CASE REPORT

Pediatrics Sciences Journal (*PedScij*) 2025, Volume 6, Number 1: 23-30
P-ISSN: 2722-0427, E-ISSN: 2722-1474

Pediatric Sciences Journal

Published by
Pediatrics Sciences Journal

Patent ductus arteriosus and pulmonary hypertension in a fifteen-year-old boy with congenital rubella syndrome and cerebral palsy: a case report

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Received: 2024-12-27
Accepted: 2025-02-18
Published: 2025-03-20

ABSTRACT

Introduction: Congenital Rubella Syndrome (CRS) is characterized by congenital cataracts, congenital heart disease (CHD), hearing loss, and developmental delay. It is caused by maternal rubella infection during pregnancy, transmitted transplacentally or via respiratory droplets. CRS carries a high risk of morbidity and mortality, with approximately 10–20% of affected infants dying within the first year of life. This case report describes a 15-year-old boy with CRS who developed pulmonary hypertension (PH) due to a persistent patent ductus arteriosus (PDA).

Case Description: A 15-year-old boy presented with progressive abdominal distension over one week. Initially suspected of having nephritic syndrome, further evaluation revealed bilateral congenital cataracts, non-cyanotic CHD in the form of PDA, sensorineural hearing loss, and developmental delay, fulfilling criteria for CRS. The patient also exhibited delayed motor milestones (walking at age seven) and limb rigidity suggestive of cerebral palsy (CP). Echocardiography confirmed PDA (0.4 cm) with severe tricuspid and aortic regurgitation, and chest X-ray demonstrated cardiomegaly with PH. The PDA was successfully closed using an Amplatzer Duct Occluder (ADO) via catheterization.

Conclusion: This case underscores the importance of early diagnosis and intervention in CRS patients with PDA to prevent irreversible pulmonary vascular disease. Despite a very late PDA closure at age 15, the patient achieved hemodynamic improvement and maintained functional capacity, highlighting that catheter-based closure remains feasible and beneficial even in adolescence. Multidisciplinary care, including timely cardiac intervention and neurodevelopmental support, can improve quality of life in CRS survivors.

Keywords: congenital rubella syndrome, patent ductus arteriosus, pulmonary hypertension, cerebral palsy.
Cite This Article: Putri, J.H.A., Haris, I.A., Nahdah, J., Putri, J.C., Hamdy, I.H., Pikir, R.R., Adam, O.M., Adiwinoto, R.P. 2025. Patent ductus arteriosus and pulmonary hypertension in a fifteen-year-old boy with congenital rubella syndrome and cerebral palsy: a case report. *Pediatrics Sciences Journal* 6(1): 23-30. DOI: 10.51559/pedscij.v6i1.96

INTRODUCTION

Congenital Rubella Syndrome (CRS) is a group of symptoms that occur in infants and are caused by the rubella virus infection during pregnancy. Serious repercussions, including miscarriage, stillbirth, and severe congenital defects in the unborn child, may arise if rubella infection happens early in pregnancy. The risk of infection and birth defects is highest in the first 12 weeks of pregnancy and decreases thereafter. Common congenital abnormalities in CRS include cataracts, hearing loss, developmental delays, and CHD, most frequently patent ductus arteriosus (PDA).[1,2]

Globally, an estimated 100,000 cases of CRS are reported annually. According to reports, newborns with CRS had a 33% mortality rate.[3] Rubella has an average incubation period of 17 days and can be spread by direct contact or droplets from nasopharyngeal secretions. The virus can pass through the placenta and enter the developing fetal circulatory system in susceptible pregnant women. Up to 80% of children with congenital rubella syndrome will exhibit symptoms within a few years of life, and between 10% and 20% of newborns with the condition pass away within the first year of life. Heart problems account for the majority of CRS deaths.[2-6]

PDA in the setting of CRS is a major contributor to morbidity and mortality if not diagnosed and managed promptly.[5,6] Untreated PDA imposes a chronic left-to-right shunt, leading to increased pulmonary blood flow, elevated pulmonary artery pressures, and eventual pulmonary vascular remodelling. Over time, this progression may result in pulmonary hypertension and right heart failure, complicating both surgical and catheter-based closure later in childhood or adolescence.[7,8] Despite being a relatively benign condition clinically, CRS can arise from a primary rubella virus infection during the early stages of pregnancy. It's possible to diagnose CRS either prenatally or postnatally. The basis for postnatal diagnosis is the use of an

