ORIGINAL ARTICLE

VAGINAL CARRIAGE RATE OF GROUP B STREPTOCOCCUS IN PREGNANT WOMEN AND ITS TRANSMISSION TO NEONATES

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Background: Maternal vaginal colonisation with Group B Streptococcus (GBS, *Streptococcus agalactiae*) at the time of delivery can cause vertical transmission to the neonate. GBS is the leading cause of sepsis, meningitis and pneumonia in the infants. Asymptomatic colonisation of the vagina with GBS varies with the geographical location. **Methods:** This was a cross-sectional study conducted in 2009 at Benazir Bhutto Hospital, Rawalpindi, Pakistan. Lower vaginal swabs were obtained from 200 pregnant women at the time of admission in the Gynaecology and Obstetrics Department for term, normal vaginal delivery and swabs from the skin of abdomen and ear canals of their respective neonates immediately after delivery were collected. Swabs were inoculated on blood agar and incubated aerobically and on Group B Streptococcus agar (GBS agar) and incubated anaerobically in an anaerobic jar. Identification of GBS was made on the basis of colonial morphology (β-haemolytic colonies on blood agar and orange pigmented colonies on GBS agar), Gram stain, catalase test and conformation was done by means of latex agglutination tests. **Results:** A GBS carriage rate of 8.5% among pregnant women before delivery and an acquisition rate of 53% on the abdominal skin and 18% in the ear canals by the neonates of colonised mothers were found. **Conclusions:** GBS colonisation in pregnant women and its transmission to the neonates is present in our population so GBS infections in the prenatal and neonatal period might not be uncommon in Pakistan, so routine screening should be carried out.

Keywords: Group B Streptococcus, vaginal carriage rate, pregnant women, transmission to neonates

INTRODUCTION

Group B Streptococcus (GBS) is a cause of cystitis, amnionitis, endometritis, and stillbirth in the pregnant women.[1] A considerable percentage of the GBS colonised neonates (1–3%) suffer invasive early-onset group B Streptococcus disease (EOGBSD).[2] Early-onset GBS infection can present as neonatal septicaemia, pneumonia or meningitis, which are associated with high mortality.[3] The infants who survive are often left with developmental disabilities, including mental retardation, hearing or vision loss[4] and speech problems.[5,6]

The rate of GBS colonisation in pregnant females varies from 5–30%.[7] The rates of maternal and neonatal GBS carriage resulting in early onset neonatal disease may vary in different communities, so it should be thoroughly evaluated in every country, thus allowing appropriate preventive strategy to be selected.[8]

The prevalence of GBS vaginal colonisation in pregnant women in different studies was as follows; India/Pakistan 12%, America 14%, Asia-Pacific 19%, Sub-Saharan Africa 19%, Middle-East/North Africa 22%.[9] Islam medium can be used to detect GBS in mixed cultures and this is especially useful for women in labour.[10]

The most likely reservoir of GBS is the gastrointestinal tract, and the most frequent site of secondary spread is the genitourinary tract. The neonates get colonised with GBS by the aspiration of infected amniotic fluid[11], or by vertical transmission during the passage through the colonised vaginal canal.[12] The GBS carriage rate is 40–70% in neonates who are born to the colonised mothers.[13] The most important risk factor for early-onset neonatal disease is maternal GBS colonisation at time of delivery.[14,15] Sepsis, low birth weight and asphyxia are the immediate predisposing factors to neonatal bacterial infections.[16]

It is recommended by Centers for Disease Control and Prevention (CDC) that all pregnant women at 35–37 weeks should have prenatal screening for GBS colonisation of the vagina and rectum which is based on the results of culture-based screening strategy relative to the risk-based strategy.[17] The successful implementation of screening recommendations is likely to have contributed to the documented 27% decrease in the incidence of EOGBSD from 1999–2001 to 2003–2005.[18] Implementation of prevention programs can decrease the morbidity and mortality resulting from GBS disease and it is more cost-effective to prevent GBS infection in the neonates than to treat GBS infections.[19]

There is need of data on the incidence of GBS in neonates, preventive measures and the outcome of infected neonates.[20] The data from different areas of Pakistan is very limited and is generally of the pregnant women and negligible work has been done on the prevalence of GBS in the neonates. This study aimed at finding out the prevalence of GBS in both the mothers and their neonates.

METHODOLOGY

This cross-sectional study was conducted in 2009 in the Department of Microbiology and Department of Gynaecology and Obstetrics, Benazir Bhutto Hospital,

Rawalpindi. Random samples of pregnant women fulfilling the specified selection criteria at the time of delivery were included and their respective neonates were included as their pair study units.

Pregnant women between 20–40 years of age at the time of admission in the hospital for term, normal vaginal delivery were included in this study. All neonates of respective included mothers were also included. Pregnant females with systemic diseases like pregnancy-induced hypertension/hypertension, diabetes mellitus, chronic infectious diseases, patients on antibiotics, and those with obstetrical problems like placenta previa, preterm delivery (less than 37 completed weeks of gestation), prolonged rupture of membranes (an interval between rupture of membranes and delivery of 18 hours or longer before the baby is born)[21] were excluded from the study.

The culture specimens from the lower vagina of 200 pregnant women prior to any management, at the time of admission in the hospital for normal, term vaginal delivery were collected without a speculum using sterilised disposable cotton swabs. Swabs were also collected from the abdominal skin and ear canals of the neonates born to these mothers immediately after delivery. The swabs were placed in Amies agar gel medium and transported to the Microbiology Laboratory within 24 hours.

