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PREVENTION OF GROUP B STREPTOCOCCAL INFECTIONS: A REVIEW OF LITERATURE

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ABSTRACT

Group B Streptococcus (GBS) is an important cause of maternal and neonatal morbidity and mortality in many parts of the world. Intra-partum use of antibiotics in these women has led to a decrease in the rate of early onset but not late onset GBS disease.dentification of women with GBS is the key factor in the prevention of perinatal GBS disease. Asymptomatic colonisation of the vagina and rectum with Group B streptococci is common in pregnancy. Maternal colonisation of GBS can vary depending on ethnicity and geographical distribution. Vertical transmission of this organism from mother to foetus may lead to neonatal GBS disease. There are different screening strategies available to identify women at risk of perinatal GBS disease. Clinicians continue to face the challenge of choosing between preventive strategies to reduce the impact of perinatal GBS disease. Controversy exists regarding the ideal preventive strategy. In India, the mortality and morbidity associated with the GBS disease remains largely a under-recognised problem. This comprehensive review summarises the salient features of GBS disease and discusses the epidemiology, risk factors, screening strategies, intra-partum antibiotic prophylaxis

Introduction

Group B Streptococcus (GBS) is an important cause of maternal and neonatal mortality and morbidity in many parts of the world. In the 1970s GBS emerged as the leading infectious cause of early neonatal morbidity and mortality in the Western world. [1] The 1990s saw the widespread adoption of antibiotic prophylaxis during labour in many Western industrialised countries. [2] The recognition that maternal colonisation with the organism is the key factor in the occurrence of GBS-related neonatal morbidity and mortality is the basis for the preventive strategies. Intensified efforts were made to prevent this devastating infection by identifying and treating pregnant women who carry GBS or who are at highest risk of transmitting the organism to a newborn

GBS Infection

GBS or Streptococcus agalactiae is a Gram-positive bacterium that causes invasive disease primarily in infants, pregnant or postpartum women. Infections in newborn occurring within the first week of life are designated early onset group B streptococcal (EOGBS) disease. Late onset disease develops in infants after 7 days and up to 3 months of age. The measures used to prevent EOGBS disease, however, do not prevent late onset GBS disease. [1]

Early Onset GBS Disease

Infants with early onset GBS disease generally present with respiratory distress, apnoea or other signs of sepsis within the first 24-48 hours of life. [1],[4] The most common clinical manifestation of EOGBS disease are sepsis and pneumonia; less frequently meningitis. Mortality is higher among preterm infants compared with full term-infants. [1],[5], [6] Early onset infections are acquired vertically through exposure to GBS from the vagina of a colonised woman. Neonatal infection occurs primarily when GBS ascends from the vagina to the amniotic fluid after onset of labour or rupture of membranes, although GBS also can invade through intact membranes. [1],

Late Onset GBS Disease

Late onset disease (7-90 days) occurs less frequently than EOGBS. Maternal obstetric complications are uncommon with late onset GBS disease. Transmission can be either horizontal (from other infected infants or healthcare workers) or vertical (from mother due to close proximity). [3] The two most common clinical manifestations of late onset disease are meningitis and bacteraemia. The initial signs usually are fever, lethargy, irritability, poor feeding and tachypnoea. [3] The mortality rate for late onset neonatal disease is 2-6%, which is significantly lower than the rate of 10% for early onset infections.

Epidemiology and Burden of Disease

GBS is the leading infectious cause of morbidity and mortality among infants in the Western hemisphere. In the United States, 10-35% of pregnant women are asymptomatic carriers of GBS in the genital and gastrointestinal tract at time of delivery. [3] At birth, 50-65% of infants who are born to colonised mothers have positive GBS cultures from mucus membranes and skin (external ear canal, throat, umbilicus and anorectal sites). [3] Approximately 98% of colonized newborns remain healthy, but 1-2% develop invasive disease. [3] In the United States, the overall incidence of neonatal GBS infection was 1.7 cases per 1000 live births prior to the introduction of intra-partum prophylaxis. As a result of preventive efforts, incidence of GBS has declined dramatically to 0.34-0.37 cases per 1000 live births in the recent years, 2006-2008. [1]