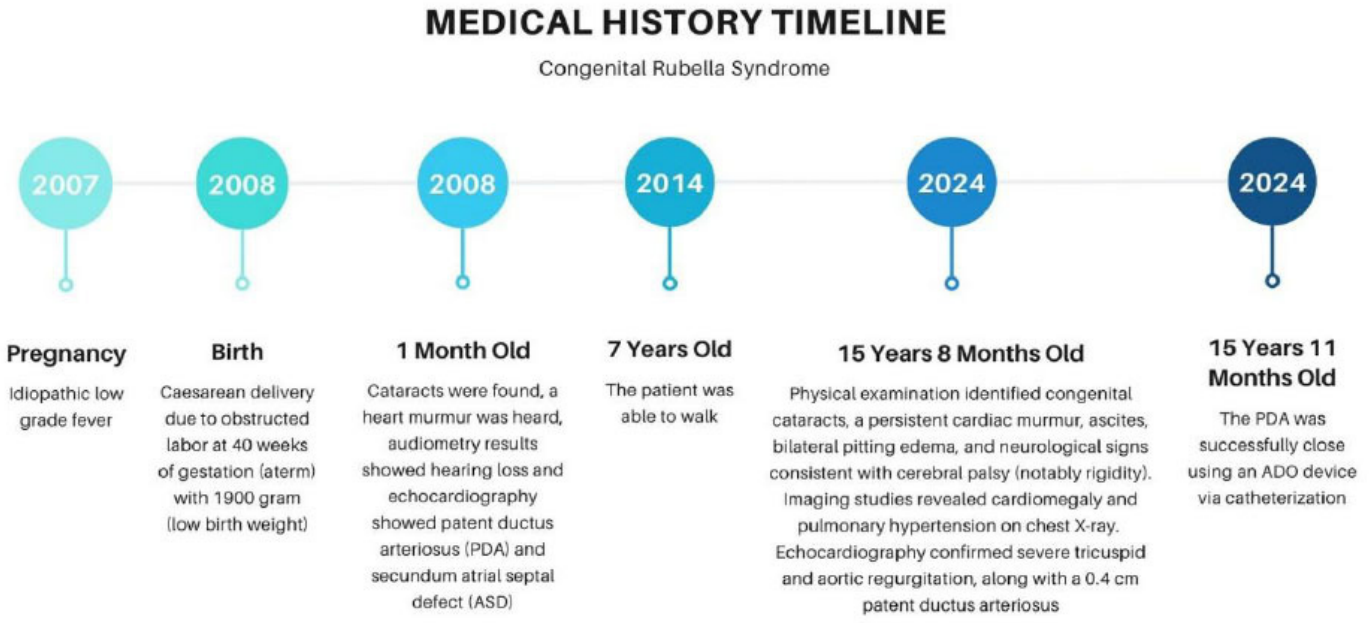

Figure 1. A comprehensive patient history timeline—from birth to the present—provides critical insight into the progression of health and disease, revealing patterns, risk factors, and pivotal clinical events that shape current diagnosis and treatment strategies.

Table 1. Comparative Summary of Echocardiographic Findings

Parameter	2008 (1 mo)	2024 (15 yr)
PDA diameter	0.41 cm	0.40 cm
Secundum ASD diameter	0.21 cm	Closed (spontaneous occlusion)*
Valvular regurgitation	None	Severe TR, Severe AR
Pulmonary artery pressure (est.)	Not elevated	≥ 60 mmHg (Pulmonary Hypertension)
Intervention	Declined surgery	ADO occlusion on April 2, 2024

*(No ASD device was placed; spontaneous closure presumed)

immunocapture ELISA to detect certain RV-IgM. Congenital infection can be verified if RV-IgM is positive by isolating the rubella virus or by using RT-PCR to find the viral genome in oral fluid, urine, and nasopharyngeal swabs.[7-13]

This case highlights the consequences of delayed PDA closure in a CRS patient, demonstrating the interplay between rubella-associated cardiac anomalies and their long-term hemodynamic impact. The objective of this case report is to describe the clinical manifestations of congenital rubella syndrome in children up to 15 years of age at RSPAL Surabaya, where the life expectancy of affected patients is typically reduced.

CASE DESCRIPTION

Pregnancy and Neonatal period

During pregnancy, the mother visited an obstetrician regularly for check-ups. She did not receive vaccinations. She experienced a slight fever for one day, but details of the gestational age are unclear. History of skin rashes, high fever, lumps around the neck, joint pain, coughs and colds, or being hospitalized, and history of traveling were denied. The patient was delivered via caesarean section due to obstructed delivery at 40 weeks of gestation with a birth weight of 1900 grams. There was no blueness or spots on the patient's body when he was born and cried spontaneously. Post-delivery, the patient required NICU admission due to low birth weight.

