Congenital Rubella Syndrome: A Case Report

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Abstract: Congenital rubella syndrome (CRS) is the effect of rubella virus infection during 8-12 weeks of gestation. In Indonesia, the incidence of CRS annually is 0.2/1000 live newborn. The most common defects of CRS are hearing defects (deafness), eye defects (cataract, congenital glaucoma), and cardiac defects (PDA). We reported a case of congenital rubella syndrome in a 5-month-old boy. He was first diagnosed with congenital rubella syndrome 4 months ago (confirmed by positive of anti-rubella IgM with congenital cataract and congenital heart disease-PDA and ASD). He was admitted to Adam Malik Hospital due to congestive heart failure Ross III, bronchopneumonia, marasmus, and anemia. We treated the patient with bed rest, oxygen supplementation, broad-spectrum empiric antibiotics, antipyretic, diuretic, angiotensin converting enzyme inhibitor, and nutritional management according to pediatric nutrition.

1 INTRODUCTION

Congenital rubella syndrome (CRS) is an illness in infants that results from maternal infection with rubella virus during pregnancy. When rubella infection occurs during pregnancy, serious consequences such as miscarriages, stillbirths, and a constellation of severe birth defects in infants may happen (Lanzieri, 2012). In Indonesia, the incidence of CRS annually is 0.2/1000 live newborn. Surveillance data of 2015 showed off 979, newly diagnosed CRS case from 4.89 million live newborns (Kemenkes RI, 2017).

The natural history of congenital rubella initiated by maternal viremia presents for several days before the onset of rash. Maternal viremia may be followed by placental infection and subsequent fetal viremia, leading to disseminated infection involving many fetal organs. Timing is crucial, as the highly susceptible period of intrauterine rubella is during the first 8 to 12 weeks (Gershon, 2004).

2 CASE REPORT

A 5-month-old male came to the emergency unit at Adam Malik Hospital with a chief complaint of shortness of breath. He had experienced the symptom since 2 weeks prior to admission, described as fast breathing and chest indrawing. It got worsened within two days prior to admission and aggravated by feeding. One week prior to admission, the patient was noted to have developed an intermittent fever. Four weeks prior to admission, the patient was noted to have an intermittent productive cough. At that period, he was also noted to be pale, without a history of bluish discoloration of skin and lips.

At 2 months old, he had the right eye cataract surgery. He was then noted to have lost weight gradually. The highest weight noted was 4000 g at 3 months old. Five months prior to admission, the patient noted to have interrupted feeding. He was noted to have fast breathing during feeding, hence the feeding activity was often interrupted.

On physical examination, his initial body weight was 3000 gr, body length was 56 cm, and mid-upper arm circumference was 9 cm. Nutrition status was marasmus (based on the WHO 2006 Growth chart weight for length z score, boys age 0-5 years). The patient was alert but looked ill. The blood pressure was 75/40 mmHg, heart rate of 160 bpm, respiratory rate of 62 bpm, and body temperature of 37.5 °C (axilla), peripheral oxygen saturation was 90% at room air and 96% at oxygen 1 lpm via nasal cannula.

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Head circumference was 36.5 cm (microcephaly), with open flat anterior fontanel, no old man face, and palpebral conjunctiva was pale and sclera was anicteric. There was no palpebral edema. The right pupil was 3 mm and reactive to light while the left pupil cannot be assessed since it was covered with whitish discoloration. Abnormal repetitive, uncontrolled bilateral eye movement from side to side was noted. There was no nasal flaring and cyanosis at lips and tongue. No cervical lymphadenopathy was noted.

He had symmetrical chest expansion, with intercostal and epigastric retractions, the prominent appearance of the intercostal area due to thinning of subcutaneous fat. The respiratory rate was 62 bpm, regular, coarse rales on both lung fields, with no wheezing, equal fremitus. Ictus cordis was not visible and but palpable at IV-V intercostal space on the left midclavicular line, the thrill was felt. The heart rate was 160 beats per minute, regular, with continuous murmur grade 4/6 on the left infraclavicular area, radiating to the back and the thrill was palpable.

Abdomen looked globular, soft on palpation, with normal bowel sound with no palpable liver, good skin turgor. There was no edema on extremities, but with muscle hypotrophy and thinning of subcutaneous fat. The blood pressure was 70/40 mmHg, peripheral pulses were full and regular, capillary refill time less than 2 seconds. There was no abnormality on genitalia and scrotum examination, both testicles are palpable.

Complete blood count revealed anemia, leukocytosis, electrolyte imbalance, low serum iron, normal liver, and kidney function. Blood culture was drawn, revealed no growth. Chest X-ray revealed bronchopneumonia and congenital heart disease with cardiomegaly. Echocardiography revealed secundum atrial septal defect (ASD) Ø 3.7 mm and PDA Ø 3.9 mm.

