow

Case Report 2 – A Term Neonate with Duct – Dependent Critical CHD – Tricuspid Atresia, Pulmonary Atresia, Double Outlet Right Ventricle (DORV) and Vsd Invlet and Small PDA

A female neonate was born at 38–39 weeks of gestation via cesarean section due to cephalopelvic disproportion, with a birth weight of 2850 grams. She initially appeared clinically stable but developed central cyanosis and poor feeding beginning on the third day of life. The infant was referred to our tertiary care center on May 5, 2025 (day 5 of life), from a secondary hospital with persistent hypoxemia. On admission, the neonate appeared lethargic, with cyanotic skin and weak feeding effort. Pulse oximetry revealed a pre-ductal SpO_2 of 80% and a post-ductal SpO_2 of 60%, raising clinical suspicion of duct-dependent congenital heart disease. Initial investigations showed hemoglobin of 16.2 g/dL, hematocrit 52.2%, leukocyte count 6610/mm^3, and platelet count 621,000/mm^3. Arterial blood gas analysis revealed

a pH of 7.38, PCO_2 of 35.1 mmHg, PO_2 of 43.2 mmHg, bicarbonate (HCO_3^-) of 20.8 mmol/L, base excess (BE) of -3.4, and SpO_2 of 74%, indicating moderate hypoxemia. A chest radiograph demonstrated cardiomegaly with prominence of the right heart border and no pulmonary infiltrates. Bedside echocardiography revealed situs solitus, atrioventricular concordance, but ventriculoarterial discordance. The cardiac anatomy included tricuspid atresia, pulmonary atresia, double outlet right ventricle (DORV), large inlet-type ventricular septal defect, primum ASD. The aorta originated from the right ventricle with a left-sided aortic arch. A vertical turtous patent ductus arteriosus (PDA) was observed, with ampulla measuring 0.4 cm and waist 0.2 cm, demonstrating forward flow. The left and right pulmonary arteries were present with mild hypoplasia. Left ventricular ejection fraction was measured at 75%. The infant was stabilized with oxygen therapy via nasal cannula (0.5 L/min), prostaglandin E1 infusion at 10 ng/kg/min to maintain ductal patency, and dobutamine at 5 mcg/kg/min to support cardiac output. After clinical stabilization, the neonate was referred to a pediatric cardiac center for further evaluation and intervention, including planned PDA stenting.

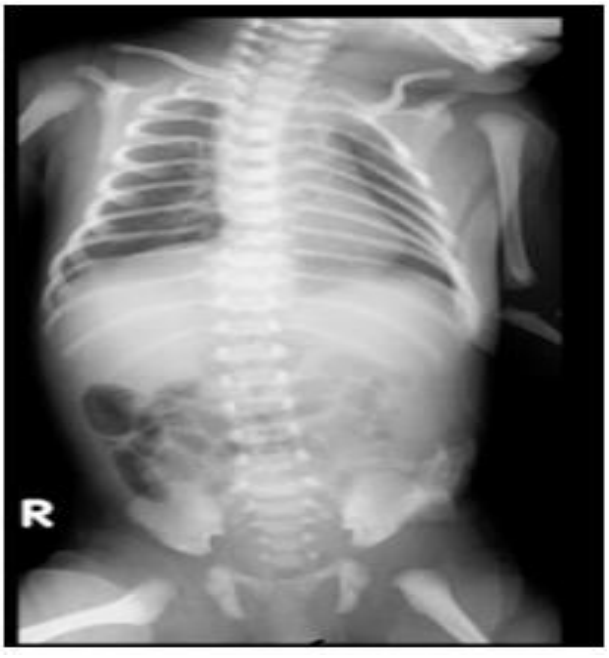

Figure 3. Postnatal anterior-posterior x-ray of chest revealed cardiomegaly, with no lung parenchymal abnormalities, and abdominal findings were within normal limits

