International Journal of Pediatrics and Neonatology
www.pediatricsjournal.net
Online ISSN: 2664-8369, Print ISSN: 2664-8350
Received: 19-10-2021, Accepted: 04-11-2021, Published: 20-11-2021
Volume 3, Issue 1, 2021, Page No. 22-24

Congenital rubella syndrome-A meta-analysis

Shramana Ray
Research Assistant, Aiims, Bhubaneswar, Odisha, India

DOI: https://doi.org/10.33545/26648350.2021.v3.i1a.25

Abstract
Congenital rubella syndrome (CRS) is an illness in infants that result from maternal infection with rubella virus during pregnancy. When rubella infection occurs during early pregnancy serious consequences such as miscarriage, stillbirth, birth defects can occur in infant. Burden of congenital rubella syndrome on the society is increasing and it is still not fully recognized. Surveillance of the cases and routine vaccination can help the people and society to get rid of this disease. In this review study focus is on disease pathology, epidemiology, pathogenesis, clinical manifestations and different strategies to prevent CRS.

Keywords: congenital rubella syndrome, meta-analysis

Introduction
Congenital rubella is a viral infection acquired from mother during pregnancy, characterized by symptoms including congenital heart disease, microcephaly, cataract, growth restriction, hearing loss, hepatosplenomegaly. The foetus is at highest risk of developmental abnormalities when infected during the first 16 weeks of gestation, particularly the first 8-12 weeks. Early in gestation the virus causes endothelial damage to blood vessels. Rubella infection during pregnancy can put the infant at the risk of getting different birth defects and severe complications.

Historical background
Till the year 1941, there was no much attention was given on congenital rubella syndrome and rubella infection. Following a rubella epidemic in 1940, Norman Gregg, an Australian Ophthalmologist had noticed an unusual number of infants with cataracts. Later he established the relationship between maternal rubella infection and infants with symptoms including purpura, hepatitis, thrombocytopenia, bone lesions as manifestation of disease [1]. Gregg's work on rubella syndrome was continued further following research work of different researcher. The occurrence of rubella during first trimester of pregnancy has been associated with significantly increased incidence of congenital malformation, still birth and abortion.
During 1963-1964, a pandemic of rubella swept across US, leaving in its wake at least20,000 affected infants in this country during the epidemic an expanded congenital rubella syndrome was described, involving not only the classic triad of cataract, deafness and cardiac anomalis but also encephalitis, hepatitis, pneumonia [2].
In 1969 the rubella virus was isolated and identified as the specific organism responsible for congenital rubella syndrome.

Pathogenicity
Rubella is a member of the Togaviridae. Rubella virus is a spherical, 40-80 mm, single stranded RNA virus with spike like, hem agglutinin containing surface projections. Rubella virus contains three major structural polypeptides, two membrane glycoproteins, E1 and E2 and a capsid protein.
Humans are the only known reservoir of rubella virus with postnatal person to person transmission occurring via direct or droplet contact with the respiratory secretions of infected person. Additional replication in selected target organs, such as spleen and lymph nodes, leading to a secondary viraemia spread to target organs. 7 days after infection and 7-10days before the onset of rash the virus can be detected. However virus shredding from the respiratory tract may continue for up to 28 days following the onset of rash [3].

Pathogenesis
The disease is transmitted via direct or droplet contact with respiratory secretions. Rubella virus multiplies in cells of the respiratory system, this is followed by viremic spread to target organs.

Epidemiology
Rubella is contagious infections disease caused by rubella virus.
The infection is usually mild sub-clinical. the incubation period for rubella ranges between 14-21 days. Out of 645 suspected CRS patients enrolled during two years 137(21.2%) were classified as laboratory confirmed CRS, 8 (1.2%) as congenital infection. Common clinical features among laboratory confirmed CRS patients included structural heart defect in 108(78.8%), one or more eye signs (cataract, glaucoma pigmentary retinopathy (59.9%) and hearing impairment (38.6%). Thirty three (24.1%) laboratory confirmed CRS patient died over a period of 2 years [4, 5].

Final case classification
Final case classification was based on the presence of group A and group B clinical signed as well as laboratory results. The sign in group A included cataract, glaucoma, Pigmentary retinopathy, congenital heart defect, hearing impairment.
The signs in group B include microcephaly, developmental delay, splenomegaly, purpura, radiolucent bone disease. Based on the clinical signs and laboratory results, suspected

CRS patients were classified into one of the following:

1. **Laboratory confirmed CRS:** infant having at least one sign from group A an meeting one of the following laboratory criteria, detection of IgM antibody or sustained level of rubella IgG antibodies, as determined on at least two occasion at age 6-12 months.
2. **Congenital rubella infection:** infants who meets the laboratory criteria for CRS but does not have any sign from group A
3. **Clinically compatible CRS:** infants who has two clinical signs from group A or one from group A and from group B but from whom adequate specimen could not collected
4. **Discarded case:** a suspected CRS case with adequate specimen not meeting the laboratory confirmed case definition or a suspected case without an adequate laboratory specimen and not meeting the case definition of clinically compatible CRS [6].

Clinical manifestation

The classical triad of CRS are cataract, congenital heart disease and sensor neural hearing loss. As per a global review of CRS among infants, 60% had hearing impairment, 45%congenital heart disease, 27% microcephaly, 25% cataract, 23%low birth weight,19%hepatosplenomegaly, 17%purpura,13%mental retardation and 10% meningoencephalitis.