Risk Factors for EOGBS Disease

Maternal intra-partum GBS colonisation is the primary risk factor for early onset disease in infants. In the USA approximately 10-30% of pregnant women are colonised with GBS in the vagina or rectum. [1]

In the absence of any intervention, an estimated 1-2% of infants born to colonised mothers develop EOGBS infection.[1] GBS colonisation during pregnancy can be transient, intermittent or persistent. [5]

Although some women with GBS colonisation during a pregnancy will be colonised during subsequent pregnancies, a substantial proportion will not. [2] The gastrointestinal tract serves as the primary reservoir for GBS and is the likely source of vaginal colonisation. Heavy colonisation, defined as culture of GBS from direct plating rather than from selective broth only, is associated with higher risk of early onset disease. [2],[3]

Table 1: Risk factors for early onset group B streptococcal disease; from Oddie and Embleton [6]

Factor	Odds ratio	95% confidence interval
Preterm birth <37 weeks	10.4	3.9-27.6
Preterm birth <34 weeks	33.6	4.0-283.3
Rupture of membranes >18 hours	25.8	10.2-64.8
Preterm prolonged rupture of membranes	30.3	6.3-144.5
Intra-partum fever >38°C	10.0	2.4-40.8
Any antenatal maternal culture of GBS	17.7	1.9-163.5
Previously affected child	Presently unquantified	

GBS: Group B Streptococcus

Risk factor-based screening

Risk factors for EOGBS are well established and have been confirmed in case-control studies; [Table 1]. The risk-based strategy requires women with any one of these risk factors to be given IAP. This strategy could deliver a clear reduction in neonatal morbidity and mortality. Mathematical modelling in the USA suggests that this approach will result in approximately 25% of women being offered IAP with a decrease in the incidence of EOGBS disease of 50-68.8%. [3,4] Therefore, even rigorous application of the risk-based strategy cannot reduce the incidence of EOGBS by more than 50-70%. While this approach has many advocates 30-40% of babies with EOGBS are born to women with no identifiable risk factors.

The Future

Rapid detection of GBS colonization Maternal GBS colonisation may be of transient or intermittent pattern and therefore a test to detect GBS colonization during the intra-partum period could be more advantageous compared with the earlier antenatal screening test. This would more accurately reflect the GBS colonisation status of the woman in labour. [1] Ideally, the use of a highly sensitive and specific test with rapid turnaround time can assess intra-partum GBS colonisation and hence guide IAP.Studies have shown that the rapid tests including florescence in situ hybridisation, latex agglutination test, optical immunoassays and enzyme immunoassays were not sensitive and specific enough to replace the established culture method. Recently, molecular testing methods have been developed, including DNA probes and nucleic acid amplification tests (NAAT) such as polymerase chain reaction (PCR). However, these assays are not currently available commercially, and therefore their benefit remains to be determined. [3] GBS vaccines Maternal immunisation against GBS prior to or during pregnancy has the potential to prevent foetal colonisation and thereby eliminating EOGBS disease. [3]

Conclusion

The IMR in India is decreasing over the past few years and is currently 47 deaths per 1000 live births. [6] The most common cause of neonatal deaths was perinatal asphyxia. Other major causes include septicaemia/meningitis, extreme prematurity and congenital malformations and the most frequent causes of neonatal sepsis are Klebsiella pneumoniae, Staphylococcus aureus and Escherichia coli. [2] It is possible that the role of GBS as a cause of neonatal sepsis has been underestimated in India. It could be due to lack of appropriate screening strategies. Detection of early onset infections may be obscured by the large proportion of deliveries that take place outside health centres. Even for infants born in hospital, bacteriological procedures are not routine in many parts of India. [3] Continued surveillance and more detailed studies are essential in the understanding of the epidemiology and spectrum of disease caused by the GBS. Whether to implement universal screening strategy or clinical risk-based strategy depends on the local incidence of EOGBS disease, the prevalence of clinical risk factors in EOGBS disease and the current obstetric practice.

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