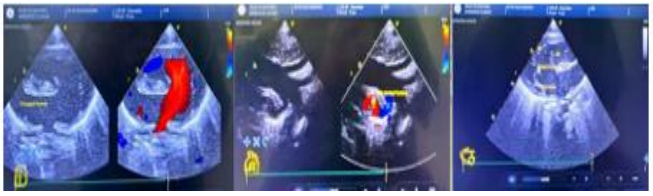

Figure 4. Fetal echocardiography showed

1. 4 chamber view showed Tricuspid atresia with large VSD and ASD
2. Suprasternal view showed PDA vertikal turtous
3. PLAX view showed aorta out flow from RV with Pulmonal atresia

Case Report 3 – A Term neonate with critical pulmonary stenosis and severe tricuspid regurgutation

A term female neonate was born via spontaneous delivery at 38 weeks of gestation with a birth weight of 2900 grams. On day 5 of life, she was referred to our NICU due to persistent central cyanosis and signs of respiratory distress. On examination, the pre-ductal oxygen saturation was 70% and the post-ductal was 50%, indicating significant right-to-left shunting and possible duct-dependent congenital heart disease. Initial stabilization included administration of oxygen via nasal cannula (1 L/min), intravenous fluids, dobutamine at 5 mcg/kg/min, and prostaglandin E1 infusion at 15 ng/kg/min to maintain ductal patency. Chest X-ray revealed cardiomegaly, with no lung parenchymal abnormalities, and abdominal findings were within normal limits. Bedside echocardiography was promptly performed and showed situs solitus with atrioventricular and ventriculoarterial concordance. Right heart structures were dilated with a severely regurgitant tricuspid valve. There was critical pulmonary valve stenosis with a peak gradient of 12 mmHg. A tiny patent ductus arteriosus (PDA) with limited flow was visualized. No ventricular septal defect was noted, and biventricular systolic function was preserved. Laboratory tests showed elevated hematocrit and hemoglobin, consistent with chronic hypoxemia, and normal renal and liver function. The neonate was diagnosed with critical pulmonary stenosis, severe tricuspid regurgitation, and a small PDA. After initial stabilization, she was referred to the pediatric cardiology center for catheter-based intervention.

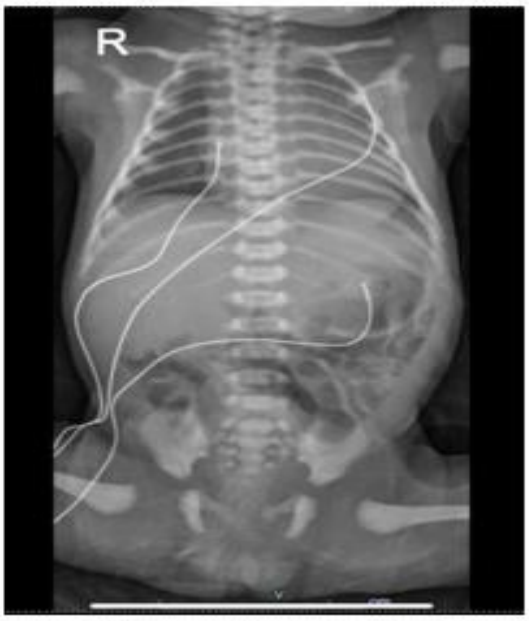

Figure 3. Postnatal anterior-posterior x-ray of chest revealed cardiomegaly, with no lung parenchymal abnormalities, and abdominal findings were within normal limits.

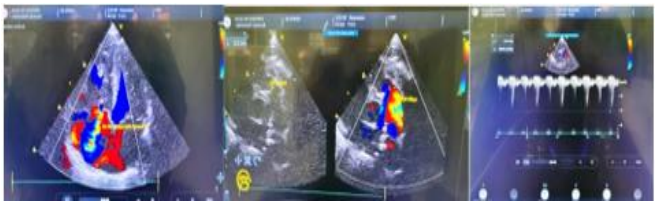

Figure 4. Fetal echocardiography showed

1. 4 chamber view showed RA RV dilated severe TR
2. Parasternal short axis view showed critical pulmonal stenosis and PDA
3. Pressure gradian RV PA showed the severe PS

