

E-ISSN: 2378-654X

Recent Advances in Biology
and Medicine

Case Report

Congenital Rubella
Syndrome—Case Report

HATASO, USA

Congenital Rubella Syndrome—Case Report

**Dileep Reddy Gottimukkula¹, Navya Sri Devulapelly¹, Rakesh Chakinala¹, Surender Kagitapu²,
Yogi Morishetty², Nagesh Adla¹, Goverdhan Puchchakayala^{1*}**

¹Department of Clinical Pharmacy, Vaagdevi College of Pharmacy, Warangal 506002, Telangana, India.

²Department of Pediatrics, Kakatiya Medical College, Mahatma Gandhi Memorial Hospital,
Warangal 506002, Telangana, India.

*Correspondence: gov_ku@yahoo.co.in

Received: Sep 22, 2016; Accepted: Dec 1, 2016; Published: Dec 20, 2016

Abstract

Congenital rubella syndrome (CRS) is a consequence of rubella infection that can occur when the virus is transmitted in utero during maternal primary infection. A newborn male baby was admitted for delayed cry, respiratory distress, having a birth weight of 2,150 g. A history of consanguinity marriage of parents. Intrauterine growth restriction (IUGR), cataractous lens, patent ductus arteriosus (PDA), and a positive serology test are present. Vaccination at high-risk groups is necessary in order to avoid the appearance of CRS.

Keywords: Congenital rubella; Newborn; Cardiothalamic groove; Lenticulostriate calcification; Cataract.

1. INTRODUCTION

CRS is a consequence of rubella infection that can occur when the virus is transmitted in utero during maternal primary infection [1]. It has a wide spectrum of presentation which ranges from silent viremia to spontaneous abortions, blindness, deafness, congenital heart disease, and mental retardation [2]. Both deafness and cardiac manifestations occur if viremia takes place before 11 weeks, and viremia between 13-16 weeks results in isolated deafness [3]. Cataracts and microphthalmia occur 60-70%; congenital glaucoma and intracranial calcifications are rare, more so in the neonate [4-7]. Though the exact teratogenic mechanism of in utero rubella infection remains unidentified, viral replication in the cells during fetal organogenesis seems to be accountable for CRS [8].

Infection occurring in the first 12 weeks of pregnancy causes CRS in 90%, with almost a 100% risk of congenital defects. From 13 to 17 weeks, the risk of infection is about 60%, and risk defects about 50%. From 18 to 24 weeks, the risk of infection is about 25%, with hardly any risk of congenital defects [9].

2. CASE REPORT

A newborn male baby was admitted for delayed cries and respiratory distress in the hospital. His history revealed that gestational age at birth was 37 weeks, having a birth weight of 2,150 g. The mother's age was 22 years and age at marriage was 19 years. A history of consanguinity marriage of parents. Amniotic fluid—Meconium stained.

On general examination, the child was lethargic; color, pale; cry, feeble; tone, limp; length, 48 cm; head circumference, 31 cm; and had IUGR.

On clinical examination, the child was moderately active, with temperature 36°C, heart rate 138 beats/min, respiratory rate 68 breaths/min, blood pressure 62/44 mmHg, blood sugar 52 mg/dL, and oxygen saturation 98% with O₂. Cataractous lens, ejection systolic murmur, and seizure-like activity are present; he had splenomegaly, hepatomegaly, and abnormal skull shape—parietal bone upper than temporal and frontal. In lungs, red-color small nodular lesions were present on lower alveolar.

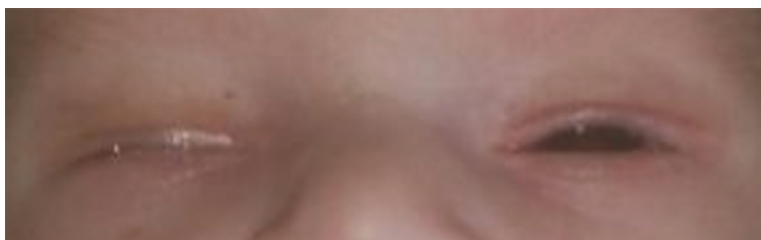
The lab exams revealed normocytic normochromic blood picture, C-reactive protein (CRP)—negative, serum bilirubin—9.8 mg/dL, serum calcium—3.5 mg/dL, and GRBS—88 mg/dL.

The ultrasound revealed prominent ventricles along with a PDA, Grade I germinomatrix hemorrhage noted at bilateral caudothalamic groove, and lenticulostriate calcification seen on bilateral side. The ophthalmologic exam revealed the presence of a cataractous lens; cornea was clear.

The antibodies for cytomegalovirus IgG, toxoplasma gondii IgG, and rubella both IgG and IgM were positive, but HIV and herpes simplex were negative.

Treatment:

1. Warm care
2. O₂ inhalation (sos)

Figure 1: Cataractous lens in a congenital rubella syndrome patient.

3. Inj. Ampicillin 108 mg IV BID
4. Inj. Amikacin 16 mg IV BID
5. IVF 10% Dextrose 32 mL IV QID
6. Inj. Calcium gluconate 2.15 mL IV QID in IVF
7. Inj. Fluconazole 12 mg IV OD
8. Inj. Phenobarbitone 5 mg IV BID
9. EBM with paladai 2nd hourly

3. DISCUSSION

CRS results from a chronic infection of the fetus with rubella virus with progressive damage to various organs. The virus spreads by droplet infection. It leads to a wide array of systemic manifestations ranging from acute multiorgan involvement in neonatal period to deafness and progressive mental retardation in infancy [5].