In 2008, when the patient was 1 month old, he was admitted to St. Vincentius a Paulo Catholic Hospital with complaints of diarrhea. During a physical examination, the pediatrician found cataracts in both eyes, and a murmur was heard, which indicated suspicion of congenital rubella syndrome. The patient underwent an audiometry examination, but his parents no longer had the audiometry examination results, which stated hearing loss. Next, a rubella serological test was performed. His parents likewise lost track of the test results, but they did recall that the mother's and patient's IgM and IgG levels were both positive. Then the patient was referred to dr. Soetomo Hospital to undergo an echocardiography. Echocardiography revealed PDA (0.41 cm) and secundum atrial septal defect (ASD) (0.21). Surgical intervention for the cardiac anomalies was recommended, but declined by the parents due to perceived low success chances.

A full-term infant presented at one month of age with clinical manifestations including cataracts, cardiac murmurs, sensorineural hearing loss on audiometry, and a patent ductus arteriosus (PDA) confirmed by electrocardiogram (EKG), leading to a definitive diagnosis of Congenital Rubella Syndrome (CRS). This constellation of findings underscores the classic presentation of CRS.

Early Childhood

He was able to walk, eat, bathe, and play with his younger sibling before being admitted to the hospital, but his activities still required parental assistance. He did not show any complaints, such as shortness of breath or a bluish face, and he also had no history of taking medication regularly since childhood.

The patient showed significant developmental delays, achieving walking milestones at seven years old. Activities such as eating, bathing, and playing required parental assistance, though there were no significant respiratory or cyanotic complaints. The patient had no history of regular medication use or recurrent hospital admissions during this period.

Current admission

The patient was referred to RSPAL Dr. Ramelan Surabaya on January 1, 2024, with complaints of abdominal swelling, periorbital edema, and leg swelling for a week, suggestive of nephritic syndrome. His parents refuted any prior experiences the patient may have had with similar problems. Physical Examination findings revealed congenital cataracts in both eyes, a persistent murmur, ascites, pitting edema in both legs, and signs suggestive of cerebral palsy (rigidity). Laboratory tests reveal a normal albumin level, which serves as the foundation for excluding the diagnosis of nephrotic syndrome. Chest X-ray indicated cardiomegaly and pulmonary hypertension. Echocardiography confirmed severe tricuspid and aortic regurgitation and a patent ductus arteriosus measuring 0.4 cm. On April 2, 2024, the PDA was successfully closed using an ADO device via catheterization. Following the successful closure of the Patent Ductus Arteriosus (PDA) and subsequent follow-up, the patient demonstrated significant clinical improvement. Notably, there was a marked enhancement in mobility, absence of the previously audible cardiac murmur, and progressive weight gain, suggesting improved hemodynamic function and overall cardiovascular health.