Result and Discussion

Critical Congenital Heart Disease (CCHD) refers to a subset of congenital heart defects that are life-threatening and require surgical or catheter-based intervention within the first year of life (Gomella et al., 2020; Mahle et al., 2009). These defects arise from malformations of one or more cardiac structures during early embryonic development. Globally, the incidence of CCHD is estimated at 2–3 per 1,000 live births (Hoffman & Kaplan, 2002; Oster et al., 2013). These anomalies represent a significant cause of neonatal morbidity and mortality. The clinical presentations of CCHD—such as shock, cyanosis, and respiratory distress—often mimic those of other neonatal conditions, increasing the risk of misdiagnosis or delayed recognition. Failure to promptly identify CCHD may result in acute cardiovascular collapse and early neonatal death. Routine screening using pulse oximetry has proven effective in differentiating CCHD from other hypoxemic conditions, supporting its role as a vital tool in early detection (Taksande & Jameel., 2021). Pulse Oximetry Screening (POS) in newborns has been shown to significantly improve early recognition of CCHD. Many countries have integrated POS into national screening protocols following its demonstrated utility (Meberg et al., 2009). The diagnostic yield of POS is comparable to other established newborn screening programs. For instance, the prevalence of CCHD (2–3/1,000 births) is similar to or higher than that of other conditions such as cystic fibrosis (0.5/1,000), hearing loss (1–3/1,000), and congenital hypothyroidism (1/4,000), all of which are routinely screened (Nelson et al., 2008). Furthermore, the false-positive (FP) rate for POS ranges between 0.05% and 0.5%, which is comparable to or better than that of universal hearing screening (0.5–4%) and newborn thyroid screening (approximately 2%) (Nelson et al., 2008). These findings highlight the cost-effectiveness and feasibility of implementing POS as part of standard newborn screening protocols. Implementation of pulse oximetry screening has improved early diagnosis of critical Congenital Heart Disease (CHD) in neonates (Desai et al., 2019; Jam et al., 2025).

In this case series, all three neonates underwent pre- and post-ductal pulse oximetry screening during early stabilization, which demonstrated persistent hypoxemia suggestive of duct-dependent circulation. These abnormal findings on pulse oximetry prompted early targeted echocardiographic evaluation, enabling timely diagnosis of right-sided obstructive lesions. The utilization of pulse oximetry screening in these cases supports the recommendation that pulse oximetry should be integrated into routine postnatal screening, especially in resource-limited settings where prenatal detection of CCHD remains suboptimal. This aligns with the findings of Taksande et al., who emphasize the pivotal role of pulse oximetry in the early recognition of CCHD and prevention of delayed intervention that may lead to high neonatal mortality. Despite advances in neonatal screening and care, delayed recognition remains a major contributor to morbidity and mortality, especially in resource-limited settings. Studies show that undiagnosed CCHD contributes significantly to early neonatal deaths, with mortality reaching 30% – 50% in duct-dependent

lesions if not promptly managed (Peterson et al., 2013). Some investigators proposed that oximetry screening should include measurements of both upper and lower extremities and that differences in Spo_2 of more than 3% or 4% be used to identify newborns with CCHD who might otherwise be missed by measuring lower-extremity Spo_2 alone (Reigh et al,, 2003).

Early identification of Critical Congenital Heart Disease (CCHD) in neonates is crucial to reduce the risk of cardiogenic shock, metabolic acidosis, and death prior to definitive intervention (Mahle et al., 2009). In our case series, all three neonates presented with persistent central cyanosis within the first days of life. Pre- and post-ductal oxygen saturation screening was instrumental in raising early suspicion of duct-dependent congenital heart lesions. Pulse oximetry has proven to be a simple and effective screening tool for CCHD. A difference >3% between pre-and post-ductal SpO_2 or absolute SpO_2 <90 – 95% are considered abnormal, warranting further evaluation (Mahle et al., 2009; Thangaratinam et al., 2012). In our cases, preductal saturations ranged between 70–80% and postductal between 50–60%, consistent with significant right-to-left shunting.