The patient was managed with bed rest, oxygen supplementation, empiric broad-spectrum antibiotics, antipyretic, diuretic, angiotensin converting enzyme inhibitor, and nutritional management according to pediatric nutrition care for marasmus child.

The admitting diagnosis was congestive heart failure Ross III due to cyanotic congenital heart disease (patent ductus arteriosus (PDA) and ASD), bronchopneumonia, marasmus, anemia, and congenital rubella syndrome.

Oxygen supplementation 1 lpm was administered. Antibiotic ampicillin 75 mg/6 hours/IV (25 mg/kg/dose) and gentamycin 15 mg/24 hours/IV (5 mg/kg/day) were started. Paracetamol 40 mg/6 hours (10 mg/kg/dose) per oral was given if temperature >38°C. Anti-cardiac failure medications namely furosemide 1x3 mg (1 mg/kgBW/day) and captopril 2x3.125 mg (1 mg/kgBW/dose) were continued.

The total caloric requirement was 80-100 kcal/kg/day during the initial phase with a total calorie of 210 kcal was received, through F75 with the mineral mix (75 kcal/100 ml) via nasogastric tube (NGT). The F75 was given in an amount of 35 ml every 3 hours and multivitamins. During the transitional phase, the total caloric requirement was 100-150 kcal/kg/day, given through oral nutrition supplement (ONS) with high-calorie infant formula (100 kcal/100 ml) via NGT-oral. The total volume of feeding was increased gradually.

Ophthalmology assessment revealed right eye aphasia and left eye congenital cataract. Subsequent cataract surgery was suggested when the clinical condition had been improved. We also planned for a hearing function test. Unfortunately, the device for hearing function test was out of order. On growth and development assessment, we found global developmental delay.

3 DISCUSSION

In 1941, Norman Gregg, Australian ophthalmologist has first described a syndrome comprising of cataracts and congenital heart disease with or without mental retardation and microcephaly that he associated with rubella infection in the mothers during early pregnancy (Mason, 2016). CRS group classification of clinical signs listed below :

A. Cataract(s), congenital glaucoma, pigmentary retinopathy, congenital heart disease (most commonly peripheral pulmonary artery stenosis, patent ductus arteriosus or ventricular septal defects), hearing impairment.
B. Purpura, splenomegaly, microcephaly, developmental delay, meningoencephalitis, radiolucent bone disease, jaundice that begins within the first 24 hours after birth.

Using these clinical signs, the final classifications of CRS are as follows: (A) Suspect CRS: infant less than 12 months of age with at least one sign from group A; (B) Clinical CRS: infant less than 12 months of age with at least two signs from group A; or At least one sign from group A and one sign from group B without any laboratory confirmation; (C) Confirmed CRS: suspect CRS

with laboratory confirmation shows infant less than 6 months of age with positive Rubella IgM; (D) Congenital rubella infection (CRI): infant less than 12 months of age without any clinical signs of CRS, but who meets the laboratory criteria for CRS (WHO, 2018 and Ministry of Health Republic of Indonesia, 2018).

This patient was diagnosed as confirmed CRS, because he had met the criteria of suspect CRS (congenital cataract, congenital heart disease-PDA, and ASD) and laboratory confirmation of positive anti-rubella IgM when he was 4 months old. Other important feature like hearing impairment may be the only manifestation of congenital rubella. It is probably caused by maldevelopment and degenerative changes in the cochlea and organ of Corti. Hearing impairment severity is variable, and may be overlooked unless detected by an audiometric examination (Gerson, 2004).

PDA occurs if the ductus arteriosus remains patent leading to the left-to-right shunt of the heart. The magnitude of the left-to-right shunt is determined by the resistance offered by the ductus (diameter, length, and tortuosity) (Mason, 2016). Patent ductus arteriosus is also associated with maternal rubella infection during early pregnancy (Park, 2014).

The patient has continuous murmur grade 5/6 on the left infraclavicular area, suggestive of murmur caused by PDA and had developed retardation of physical growth due to the shunts. Transcatheter PDA closure was performed to treat a PDA with a left-to-right shunt that results in any of the following: congestive heart failure, failure to thrive, increased pulmonary blood flow, or an enlarged left atrium or left ventricle, provided the anatomy and patient size are suitable (AAP, 2011)

The treatment of CHF consists of elimination of the underlying causes, treatment of the precipitating or contributing causes, and control of heart failure state (Mason, 2016). If the cause of CHF is a congenital heart defect amenable to surgery, medical treatment is indicated to prepare the patient for surgery (Masarone, 2017). The heart failure state is controlled by the use of multiple drugs, including inotropic agents, diuretics and afterload-reducing agent, along with general supportive measures (Park, 2014).