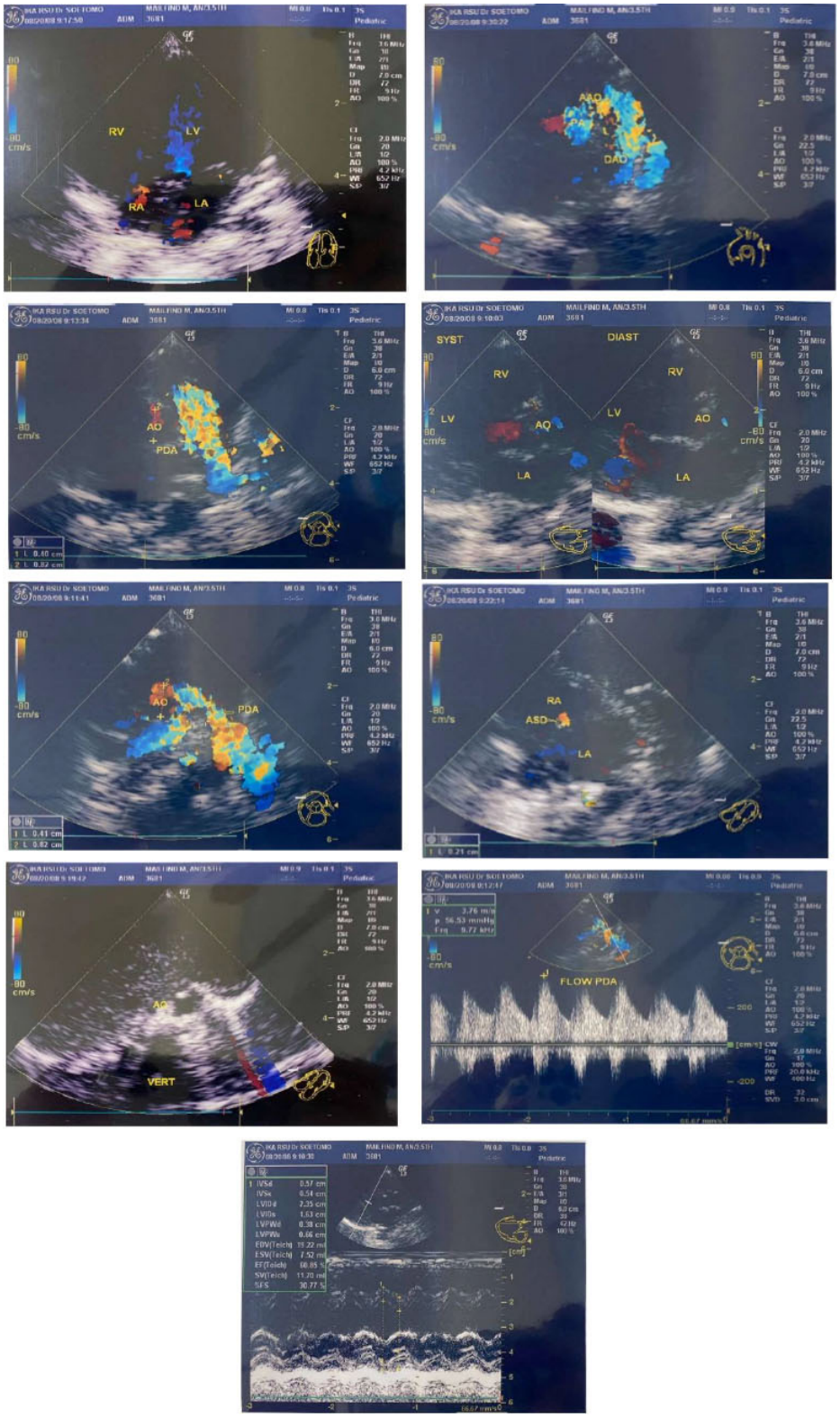

Figure 2. Echocardiography results in 2008 at dr. Soetomo Hospital, Surabaya.

Prognostic Characteristic

The patient exhibits multiple long-term complications associated with Congenital Rubella Syndrome (CRS), including sensorineural hearing loss, bilateral congenital cataracts, congenital heart defects (PDA and ASD), and neurodevelopmental delays suggestive of cerebral palsy. These comorbidities, particularly the combination of cardiac and neurological impairments, indicate a guarded prognosis. However, the successful PDA closure has resulted in notable clinical improvement, especially in cardiovascular stability and functional

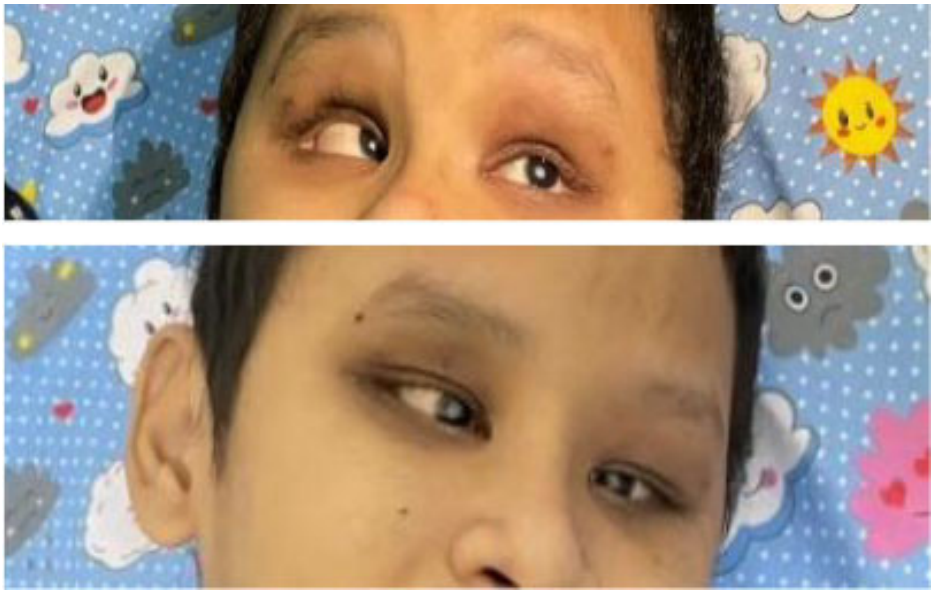

Figure 3. Congenital Cataracts visible in both eyes of the patients with CRS.