Delayed diagnosis of Critical Congenital Heart Disease (CCHD) carries a serious risk of mortality, morbidity, and handicap. As echocardiography is commonly used to diagnose congenital heart disease (CHD), echocardiographic investigations in newborns may be helpful in detecting CCHD earlier and with higher sensitivity than when using other screening methods. The present study aimed to evaluate the effectiveness of echocardiographic screening for CCHD in a tertiary care center(Kondo et al., 2018). Bedside echocardiography performed shortly after initial suspicion confirmed the diagnosis in each case. Echocardiography is a key diagnostic modality when taking care of ACHD secondary to its availability, portability, safety, and ease of use. It has the ability to define anatomy, estimate hemodynamics, and provide data to measure outcomes. The spectrum of echocardiography involves 2-dimensional (2D), Color Flow Doppler (CFD), Doppler-Tissue Imaging (DTI), pulse wave Doppler (PWD), agitated saline, ultrasound-enhancing agents, stress echocardiography, and Transesophageal Echocardiography (TEE). Over the last 10 years, conversion from analog to digital signal processing has allowed for the advent of harmonic, 4-dimensional (4D), and speckle tracking imaging (McLeod et al., 2018).

In all three presented cases—Ebstein anomaly with pulmonary atresia, duct-dependent critical CHD with DORV, and critical pulmonary stenosis with severe tricuspid regurgitation—echocardiography played a pivotal role in establishing the definitive diagnosis. Despite initial clinical suspicion raised by symptoms such as central cyanosis and abnormal pulse oximetry readings (notably a significant pre- and post-ductal SpO_2 gradient), it was bedside echocardiography that confirmed the specific anatomical defects in each case.

Echocardiography allows for detailed visualization of complex structural anomalies, including right atrial and ventricular enlargement, tricuspid valve malformation, pulmonary atresia, Double Outlet Right Ventricle (DORV), Ventricular Septal Defect (VSD), Atrial Septal Defect (ASD), and the presence and flow dynamics of the ductus arteriosus. Its ability to provide real-time, non-invasive, and comprehensive anatomical and hemodynamic information makes it an essential diagnostic tool for the early and accurate identification of Critical Congenital Heart Disease (CCHD), especially in neonates presenting with nonspecific clinical signs. These cases highlight that while screening tools such as pulse oximetry are valuable for detecting at-risk neonates, echocardiography remains the gold standard for confirming diagnoses and guiding timely clinical decisions, including the administration of prostaglandin E1 or planning for surgical or catheter-based interventions. Echocardiography is regarded as the most valuable method for diagnosing Congenital Heart Disease (CHD), enabling detailed anatomical assessment through multiple two-dimensional views. These include subcostal long and short axes, apical four-chamber, parasternal long and short axes, and suprasternal windows, all of which delineate comprehensive cardiac anatomy across different imaging planes (Gewitz et al., 2006). Echocardiography remains the gold standard for identifying structural cardiac anomalies and assessing ductal patency in neonates (Myung et al., 2015; Zhao et al., 2021). Critical Congenital Heart Diseases (CCHDs) comprise a spectrum of structural cardiac malformations that can severely compromise systemic or pulmonary circulation in the early neonatal period. A significant proportion of these conditions are ductus arteriosus–dependent, wherein neonatal survival relies on the continued patency of the ductus arteriosus—a vital fetal vascular connection between the pulmonary

artery and the aorta. Timely echocardiographic diagnosis enables early initiation of prostaglandin E1 (PGE1) infusion to maintain ductal patency and preserve adequate systemic or pulmonary blood flow (Hoffman & Kaplan., 2002). The global incidence of Congenital Heart Disease (CHD) is estimated to range between 6 to 8 per 1,000 live births (Friedman & Fahey., 1993), with CHD-related mortality accounting for approximately 3% of infant deaths. Not all CHDs are detectable prenatally or immediately postnatally, and delays in diagnosis may result in significant morbidity and mortality. High-risk lesions associated with early neonatal demise if untreated include hypoplastic left heart syndrome (HLHS), Coarctation Of The Aorta (CoA), Interrupted Aortic Arch (IAA), Transposition Of The Great Arteries (TGA), Total Anomalous Pulmonary Venous Return (TAPVR), critical Aortic Stenosis (AS), Pulmonary Atresia (PA), and Tricuspid Atresia (TA). Consistent with these life-threatening defects, the cases presented in this report highlight three distinct forms of CCHD that required urgent echocardiographic evaluation: (1) Ebstein anomaly with pulmonary atresia and small PDA, (2) ductus-dependent CCHD with tricuspid atresia, pulmonary atresia, double outlet right ventricle (DORV), and inlet VSD, and (3) critical pulmonary stenosis with severe tricuspid regurgitation and small PDA. Although individually rare, CHDs collectively contribute substantially to neonatal mortality. Early detection and intervention can significantly reduce the mortality rate associated with CCHD, from approximately 2–3 per 1,000 to 0.6–0.8 per 1,000 live births (Yun, 2011).