The benefits of a diuretic include the reduction of systemic, pulmonary, and venous congestion. Angiotensin-converting enzyme inhibitors decrease the afterload and possibly reverses the pathophysiological myocardial remodeling. Inotropes should be reserved for severe reduction of cardiac output (Masarone, 2017). In this case, the patient receives a diuretic (furosemide) and an ACE inhibitor (captopril). However, the inotropes were not given since the hemodynamic was stable and echocardiography revealed good contractility. He was planned for PDA and ASD transcatheter closure when he reached a minimum weight of 6 kg.

Respiratory complications are frequent in children presenting with congenital heart disease. This group of patients is at a greater risk for viral or bacterial pulmonary infections. Pongiglione (2016) stated that in Italy the incidence of respiratory disease was 63.1%. A total of 26.2% underwent hospitalization with a median length of hospital stay was 7 days, and the median age of the patients hospitalized was <1 year old. The most frequent respiratory diseases associated with hospitalizations were lower respiratory tract infections.

This patient experience gradual weight loss when he was 3 months old, without any data regarding the length. On admission, he was presented with severe underweight, severe stunted, diagnosed as marasmus. The etiology of malnutrition in the children with CHD can be grouped into the following three categories: (1) inadequate intake, (2) inefficient absorption and utilization, and (3) increased energy needs (Rodica, 2013). Viera (2007) stated that the caloric intake in hospitalized-children due to congenital heart disease was 50% below the recommended daily allowance. Fatigue upon feeding may explain the decreased intake (Vieira, 2007).

4 CONCLUSION

We have treated a 5-month-old boy who came to Adam Malik Hospital, Medan, Indonesia due to shortness of breath. He was diagnosed with congestive heart failure Ross III due to cyanotic congenital heart disease (PDA and ASD), bronchopneumonia, marasmus, anemia, and congenital rubella syndrome. The patient was treated with adequate therapy. Congenital rubella syndrome was confirmed when he was 4 months old, both laboratory and clinically.

REFERENCES

American Academy of Pediatrics and Society for Cardiovascular Angiography and Intervention. (2011) Indications for cardiac catheterization and intervention in pediatric cardiac disease: A scientific statement

from the American Heart Association. Circulation. 2011; 123:2607-2652.

Gershon A. Congenital rubella. In: Gershon A, Hotez PJ, Katz SL, editors. Krugman's Infectious diseases of children. 11th edition. Pennsylvania: Mosby; 2004.

Kementerian Kesehatan Republik Indonesia. (2017). Petunjuk teknis kampanye imunisasi measles rubella (MR). Direktorat jenderal pencegahan dan pengendalian penyakit Kementerian Kesehatan RI.

Kementerian Kesehatan Republik Indonesia (2018). Pedoman surveilans c*ongenital rubella syndrome* (CRS). Direktorat surveilans dan karantina kesehatan, Direktorat pencegahan dan pengendalian penyakit Kementerian Kesehatan RI.

Lanzieri T, Redd S, Abernathy E, Icenogle J. (2012). Congenital rubella syndrome. Centers for Disease Control and Prevention (CDC): Vaccine Preventable Disease (VPD) Surveillance Manual.

Masarone D, Valente F, Rubino M, Vastarella R, Gravino R, Rea A, et al. Pediatric heart failure: A practical guide to diagnosis and management. Pediatrics and Neonatology. 2017; 58:303-312.

Mason WH. Rubella. In: Kliegman RM, Stanton BF, Geme III JWS, Schor NF, Behrman RE, editors. Nelson Textbook of Pediatrics. 20th edition. Philadelphia: Elsevier; 2016. p1337-1341.

Park MK. Pathophysiology of left-to-right shunt lesions. In: Park's pediatric cardiology for practitioners. 5th edition. Philadelphia: Elsevier; 2014. p124-130.

Pongiglione G, Possidoni A, Paparatti UL, Costanzo AM, Gualberti G, Bonvicini M, et al. Incidence of respiratory disease during the first two years of life in children with hemodynamically significant congenital heart disease in Italy: A retrospective study. Pediatr Cardiol. 2016; 37:1581-1589.

Rodica T. Nutritional approach of pediatric patients diagnosed with congenital heart disease. Acta Medica Marisiensis. 2013;59(2):121-125.

Vieira TCL, Trigo M, Alonso RR, Ribeiro RHC, Cardoso MRA, Cordoso ACA, et al. Assessment of food intake in infants between 0 and 24 months with congenital heart disease. Arq Bras Cardiol. 2007;89(4):197-202.

World Health Organization. (2018). Congenital rubella syndrome. Vaccine Preventable Diseases (VPD) Surveillance Standards.