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Antenatal prevention of neonatal group B streptococcal infection

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Abstract

Group B streptococci can be isolated from the vagina of 15–40% of pregnant women. Vertical transmission to the infant occurs in 50% of deliveries involving colonised women. Most infants remain asymptomatic, but 1–2% develop clinical infection, which is associated with significant morbidity and mortality. Vertical transmission can be successfully prevented by intrapartum administration of antibiotics. Other proposed methods include vaccines and intrapartum vaginal or neonatal washing with antiseptics.

Selection of women for prophylactic antibiotics can be based on risk factors, screening or a combination of both. Benefits of prophylaxis should be balanced against cost, medicalisation of labour and the risks of anaphylaxis and bacterial resistance.

We present an overview of vaginal group B streptococcal isolation methods and antenatal strategies for prevention of neonatal infection.

Keywords: Group B streptococcus; *Streptococcus agalactiae*; Screening; Antibiotic prophylaxis; Pregnancy complications (infectious); Vaccines

1. Introduction

Maternal genital tract colonisation with *Streptococcus agalactiae* or Group B streptococci (GBS) is known to be associated with neonatal colonisation, and neonatal and maternal puerperal infections. GBS may contribute to intrauterine infection leading to stillbirth, neonatal pneumonia, meningitis and septicaemia. It has also been implicated in mid trimester miscarriage and premature rupture of membranes and is frequently associated with maternal postnatal endometritis, wound infection, asymptomatic bacteriuria and urinary tract infections [1].

Perinatal GBS infections were first described in the 1960s. By the 1970s GBS were the leading cause of neonatal infection and one of the most important causes of maternal endometritis and septicaemia. Initially neonatal GBS infection was associated with 20–50% mortality. Morbidity and mortality rates have both improved with advances in detection, prophylaxis and treatment [2].

In the early 1980s clinical trials demonstrated vertical GBS transmission could be interrupted by the use of prophylactic intrapartum antibiotics (IAP) [3]. There is considerable controversy as to the best method of selection of parturient women for prophylaxis. The Royal College of Obstetricians and Gynaecologists (RCOG) has decided to opt for risk-based management rather than the universal screening currently advocated by the US Centre for Disease Control (CDC) [4,5].

This review examines the epidemiology of this organism, methods of identification and antenatal strategies for the prevention of neonatal infection.

2. Data selection

We performed Medline, Embase and Cochrane database searches using the following keywords Group B streptococcus; *S. agalactiae*; prophylaxis; treatment; diagnosis; isolation; rapid testing; risk factors; screening; molecular biology; vaccines; polymerase chain reaction; latex agglutination; antigen detection; serotyping. We also looked at GBS guidelines produced by the RCOG, CDC and the Canadian Taskforce on Preventative Healthcare (CTFPHC) [4–6].

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3. Definition of used terms

Colonisation is defined as the presence of a potential pathogen, e.g. GBS, that can be cultured without causing symptoms or disease. Infection is defined as the presence of clinical symptoms or illness due to a microorganism. Neonatal early-onset GBS infection is defined as GBS infection occurring within the first 7 days of life. In this review, transmission is describes the transmission of a potential pathogenic organism from one individual to another. Vertical transmission describes the transmission of such an organism from mother to fetus. Sepsis is a syndrome characterised by signs of infection and accompanied by septicaemia.

4. Colonisation and transmission

It has been estimated that GBS can be cultured from the vagina of 15–40% of pregnant and postpartum women [1,7]. The gastro-intestinal tract acts as a reservoir for GBS in both men and women [6]. There is vertical transmission to the neonate in approximately 50% of pregnancies where the mother's vagina is colonised [8]. Transmission is more likely if there is heavy maternal colonisation, maternal GBS bacteriuria, ruptured membranes, pre-term delivery or intrapartum pyrexia [9]. Nosocomial transmission between neonates may occasionally occur [2].

5. Infection

5.1. Neonatal infection

Most neonates carrying GBS remain asymptomatic but in 1–2% of cases the neonate develops life-threatening infection with a risk of major long-term morbidity in survivors [1]. The most recent research estimates this to occur in 0.7/1000 live births within the UK [10]. The incidence varies geographically.

GBS early-onset infection (EOI) occurs within the first 7 days of life, but most cases are evident within 72 h [2]. It is most commonly manifest as pneumonia or meningitis or has an unidentifiable focus of infection [1]. In the UK it is usually associated with serotypes 3 (38%), 1a (32%), and 5 (13%) [11]. Overall, the UK and US have similar incidences of 0.5/1000 live births [4,10]. The incidence within the UK varies from 0.21/1000 live births in Scotland to 0.73/1000 live births in Northern Ireland [10].

Late onset infection, associated predominantly with serotype 3, may occur at any time after the first week, even months later [10]. Cases are evenly distributed throughout the remaining first 90 days of life and generally present with either bacteraemia or meningitis. Less frequently, it may cause focal infections such as osteomyelitis, septic arthritis or cellulitis [1,2]. It is not always associated with maternal vaginal colonisation and may represent horizontal transmission from hospital or community sources [1,12]. The risk of nosocomial transmission is increased with cramped nursery conditions, poor hand hygiene, prolonged hospital stay and high maternal population colonisation rates [2].

Mortality continues to decline due to neonatal care advances. It is greater in EOI (6% in term and 18% in preterm babies in the UK) [5].

5.2. Maternal infection

GBS is identified in 15% chorioamnionitis, 16% endometritis, 2–15% wound infections, 15% bacteraemia, 9–15% stillbirth and a 2–4% of maternal urinary tract infections [2,4]. GBS often co-exists with other bacteria in chorioamnionitis and endometritis [2]. Most maternal GBS infections, including bacteraemia, respond quickly to antibiotics, but bacteraemia is occasionally associated with abdominal abscess, necrotising fasciitis and meningitis [1].

6. Microbiological techniques

GBS is a Gram-positive facultative coccus. Approximately 99% of GBS show β-haemolysis on blood agar plates [2].

6.1. Isolation

Isolation rates depend on the clinical and laboratory techniques used. They are improved when more than one appropriate site is swabbed (lower vagina, peri-urethral or ano-rectal areas). Combined antenatal vaginal and rectal swabs have a greater positive predictive value for intrapartum vaginal colonisation. Boyer et al. found a 17% vertical transmission rate when the rectal culture was positive but the vaginal culture negative [13].

The use of broth and antibiotic-containing media produce better isolation rates than non-selective media. One example of such is Todd-Hewitt broth developed for Gram-positive organisms, supplemented with nalidixic acid and either gentamicin or colistin (Lim Broth). In Ferreri and Blair's study, GBS were isolated in 37% of women using antibiotic-containing broth but not agar [14]. However, all traditional culture techniques take at least 36 h, which means that the results of an intrapartum swab will usually be delayed until after delivery.

The CDC defines heavy GBS colonisation as GBS isolated using standard agar techniques alone rather than antibiotic media. It is associated with a higher risk of neonatal transmission and infection [4].

6.2. Identification

6.2.1. Traditional culture techniques

Definitive GBS identification requires serologic detection of the group B carbohydrate antigen, however clinical