Prior to the use of prostaglandins (PGE_1) many infants with ductus arteriosus dependent Congenital Heart Disease (CHD) born outside tertiary care centres did not survive the period from diagnosis to inter-hospital transportation for surgery. Development in the area of neonatal cardiac surgery has increased the importance of rapid diagnosis and stabilisation of infants with CHD. Survival after cardiac defect repair has increased in the past decade. Perhaps an important factor in this increase appears to be the early use of PGE_1 to provide a more clinically stable patient for surgery (Sharma et al., 2011). In our case series, three neonates presented with critical duct-dependent CHD requiring urgent recognition and intervention. The first case involved Ebstein anomaly with functional pulmonary atresia, in which PGE_1 was vital to maintain right-to-left shunting through a patent ductus arteriosus. The second case was diagnosed with Double Outlet Right Ventricle (DORV) with large VSD, where systemic perfusion was dependent on ductal flow, and early PGE_1 infusion was life-saving. The third case featured critical pulmonary stenosis with severe tricuspid regurgitation, where PGE_1 maintained adequate pulmonary circulation pending definitive intervention. These cases illustrate the life-saving role of early prostaglandin therapy in ductal-dependent cardiac lesions and emphasize the necessity of prompt diagnosis and stabilization in peripheral or resource-limited settings.

Since mid-1970s the use of PGE_1 to maintain ductal patency has significantly improved the outlook of neonates with CHD characterised by marked restriction of pulmonary blood flow. Dilatation of the ductus is manifest by an immediate rise in PO_2 often by as much as 20 to 30 mmHg. Selective aotograms have revealed wide dilatation of the ductus minutes after an infusion of PGE_1 in prescribed dosage (Coeni et al., 1973). Prostaglandin E1 (PGE1) is used to keep the ductus arteriosus patent and can be life-saving in neonates with ductal-dependent cardiac lesions. PGE1 is used to promote mixing of pulmonary and systemic blood flow or improve pulmonary or systemic circulations, prior to balloon atrial septostomy or surgery. PGE1 therapy may cause several short-term and long-term adverse effects (Akkinapally et al., 2018). In our case, a term neonate was delivered with immediate central cyanosis and severe hypoxemia, unresponsive to supplemental oxygen therapy. Initial pulse oximetry revealed a pre-ductal SpO_2 of 62% and post-ductal SpO_2 of 60%, suggesting critically reduced pulmonary blood flow. Echocardiographic assessment identified Pulmonary Atresia with Intact Ventricular Septum (PA-IVS)—a prototypical ductal-dependent cardiac lesion. Intravenous PGE_1 infusion was promptly initiated at a dosage of 0.05 µg/kg/min, resulting in a marked clinical improvement. Within the first hour, oxygen saturation increased to 88%. Repeat echocardiography confirmed ductal patency with improved antegrade flow into the pulmonary artery. The neonate was subsequently stabilized and safely transferred to a tertiary cardiac center for further evaluation and definitive surgical management. This case underscores the pivotal importance of early recognition and timely administration of PGE_1 in neonates with ductal-dependent congenital heart disease, as a strategy to minimize hypoxic injury and improve clinical outcomes.