mobility. Although the initial presentation included edema and ascites, which raised concern for renal involvement, the presence of normal serum albumin levels reduces the likelihood of nephrotic or nephritic syndrome. Continued multidisciplinary care and early rehabilitative interventions remain essential to support the patient's long-term development and quality of life.

DISCUSSION

CHD that develops as a part of a congenital condition, such as congenital rubella syndrome brought on by the rubella virus, is also influenced by genetic factors.[14-16] This case highlights an exceptionally late PDA closure at 15 years in a child with congenital rubella syndrome (CRS) and long-standing hemodynamic stability despite a large 0.4 cm ductus. The patient in our case met the criteria for congenital heart abnormalities, sensorineural deafness, and cataracts, the typical trio of CRS. Following a one-month hospital stay at the St. Vincentius a Paulo Catholic Hospital in 2008, the patient was diagnosed with CRS based on findings from a pediatrician's physical examination, which revealed bilateral congenital cataracts and cardiac murmurs. Additionally, the patient had an audiometry evaluation, which showed hearing loss. Suspicion of congenital rubella syndrome led to the patient being referred to Dr. Soetomo Hospital for further examination.

Toizumi et al. reported the features of heart abnormalities detected by echocardiography and angiography. Mitral regurgitation occurred in children with CRS-PDA (18%) less frequently than in children without CRS-PDA (41%). In contrast, children with CRS-PDA had higher pulmonary (34%) and aortic (22%) stenosis than children without CRS-PDA (0.7% and 1.7%, respectively). Pulmonary hypertension was shown to be more common in CRS-PDA (76%) than in non-CRS-PDA (51%). Patients with CRS-PDA had a higher proportion of tubular type PDA (16%) than those without CRS-PDA (3%). The diameter of the pulmonary artery (PA) side and the length of the PDA were significantly greater and longer in CRS-PDA compared to non-CRS-PDA. The CRS-PDA had a higher ratio of PA side diameter to aortic side diameter than the non-CRS-PDA.[17-18]

Echocardiography detected pulmonary and aortic stenosis in CRS-PDA patients significantly more frequently than in non-CRS-PDA patients. The findings in this study suggest that both the pulmonary artery and the aorta are undeveloped in patients with CRS, which is followed by poor body size growth and/or arterial narrowing due to intimal proliferation. CRS-PDA has a lower incidence of mitral regurgitation than non-CRS-PDA, presumably because progressive PH lessens the volume load of the left heart due to high pulmonary resistance.[18]

Patients with CRS-PDA possess a higher proportion of tubular type PDA (type C) (according to the Krichenko classification), a bigger diameter of the pulmonary artery side, and a longer PDA than those with non-CRS-PDA. Pulmonary hypertension is particularly common in CRS-PDA, leading patients to develop symptoms of PH or heart failure at an earlier age.[18] Smooth muscle cells move into the subendothelial region, resulting in intimal thickening and ductal closure. PDA tissue from patients with CRS has poorly established intimal thickening. However, no studies have addressed the issue of histological differences between types of PDA; however, based on the morphological similarity (tubular shape) of CRS-PDA to the ductus arteriosus seen in the foetus, it is suspected that rubella virus infection prevents intimal thickening in the early foetal phase.[18] Several factors contributed to the development of ascites in this patient, including a 0.4 cm PDA that did not close until the age of 15 years.

Typically, the systemic and pulmonary circulations are distinct. The pulmonary vein transports high-oxygen blood from the lungs to the left atrium, where it is pumped to the left ventricle, from which it is sent to the rest of the body via the aorta. Furthermore, blood with low oxygen levels from the rest of the body flows through the vena cava to the right atrium and is pumped to the right ventricle, after which the pulmonary artery transports low-oxygen blood to the lungs to exchange carbon dioxide for oxygen. However, in infants with PDA, where the aorta and pulmonary artery remain connected, high-oxygen blood transported by the aorta can go to the pulmonary artery, causing blood to flow back to the pulmonary circulation rather than throughout the body. If the PDA is significant, the blood pushed into the pulmonary artery might make the heart and lungs work harder, causing the lungs to become clogged. Uncompensated water retention leads to lung congestion. This is related to the pathophysiology of cardiogenic lung oedema, which raises pressure in the pulmonary vein and pulmonary capillaries, causing fluid to build up in the lungs' interstitial space. Pulmonary hypertension was caused by an increase in pulmonary artery pressure