Swabs were inoculated on Blood Agar and incubated aerobically for 24 hours at 37 °C and on Group B Streptococcus agar (Islam medium) and incubated anaerobically at 37 °C for 24–48 hours in an anaerobic jar using AnaeroGen sachets. *Pseudomonas aeruginosa* and *Bacteroides fragilis* were used as controls for checking the efficacy of the anaerobic jar. GBS was identified using colonial morphology (presence of β-haemolytic colonies on Blood agar and orange pigmented colonies on GBS agar), Gram stain, catalase test and was confirmed by means of latex agglutination tests (Omega's Avipath Strep).

RESULTS

Out of a total of 200 vaginal samples of pregnant women, 17 (8.5%) specimens were found positive for GBS. All specimens of mothers found to be positive by culture (n=17) were also positive by GBS antigen detection (Table-1).

In case of the neonates samples from the abdominal skin, 9 (52.9% of the GBS positive mothers) tested positive, while in the case of the samples from the ear canals of the neonates 3 (17.6% of the GBS positive mothers) were positive (Table-2).

Table-1: Status of GBS in vaginal samples of mothers

Category	Frequency	Percentage
Positive	17	8.5
Negative	183	91.5
Total	**200**	**100.0**

Table-2: Status of GBS in neonates according to site of sample collection (n=200)

GBS positivity in infants		GBS positivity in mothers		Total	χ^2 (*p*-value)
		Yes	No		
Abdominal skin	**Yes**	9 (52.9%)	0 (0.0%)	9 (4.5%)	101.447* (0.000)
	No	8 (47.1%)	183 (100%)	191 (95.5%)	
Ear canal	**Yes**	3 (17.6%)	0 (0.0%)	3 (1.5%)	32.786* (0.000)
	No	14 (82.4%)	183 (100%)	197 (98.5%)	

*Significant

DISCUSSION

Group B Streptococcus, though known for decades, only emerged as a major perinatal pathogen in the 1970s. It is the leading cause of early onset neonatal infection in North America, Australia, in almost all developed countries, and is an escalating problem in developing countries, as they become more industrialised.[22] In spite of the great accomplishments in decreasing the mortality rate; GBS remains the leading cause of infant morbidity and mortality in the United States of America.[23]

In the present study the lower vaginal specimens were collected. Rectal samples were refused by majority of the pregnant women. The most advantageous method for GBS screening is collection of a single ordinary culture swab or two separate swabs of the distal vagina (without speculum examination) and anorectum.[24]

In our study all isolated GBS were β-haemolytic and pigment producing and group B Lancefield positive by serological testing. In Czech Republic in 2001, a study pointed out that both GBS agar and GBS broth are reliable methods, when compared to the recommended method and can be used to screen for GBS colonisation in the pregnant women and their neonates.[25]

We found a GBS carriage rate of 8.5% in the mothers which is considerable. The rate of GBS colonisation in pregnant females varies from 5 to 30% with different geographical distribution.[7] The colonisation rate was 53% in the neonates of the colonised mothers, which is reasonably noteworthy. The risk of a neonate to be colonised at birth is directly related to the intensity of maternal colonisation.[26]

The GBS colonisation rate might have been higher in the neonates of our study. Typically the neonates are colonised only briefly after rupture of membranes. Thus, their bacterial load is likely to be lower and isolation by culture more difficult.[2]

A study in Peshawar in 1984 reported a GBS carriage rate of 30.9% amongst the pregnant women.[27] Another study was carried out in Lahore in 1997 in which two hundred pregnant women in the third trimester were screened. GBS was found in 4.5% of the pregnant women.[28] The results of these two studies are quite different from our study. The differences in the vaginal carriage rates of GBS depend on the time of

gestation at which specimens were obtained [27], particular population and specially on the laboratory methods used to detect GBS.[29]

Some studies carried out in India, document vaginal colonisation rates of GBS between 5%–16 %.[30] GBS colonisation rate among term pregnant women in Saudi Arabia is relatively high (27.6%); and thus constitutes a group of women whose neonates are at great risk of early-onset invasive disease.[31] The carriage rate in this study was quite high as compared to our study, in spite of the fact that Islam medium was used in both studies. The difference might be because of the difference in the timing of collection of the specimens and the geographical difference.[27]

A study conducted in Iran in 2003 identified a 9.1% GBS carriage rate in recto-vaginal samples of pregnant women, with a 60% transmission rate to their neonates.[32] GBS was isolated in 8.7% of the pregnant women in a study in Turkey in which rectal, vaginal and cervical swabs were taken from 114 women.[33] The results of these studies are very close to our study, in spite of the fact that we screened the pregnant women culturing only the vaginal samples.

In view of the fact that maternal GBS colonisation at delivery is the most important risk factor for neonatal disease, microbiological techniques must be designed in order to maximize detection rates.[34]

The prevention of GBS transmission from mother to infant is required before the birth of the neonate.[35] So we should carry out more multi-centre studies on GBS colonisation in pregnant women and its transmission to their neonates in different parts of our country and if the results are significant then guidelines should be formulated to prevent the transmission of GBS from the mothers to their neonates. Nationwide epidemiological data on neonatal GBS disease should also be collected.

Increasing data suggests that treating GBS-infected neonates is more expensive than preventing the infection and that properly implemented prevention programs can significantly decrease illness and death resulting from GBS disease.[5] It has been estimated by the CDC that $300 million dollars were spent in a year to treat almost 7,500 cases of EOGBSD.[29] Attention should be focused on prevention of GBS infection in neonates which can only be possible by identification and treatment of carrier mothers, so that potential lethal consequences can be prevented.

CONCLUSION

GBS colonisation in pregnant women and its transmission to the neonates is present in our population. More specific national epidemiological data on the incidence, morbidity, and mortality of neonatal EOGBS infection are required.

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