



RESEARCH ARTICLE

EFFECT OF RUBELLA INFECTION DURING PREGNANCY IN MARRIED WOMEN

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ARTICLE INFO

Article History:

Received 18th February, 2016
Received in revised form
01st March, 2016
Accepted 29th April, 2016
Published online 31st May, 2016

ABSTRACT

Rubella infection is normally of minor impact characterized by a mild, self-limited disease associated with a characteristic rash, but during pregnancy due to maternal infection many complications were seen in mother as well as in fetus as rubivirus has capability to pass through placenta and can affect organogenesis which cause congenital rubella syndrome (CRS). In study women suffering from Infertility (primary and secondary) show highest percentage of infection i.e. 50% of them are found to be susceptible.

Key words:

Organogenesis, Infertility and Susceptible.

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Citation: Dr. Chetna Savita, 2016. "Effect of rubella infection during pregnancy in married women", *International Journal of Current Research*, 8, (05), 31352-31353.

INTRODUCTION

During pregnancy, the virus can have potentially devastating effects on the developing fetus. It has been directly responsible for inestimable wastage and for severe congenital malformations. Rubella disease generally has two symptoms, primary or mild effect and secondary, i.e. CRS (Congenital Rubella Syndrome). While in about 50% of the cases the infection is silent, but the individual still has the potential to transmit the disease. Generally, the disease manifests itself with mild symptoms such as fever, rashes, respiratory disorder, joint pain, swollen glands, headache, conjunctivitis etc., which rarely causes complication in some cases such as arthritis or encephalitis. Secondary effect (Plotkin, 2001) of the disease is the disastrous one which follows the intrauterine infection by the Rubella virus and comprises of malformation and complication in the fetus and also shows a bad obstetric history (BOH), repeated pregnancy loss (RPL) in women whereas it is also responsible for maternal mortality.

Bad obstetric history (BOH) implies previous unfavorable fetal outcome in terms of two or more consecutive spontaneous abortions, early neonatal deaths, stillbirths, intrauterine fetal deaths, intrauterine growth retardations and congenital anomalies. Cause of BOH may be genetic, hormonal, abnormal maternal immune response and maternal infection.

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Maternal infections transmissible in uterus at various stages of gestation lead to recurrent pregnancy wastage.

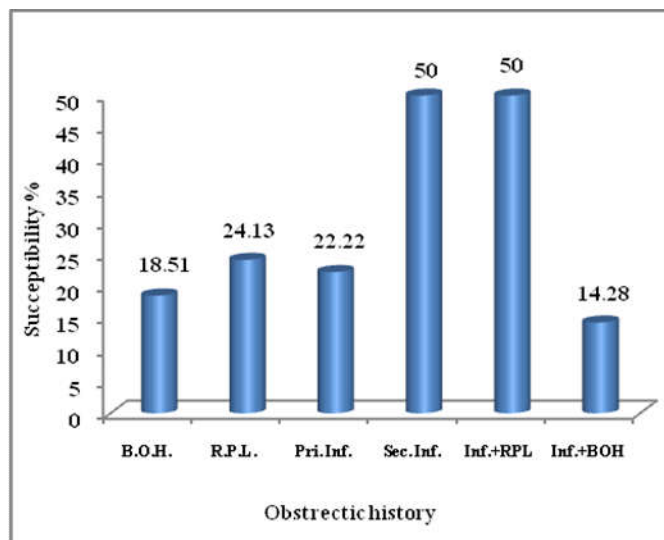
Recurrent Pregnancy Loss, when labor, resulting in live birth before the 37th week of pregnancy is termed as premature birth, even if the infant dies shortly afterward. The limit of viability at which 50% of fetuses survive long-term is around 24 weeks; with moderate or major neurological disability dropping to 50% only by 26 weeks is described by Banatvala et al. (1974).

Infertility primarily refers to the biological inability of a person to contribute to conception. Infertility may also refer to the state of a woman who is unable to carry a pregnancy to full term. There are many biological causes of infertility, some which may be bypassed with medical intervention. Infertility is defined as failure to conceive after one year of regular unprotected sexual relationship (WHO, 1998).