shortly after lung congestion. Prolonged hypertension in the lungs might result in right heart failure.[19,20] There is cardiomegaly and PH on the chest x-ray that this case presents. Tricuspid regurgitation and severe aortic regurgitation are seen on echocardiography. The state of this illness may deteriorate circulation and heart health. Right heart failure may result from a combination of pulmonary hypertension, PDA, and valve regurgitation. Ascites and oedema in the extremities can result from right heart failure, which can also raise systemic vein pressure.

Eisenmenger Syndrome (ES) proceeds from pulmonary hypertension. Patients suffering from VSD, ASD, or PDA may also experience Eisenmenger syndrome. Lung vascular hypertension, which is subsequently brought on by a chronic systemic left-to-right shunt, will eventually raise lung vascular resistance. Right-to-left shunt deteriorates the circulatory system over time. There will be systemic desaturation in every organ. As a result, patients suffering from ES may exhibit symptoms like syncope, exhaustion, and dyspnea with exertion. Secondary erythrocytosis brought on by chronic cyanosis will result in iron deficiency, blood hyperviscosity, and thrombocytopenia.[21]

In a case presentation by Dimopoulos, ES can arise in PDA patients with pulmonary arterial hypertension (PAH). Patients with PDA who also have PAH in their teens or adult years may develop ES.[22] Our patient's activities were intended to stop ES from happening. According to research by John et al., which also contains a case study on a PDA patient, ES that results from PDA is uncommon since PDA is treated before PAH develops. If PDA is left untreated, this leads to the development of ES.[23]

The single-stranded RNA Rubella virus can bring on Congenital Rubella Syndrome. An infection contracted during the first trimester, particularly in the first month of pregnancy, will result in substantial congenital abnormalities in pregnant women who are not immune to the rubella virus. The outcome of the infection is contingent upon the fetus's gestational age at the time of infection, but it can happen at any point throughout pregnancy. 90% of foetuses with congenital rubella infection have an almost 100% chance of developing congenital abnormalities if the infection occurs during the first 12 weeks of pregnancy. 50% of congenital abnormalities and 60% of infections occur between 13 and 17 weeks of pregnancy. There is a very slight chance of congenital abnormalities and a 25% risk of infection between weeks 18 and 24 of pregnancy. Congenital heart disease, congenital cataracts, or microphthalmos are examples of the ocular defects that make up the classic trio of CRS, along with sensorineural deafness. Low birth weight, microcephaly, hepatosplenomegaly, and thrombocytopenic purpura, which presents as "Blueberry Muffin Spots", are some other symptoms.[10]

The simultaneous development of the heart, inner ear, and lens during embryonic development may result in clinical symptoms. An infection on day 36 can result in cataracts; an infection on day 46 can cause heart problems; and an infection on day 62, up to 16 weeks of age, can result in inner ear defects. Developmental and growth abnormalities, learning difficulties, autism, and schizophrenia are all possible outcomes for children with CRS.[1,14,24,25]

One of the congenital cardiac disorders that can affect CRS patients is PDA. PDAs can be classified into three categories according to their diameter: small PDAs, which have a diameter of ≤ 1.5 mm; moderate PDAs, which have a diameter of between 1.6-2.5 mm; and large PDAs, which have a diameter > 2.5 mm.[26]

Congenital cataracts and murmurs were discovered during the patient's physical examination by the pediatrician, who then referred the patient to Dr. Soetomo Hospital for echocardiography, where it was found that the patient had a PDA measuring 0.41 cm in diameter and a secundum ASD measuring 0.21 cm. Based on the patient's clinical presentation, it all fit the criteria for congenital rubella syndrome (congenital cataracts, hearing loss, and CHD). When the patient was admitted to the hospital at RSPAL, dr.. Ramelan on January 3, 2024, fifteen years later, PDA was still discovered during another echocardiogram.