Prostaglandin E1 (PGE1) has been used for decades in the medical treatment of ductal dependent critical congenital heart disease in neonates. In ductus-dependent critical congenital heart disease (CCHD), Prostaglandin E1 (PGE1), also known as alprostadil, is an essential life-saving medication. Prostaglandin E1 (alprostadil marketed as 'Prostin VR ') and prostaglandin E2 (dinoprostone) are used to maintain a patent ductus arteriosus and the dose of medication depends on the clinical presentation. Delay in starting prostaglandin infusion can have deleterious effects on infants and can even lead to death. PGE1 functions by dilating the vascular smooth muscle of the ductus arteriosus, thereby preventing its closure (Sing et al., 2018). Prostaglandin infusion is indicated as a brief therapy in neonates with critical CCHD with duct dependent circulation to maintain patency of the ductus arteriosus until definitive intervention can be done (Alhussin et al., 2016). The closed ductus arteriosus can be reopened within 30 minutes after initiation of PGE1. The opened ductus arteriosus will increase pulmonary or systemic circulation and improve the cyanosis and shock (Momma et al., 1980). It plays a critical role in maintaining ductal patency in neonates with cardiac lesions that obstruct normal systemic or pulmonary blood flow. In neonates with ductus-dependent critical Congenital Heart Disease (CCHD), Prostaglandin E1 (PGE1) serves as more than an emergency therapy—it functions as a vital bridge that buys time for safe transfer, comprehensive evaluation, and definitive catheter- based or surgical intervention. Without a Patent Ductus Arteriosus (PDA), these infants are at risk of rapid cardiovascular collapse, hypoxemia, or shock soon after birth (Willim & Supit, 2021). All three cases of neonates with ductal-dependent critical congenital heart disease (PA-IVS, critical pulmonary stenosis with severe tricuspid regurgitation, and Ebstein anomaly) demonstrate that early administration of Prostaglandin E_1 (PGE_1) is highly effective in maintaining ductal patency, improving oxygenation, and stabilizing hemodynamic status. PGE_1 serves as a vital temporizing therapy before definitive intervention, such as catheter-based or surgical procedures. Timely use of PGE_1 can prevent hypoxic injury and significantly enhance the chances of survival during transfer to a specialized pediatric cardiology center. Importantly, the injudicious use of supplemental oxygen in neonates with undiagnosed duct-dependent CCHD can be harmful. High-concentration oxygen may lower pulmonary vascular resistance, reduce right- to-left shunting, and potentially close the ductus arteriosus prematurely, leading to sudden cardiovascular collapse (Martin et al., 2007). Therefore, accurate differentiation of pulmonary versus cardiac causes of cyanosis using SpO_2 screening and echocardiography is essential prior to initiating high FiO_2 therapy. These cases highlight that early diagnosis of CCHD using SpO_2 screening and bedside echocardiography is vital to initiate appropriate early treatment, particularly prostaglandin therapy, and avoid inappropriate interventions such as excessive oxygen administration. Prompt stabilization enables safe referral and definitive intervention, reducing neonatal morbidity and mortality associated with delayed CCHD recognition. Infants improve clinically and since hypoxemia and acidosis is reversed, pertinent diagnostic studies can be implemented and the patient transferred to a tertiary care centre in a stable condition. PGE_1 has been shown to dilate pulmonary vascular bed and PGE_1 infusion may improve pulmonary blood flow by reducing pulmonary vascular resistance as well (Cassin et al., 1986). In our case series, three neonates with different ductus-dependent cardiac lesions—atrioventricular septal defect (AVSD) with severe pulmonary hypertension, Double Outlet Right Ventricle (DORV) with critical pulmonary stenosis, and Ebstein anomaly with functional pulmonary atresia—showed clinical improvement following the initiation of PGE_1 infusion and respiratory stabilization. Each case required timely recognition and echocardiographic confirmation to prevent rapid decompensation and facilitate appropriate transfer. Neonates with critical congenital heart disease of the ductal-dependent pulmonary circulation type (CCHD-DDPC) require prostaglandin E1 (PGE1) to maintain oxygen saturation until surgery (Najjaroonsri et al., 2024).

Conclusion

This case series highlights the critical importance of early recognition, timely initiation of prostaglandin E1 (PGE1), and prompt referral in the management of ductus-dependent Critical Congenital Heart Disease (CCHD). Clinical signs such as central cyanosis unresponsive to oxygen and pre-/post-ductal SpO2 discrepancies should raise immediate suspicion. Bedside echocardiography and access to PGE1 remain essential tools for initial stabilization. When initiated promptly, PGE1 effectively restores ductal patency, serving as a life-saving bridge to definitive catheter-based or surgical intervention. Improving awareness, screening protocols, and

access to essential therapies can significantly reduce morbidity and mortality in neonates with CCHD, especially in resource-limited settings.

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