OBSEVATION AND RESULTS

Susceptibility of married women, according to their obstetric history

S. No.	Obstetric history	N	Susceptibility	%
1	BOH	54	10	18.52
2	R.P.L	58	14	24.14
3	Pri. Inf.	54	12	22.22
4	Sec. Inf.	16	8	50.00
5	Inf. + R.P.L	4	2	50.00
6	Inf. + BOH	14	2	14.29



DISCUSSION

All married women were classified into 6 classes according to their obstetric history, i. e. bad obstetric history (BOH), repeated pregnancy loss (RPL), primary infertility (Pri. Inf.), secondary infertility (Sec. Inf.), infertility with RPL and infertility with a BOH maximum percentage of susceptibility (50%) was found in women suffering from primary and secondary infertility while minimum susceptibility was found in women suffering from Infertility with BOH that is 14.29%. The similar susceptibility rate among pregnant women from Chandigarh (Pal *et al.*, 1974) and Delhi (Seth *et al.* 1972), were much lower at 19% and 12.7%, respectively whereas in present study susceptibility rate is very high i.e. 27.16%. Similar rubella susceptibility in women was seen in the study of Mathur *et al.* (1982), a hospital based study from Lucknow; out of 300 women nearly 21% susceptibility was recorded and Shanmugam *et al.* 1982 found susceptibility 25.1% for Rubella in 526 pregnant women. Robertson *et al.* (2003) stated that minimum 100,000 cases of CRS occur worldwide and about 9.8% of children in India were born with this complication. In 2004, Atreya discovers the specific functioning pathway of Rubella virus causing CRS in the fetus, which results in severe organogenesis. Singla *et al.* (2004) stated that serologically, immune status shows, poor correlation with history of past Rubella virus infection in Amritsar (Punjab). Centers for Disease Control and Prevention (2004) convened an independent panel of internationally recognized authorities on public health, infectious disease, and immunization to assess progress toward elimination of rubella and congenital rubella syndrome in the United States, a national health objective for 2010. Since rubella vaccine licensure in 1969, substantial decline in rubella and CRS have occurred, and absence of endemic transmission in the United States is supported by recent data: fewer than 25 reported rubella cases each year since 2001, at least 95% vaccination coverage among school aged children, estimated 91% population immunity, adequate

surveillance to detect rubella outbreaks and a pattern of virus genotypes consistent with virus originating in other parts of the world.

Conclusion

Generally the incubation period for rubella is 12 to 23 days. The infectious period is from 7 days before to 5–7 days after rash onset. Although rubella is asymptomatic in 25% to 50% of cases, some individuals may experience mild symptoms such as low-grade fever, conjunctivitis, sore throat, headaches or malaise, and tender lymphadenopathy but in pregnancy it shows many complications. Clinical diagnosis is unreliable because a large proportion of cases are subclinical and because clinical features can be very similar to those of other illnesses. If a pregnant woman develops signs or symptoms of a rubella-like illness or has recently been exposed to rubella, gestational age should be determined as well as her state of immunity.

REFERENCES

- Atreya CD., Mohan KV. And Kulkarni S. 2004. Rubella virus and birth defects: molecular insights into the viral teratogenesis at the cellular level. *Birth Defects Res. Part A Clin. Mol. Teratol.*, 70 (7): 431–7..
- Banatvala JE., Best JM 1984. Rubella. In: Principles of bacteriology, virology and immunity. Vol 4, GS Wilsonm, AA Miles, MT Parker. Eds. (Edward Arnold, London): p271.
- CDC 2004. Control and Prevention of Rubella. Evaluation and management of suspected outbreaks: Rubella in pregnant women, and Surveillance for Congenital Rubella Syndrome. Morbidity and mortality weekly report (MMWR).CDC 2004. Rubella Overview: Immunization Information. p. 131.
- Mathur A., Tripathi R., Chaturvedi UC. and Mehra P. 1982. Congenital rubella following inapparent rubella infection. *Indian J Med Res.*, 75:469-73.
- Pal SR, Chitkara NL., Broor S., Murthy JG., Choudhary S., and Devi PK. 1974. Serological investigation of Rubella virus infection in and around Chandigarh – A preliminary communication. *Indian J Med Res.*, 62:204-45.
- Plotkin SA. 2001. Rubella eradication. *Vaccine* 19 (25-26): 3311–9.
- Robertson SE., Featherstone DA. and Gacic-Dobo M. 2003. Rubella and congenital rubella syndrome: global update. *Pan Am J Pub Health*, 14(5): 306-15.
- Seth P., Balaya S. and Mohapatra LN. 1972. Rubella antibody in pregnant women. *Indian J Pathol Bacteriol.*, 15:23-6.
- Shanmugam J., Raveendranath M. and Nair VR. 1982. Seroprevalence of rubella and cytomegalovirus (CMV) infection in pregnant women from Kerala State. *J Indian Assoc Commun Dis.*, 5:58-63.
- Singla N., Jindal N. and Aggarwal A. 2004. The seroepidemiology of rubella in Amritsar (Punjab). *Indian J Med Microbiol.*, 22:61-3.