Congenital rubella syndrome is thought to affect 100,000 people annually, and infants who contract CRS may die at a rate of up to 33%.[3] Rubella virus infections are asymptomatic in more than half of cases. In some cases, pregnant women infected with the rubella virus will experience mild fever with an incubation period of 12-23 days, and a maculopapular rash that starts on the face and spreads to the extremities. Other complaints may also include arthralgia, regional lymphadenopathy, coli, and conjunctivitis.[10,27]

Diagnosing congenital rubella syndrome is done by anamnesis, physical examination, and supported examination, such as serology testing for the rubella virus. In this case, the classic triad of clinical features of CRS is present, namely CHD in the form of PDA, hearing impairment, and bilateral congenital cataracts. During the physical examination of the heart, a continuous murmur was heard. This can occur due to a significant pressure gradient between the aorta and the pulmonary artery during both systole and diastole; a left-to-right shunt happens in both phases, resulting in a continuous murmur.[28-31] During the echocardiographic examination, a PDA was found, and when a chest X-ray was performed, cardiomegaly and PH were observed as a result of the PDA. Excess volume in the left ventricle and atrium will lead to left ventricular hypertrophy (LVH), and if an examination is conducted with an electrocardiogram (ECG), left atrial hypertrophy (LAH) will be observed. The free flow from the aorta will cause an increase in pressure in the pulmonary artery, leading to PH.

The development of PH can occur right after birth. After birth, pulmonary arterial pressure decreases; this is a physiological mechanism. There is also thinning of the tunica media of the smooth muscle in the blood vessels. If there is a significant PDA, the systemic and pulmonary pressures will become balanced, leading to a delay in the decrease of pulmonary arterial pressure. Due to the high pressure, the pulmonary arterioles that are not mature or are thinner than normal lead to a slower decrease in pulmonary resistance during the first 3-4 months of life. Although the pulmonary resistance is not normal, it is low enough to allow for excess pulmonary blood flow. The pulmonary blood flow will continue to increase along with the decrease in

pulmonary vascular resistance. However, the pulmonary arterial pressure remains high due to the equalization of pressure and high flow through a large shunt, rather than an increase in pulmonary vascular resistance. The tunica media of the pulmonary artery is the first thing to change from early infancy. In addition to the inflammation of the blood vessel walls, there is endothelial cell dysfunction and an imbalance of vasoactive mediators that lead to vasoconstriction. There is intracellular matrix deposition and remodelling of blood vessels involving smooth muscle hypertrophy and proliferation due to abnormal expression of fibroblast and vascular endothelial growth factors. Hemodynamically significant PDA (hsPDA) persisting for more than a year will cause the tunica intima to thicken. If it lasts more than 2 years, it can cause fibrosis, which may exceed the lumen of blood vessels and reduce the compliance of the pulmonary arteries. This will increase the resistance of the pulmonary blood vessels and decrease blood flow. Thus, the left-to-right shunt decreases, and there is a temporary improvement in the complaints of heart failure. However, the right-to-left shunt will increase pulmonary artery resistance, which can surpass systemic vascular resistance. In at least 50% of patients with large chronic hsPDA who are untreated, irreversible changes in the pulmonary blood vessels can occur by the age of 2 years. Even in patients with PAH and reversible pulmonary vessels, the resistance of the pulmonary blood vessels takes time to return to normal after PDA closure. This, at least theoretically, may indicate the need for earlier PDA closure during the medial muscularization phase.[28]

To help establish the presence of a rubella virus infection, serological tests for rubella IgM and IgG are needed. IgM can be detected 4-5 days after rubella symptoms appear. The IgG antibody titer increases 1-2 weeks after the rash appears on the body. As a preventive measure, women planning to become pregnant with unclear immunization status are given the MMR vaccine. The contraindication for the MMR vaccine is one month before pregnancy or during pregnancy because the MMR vaccine contains live viruses, which pose a risk of causing fetal malformation.[10]

In our case, the patient's mother only reported having experienced a mild fever for one day, without the presence of maculopapular rashes, swollen lymph nodes, arthralgia, or conjunctivitis. The patient's mother did not undergo further examination regarding the mild fever she experienced. The patient's mother has an unclear immunization status.