Cardiovascular

Congenital heart disease is the most commonly associated with intrauterine rubella infection. The incidence of cardiac anomalies found in CRS infant is 65%. The most common cardiovascular lesions are patent ductus areteriosus followed by pulmonary artery stenosis.

Ocular

The most common ocular abnormalities seen in CRS infants are cataract, micropthalmos and pigmentary retinopathy. P Vijaylakashmi described both eyes were affected in 41(89%) patients. Cataract was present in 81 (93.1%) eyes, most of them were nuclear cataract (97.5%). other common ocular presentations include micropthamosin b74 (85.1%) eyes, iris abnormalities in 5(58.6%) eyes and pigmentary retinopathy in 33(37.9%) eyes [6].

Auditory

Hearing impairment is the most commonly associated problem in CRS infants. M M Raahma described rubella antibody was detected in 74% of the hearing impaired children and in 18% of those with normal hearing. The study indicates a strong association between rubella infection and hearing impairment in children. Examination of foetus and neonates after rubella infection showed damage to the epithelium of the cochlear duct and stria vascularis [7].

Neurological

Sequelae of rubella virus infection included there distinct neurological syndromes, a post infectious encephalitis following acute infection, extremely rare neurodegenerative disorder, progressive rubella panencephalitis. Brain damage only occur after rubella infection in the first 16 weeks of gestation, causing mild to severe mental retardation with spastic diplegia.

Serological testing

Maternal screening with rubella titre in early pregnancy is considered standard care. Laboratory diagnosis is based on use of IgM and IgG titres. Prenatal fetal diagnosis is based on detection of viral genome in amniotic fluid, foetal blood or chorionic villus biopsies. Prenatal diagnosis is done by detecting RV IgG antibodies in neonatal serum using ELISA. Confirmation of infection is made by detection of rubella virus in nasopharyngeal swabs, urine and PCR. Congenital infection can also be confirmed by stable or increasing serum concentration of rubella-specific IgG over the first year of life [8].

Treatment

Prenatal management of the mother and foetus depends on gestational age at onset of infection. If infection happens before 18 weeks gestation, the foetus is at high risk for infection and severe symptoms. Detailed ultrasound examination and assessment of viral RNA in amniotic fluid is recommended. For infection after 18 weeks of gestation, pregnancy could be continued with ultrasound monitoring followed by neonatal physical examination and testing for RV-IgG [9].

There is no specific antiviral treatment available for rubella, only supportive management is available for infants with rubella infection. Early diagnosis can prevent the impairment to certain extent.

Surveillance

During 2000-2016, the number of countries reporting rubella cases increased 42% from 102 in 2000t to 176 in 2012, but the number of reporting countries declined 6%, to 165 in 2016. The number of countries reporting countries CRS cases increased 42% from 2000 to 202, then decreased 4% to 125 countries in 2016. In 2016, 22361 rubella cases were reported to WHO, a 97% decrease from 670894 case reported in 2000, and a 76% decrease from 94277 cases reported in 2012 [10].

Recommendation

CRS is a preventable disease, so early detection of CRS can reduce CRS related mortality and morbidity. A team approach containing neonatologist, paediatrician, ophthalmologist, cardiologist is needed to identify the early symptoms of CRS. Women in pregnancy should be monitored for rubella infection and infants should be monitored for IgM antibody test after birth.

Conclusion

Rubella is a major public health problem characterized by mild rash, arthralgia, low grade fever. When rubella virus crosses the placental barrier and infects infant with congenital rubella syndrome. There is no permanent solution to recovery from CRS but early detection and prompt vaccination, surveillance of problem and preventive strategies can prevent congenital rubella infection.

References

1. Felicity T Cutts, Susan E Robertson. Control of rubella and congenital rubella syndrome in developing countries;Bulletin of the World Health Organization, 1997, 75(1).
2. Cooper LZ. The history and medical consequences of rubella. Reviews of infectious diseases,1985:7(1):S2-S1

0.
3. Orenstein WA *et al*. Methods of assessing the impact of congenital rubella infection. Reviews of infectious diseases,1985:7(suppl. 1):S22-S28.
4. Swartz TA *et al*. Early results of an extensive rubella epidemic. International journal of epidemiology,1975:4:331-335.
5. Sachdeva R. Congenital rubella syndrome at Mombasa. East African medical joumal,1973:50(3):146-152.
6. Ali ZA, Hull B, Lewis M. Neonatal manifestation of congenital rubella following an outbreak in Trinidad. Journal of tropical pediatrics,1986:32:79-82.
7. Baxter DN. Control of the congenital rubella syndrome in Jamaica. West Indian medical journal,1986:35:50-54.
8. Menser MA *et al*. Epidemiology of congenital rubella and results of rubella vaccination in Australia. Reviews of infectious diseases,1985:7(suppl. 1):S37- S41. 37.
9. Maselle SY, Haukenes G, Rutahindurwa A. Preliminary observations on rubella infection in Tanzania and the challenge for its control. East African medical journal,1988:65(5):319-324.
10. Strauss J *et al*. Serological survey of rubella in Yemen in 1985. Journal of hygiene, epidemiology, microbiology and immunology,1989:33:163-167.