However, maternal infection can transfer the infection transplacentally and causes congenital defects in the fetus. During the period of maternal viremia, the placenta may become infected. These culminate in placental hypoplasia and placentalitis, which in turn result in viral entry into the fetal circulation by embolic transport [10]. CRS manifestations may be transient such as purpura, permanent structural manifestations (deafness, central nervous system defects, congenital heart disease, cataract), or late-emerging conditions (diabetes mellitus) [11].

Intrauterine growth retardation and prematurity frequently manifest in CRS. The commonest defect is central hearing loss [5]. Characteristic cardiac defects that occur in CRS include PDA, pulmonary stenosis, and ventricular septal defect (VSD). Cardiac lesions are more frequently prevalent in neonates with ocular lesions [2, 5, 12].

Other manifestations include intracranial calcifications, hepatosplenomegaly, rubelliform rash, microcephaly with mental retardation, thyroid abnormalities, and polycystic kidney disease.

Isolating rubella virus from oropharynx and urine and detecting rubella-specific IgM in the cord blood or neonatal blood along with elevated rubella IgG titers over time are diagnostic modalities of CRS [13]. IgM in a fetus indicates intrauterine infection. IgG is the only maternal immunoglobulin that is normally transported across the placenta [14].

There is no specific treatment for congenital rubella. However, preventing CRS by immunizing all susceptible adults plays a paramount role in eliminating or reducing the incidence of this dreadful disease. Conception should be delayed for three months after rubella vaccination to avoid the risk of fetal infection.

4. CONCLUSION

We presented a case report of a newborn with CRS; though uncommon nowadays, it is still present in India. Its rare manifestations include congenital nuclear cataracts and lenticulostriate calcification. Coexistence of many other less frequent systemic manifestations and positive serology makes this case unique. Vaccination of high-risk groups is necessary in order to avoid the appearance of CRS.

References

1. Robert-Gnansia E. Congenital rubella syndrome. Orphanet Encyclopedia, November 2004.
2. P Vijayalakshmi, Rajasundari TA, Prasad NM, Prakash SK, Narendran K, *et al.* Prevalence of eye signs in congenital rubella syndrome in South India: A role for population screening. *Br J Ophthalmol.* 2007; 91:1467-70.
3. Jenkins KJ, Correa A, Feinstein JA, Botto L, Britt AE, *et al.* Non inherited risk factors and congenital cardiovascular defects: Current knowledge: A scientific statement from the American Heart Association Council on Cardiovascular Disease in the Young: Endorsed by the American Academy of Pediatrics. *Circulation.* 2007; 115(23):2995-3014.

4. Robinson JL, Lee BE, Preiksaitis JK, Sabrina Plitt, Grahan A. Tipples. Prevention of congenital rubella syndrome – what makes sense in 200? *Epidemiol Rev.* 2006; 28(1):81-87.
5. Givens KT, Lee DA, Jones T, Ilstrup DM. Congenital rubella syndrome: Ophthalmic manifestations and associated systemic disorders. *Br J Ophthalmol.* 1993; 77(6):358-63.
6. Boger WP III. Late ocular complications in congenital rubella syndrome. *Ophthalmology.* 1980; 87(12):1244-52.
7. Numazaki K, Fujikawa T. Intracranial calcification with congenital rubella syndrome in a mother with serologic immunity. *J Child Neurol.* 2003; 18(4):296-97.
8. De Oliveira SA, Camacho LA, de Medeiros Pereira AC, Bulhoes MM, Aguas AF, *et al.* Performance of rubella suspect case definition: Implications for surveillance. *Rev Saude Publica.* 2006; 40(3):450-56.
9. Deka D. Diagnosis of acute rubella infection during pregnancy. *J Obstet Gynaecol India* 2006; 56(1):44-46.
10. William Webster. Teratogen update: Congenital rubella. *Teratology.* 1998; 58:13-23.
11. Felicity T, *et al.* *Guidelines for surveillance of congenital rubella syndrome and rubella.* Field test version, May 1999. www.who.int/gpv-documents
12. Reddy UN, Chacham S, Rao JN, Priya AV, Mahmood A, *et al.* A rare case of buphthalmos and bilateral nuclear cataracts in a neonate with congenital rubella syndrome. *J Evol Med Dent Sci.* 2014; 3(25):6965-66.
13. World Health Organization. Surveillance guidelines for measles, rubella and congenital rubella syndrome in the WHO European region. 2009: 1-48.
14. Verma IC. Epidemiology, transmission, pathogenesis and laboratory diagnosis of maternal infection, rubella. In: "Congenital Intrauterine TORCH Infections." Editor – Deka D. New Delhi. Jaypee Brothers.

Citation: Gottimukkula DR, Devulapelly NS, Chakinala R, Kagitapu S, Morishetty Y, *et al.* Congenital rubella syndrome—Case report. *Recent Adv Biol Med.* 2016; 2:128-130.