The management of PDA can be carried out through either pharmacological or non-pharmacological methods. The pharmacological management includes the use of indomethacin, which is a cyclooxygenase inhibitor that targets the synthesis of prostaglandins, followed by acetaminophen and ibuprofen. Meanwhile, non-pharmacological management can be performed through catheterization or surgery. In general, if the ductus size is >5 mm, surgical closure of the PDA can be performed. Closure is generally performed surgically by placing a surgical clip through a posterolateral left thoracotomy. Ductus ligation is often followed by cardiopulmonary deterioration within a few hours after the procedure, seemingly due to changes in afterload, which leads to impaired left ventricular systolic performance; this complication is more likely to occur in infants who undergo ligation at less than 30 days postnatally. However, if the duct size is relatively small, < 5mm, then closure can be performed through coil catheterization.[29]

Although there is no clear consensus regarding the selection of tools used in catheterization closure methods, this approach has several advantages, such as a high success rate, shorter hospital stays, minimized bleeding, low morbidity rates, and the absence of traumatic wounds. This method is also chosen when patients have heart or respiratory conditions that increase the risk of surgery, and for patients who experience psychological stress due to unacceptable surgery. This method can also be used on patients of all ages.[30]

The Amplatzer duct occluder is a device that is widely used to close medium to large-sized ducts in the United States. Closure of a PDA with a small duct size is performed using Gianturco vascular occlusion coils. When installed in the aortic ampulla, a blood clot forms around the wire, blocking blood flow and leading to endothelialization. Meanwhile, the ADO will be implanted antegrade from the femoral vein. This tool is a cone-shaped device that can expand itself. There is a closure rate of 98% or more within 6 months with minimal complications and no deaths.[30,31] In our case, an ADO procedure was performed to close the PDA with access through the right femoral vein.

According to Downing et al.'s research, one of the factors that makes patients with CHD able to maintain survival is the type of CHD suffered. CHD, such as hypoplastic left heart syndrome, single-ventricle defects, and tricuspid atresia relatively short survival.[32] Patients suffering from PDA, which supports longer survival than patients with CHD mentioned above. Trojnarska et al.'s research indicates that surgical intervention does not predict a patient's survival in CHD because some patients with simple CHD have stable hemodynamics, meaning their blood oxygenation and heart function are still normal.[33] The patient had been diagnosed with PDA since he was 1 month old, and no surgery was performed at all. The patient only underwent surgery when he was 15 years old. The patient may have stable hemodynamics so that blood oxygenation can work properly and enable the patient to maintain his survival until now.

The patient is experiencing developmental delays, having only been able to walk at the age of seven. During the physical examination, rigidity was observed, which may indicate CP. Infections that occur during pregnancy, such as those caused by TORCH infection, are causes of neurological developmental disorders that can have long-term impacts. However, the proportion of children with specific disabilities caused by TORCH infections appears to be only 5% to 10% or even less. Infections that occur within the womb, especially subclinical infections associated with premature birth, are suspected to be a cause of neurological developmental disorders. Rubella infection is one of the common congenital infections that can lead to long-term neurological developmental disabilities,

including CP. The rubella virus can infect the uterus and placenta, which can cause brain damage in the fetus.[34-38]

Limitations of the study

There are some study limitations. First, the diagnosis of cerebral palsy in this case was limited by the patient's acute illness and inability to walk, which restricted the ability to perform a thorough physical examination, particularly in assessing muscle tone and gait pattern. In addition, the anamnesis was suboptimal due to the limited information provided by the parents. These factors may affect the accuracy of the diagnosis and the classification of the CP subtype. Second, Neuroimaging (e.g., brain MRI) was not available to confirm the subtype and severity of CP, limiting correlation between neurological and rubella-related pathology. Third, Lack of serial pulmonary vascular resistance measurements precludes formal assessment of the reversibility of pulmonary hypertension. Fourth, missing parental records from 2008 (IgM/IgG titers, early audiometry) limit the precise timing of CRS confirmation.

CONCLUSION

CRS is a collection of symptoms with a classic triad of cataracts, heart defects, and sensorineural deafness caused by the rubella virus infection during pregnancy. The risk of CRS will decrease as the mother's gestational age increases. Prenatal diagnosis of CRS is recommended for mothers diagnosed with rubella by RT-PCR or with rubella-specific IgM antibodies. Treatment of CRS is adjusted according to its clinical manifestations. This case report shows a patient with congenital rubella syndrome with PDA who has stable hemodynamics, so that the patient can survive until the age of 15 years.

DISCLOSURE

Funding

None.

Conflict of Interest

The authors declare that there is no conflict of interest.

Author Contribution

All authors equally contribute to the study from the conceptual framework, data acquisition, literature search, manuscript preparation, and editing. We agree on this final version of the manuscript to be submitted to this journal.

Ethical Approval

The case report has been approved by the Ethics Committee of Dr. Ramelan Naval Central Hospital under approval number 134/EC/KEP/2024.

ACKNOWLEDMENTS

We want to acknowledge the assistance of the case-patient's family for being approachable, cooperative, and for spending their time answering all the questions being asked.

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