

Neonatal and Infant *Streptococcus agalactiae* (Group B Streptococcus-GBS) Sepsis

BACKGROUND

GBS emerged in the 1970s as the commonest cause of neonatal and obstetric sepsis, mainly due to serotype III and, in the 1990s, as an increasingly common cause of septicaemia in adults, mainly due to serotype V. It is carried in the vaginas of 25-50% of healthy pregnant women and often transmitted to their infants before or during birth. Usually, colonisation is benign, but ~1% of the infants of carriers (~2/1000 overall) develops life-threatening sepsis.

A high proportion of cases of early onset infection are initiated *in utero* as a result of ascending infection, even in the presence of intact membranes. In some cases there are no obvious clinical risk factors, but the risk is increased by any condition that reduces the mother's ability to contain the organism, especially if conditions favourable to invasive infection occur. A past history of a GBS-infected infant and a previous GBS UTI are markers of increased risk and reflect a poor immune response to carriage. The risk is increased in women with conditions associated with immunosuppression (e.g HIV infection, diabetes or immunosuppressive therapy) or those that increases the risk of ascending infection (e.g. prolonged rupture of membranes or instrumental delivery). Preterm labour, with or without premature rupture of membranes, may be either a risk factor for or a clinical sign of intrauterine GBS infection. The risk of neonatal infection can be reduced by intrapartum antibiotic prophylaxis in women whose infants are at risk but there is controversy about how best to identify those at risk; routine antenatal screening for GBS carriage is recommended but has poor specificity.

Development of safer, more efficient ways to prevent GBS will require:

- better understanding of bacterial virulence and host susceptibility;
- surveillance to monitor genotype distribution and antibiotic resistance;
- methods to identify the small subset of GBS carriers whose infants are at risk.

STUDY OBJECTIVES

This study aims to determine:

1. the current incidence of early and late onset neonatal GBS infection
2. the incidence of currently accepted maternal and infant risk factors in children with GBS
3. the proportion, if any, of early onset GBS infections occurring in infants of women who have been given intrapartum antibiotic prophylaxis
4. the short-term mortality and morbidity of early and late onset GBS infection
5. the distribution of GBS genotypes among invasive isolates from different types of neonatal sepsis
6. differences in distribution of genotypes between isolates from infected neonates, pregnant women who are vaginal carriers and adults with bacteraemia.

CASE DEFINITION

Please report any infant with group B streptococcal disease confirmed by isolation of GBS from a normally sterile site e.g. blood, cerebrospinal fluid, joint fluid etc. Report all incident cases, irrespective of symptoms, in infants aged 0-7 days (early onset) or 8 days to 12 months (late onset) of age.

GBS may present clinically as:

- Early onset neonatal sepsis (birth to 7 days) with symptoms and signs varying in severity from overwhelming multi-organ system disease with shock, respiratory failure, meningitis, DIC or acute tubular necrosis (especially in preterm infants) to non-specific signs such as fever, lethargy and poor feeding, localised infection e.g. pneumonia, or even apparently asymptomatic bacteraemia (more likely in fullterm infants)
- Late onset sepsis (8 days to 12 months^a) with evidence of fever, lethargy, poor feeding, with or without signs of focal infection such as meningitis, bone or joint infection or urinary tract infection. Occasionally late onset infection presents as overwhelming sepsis with shock.

^a Most cases occur within the first 3 months of life but in some cases, especially localised infections such as meningitis, subdural abscess, septic arthritis, osteomyelitis etc. infection is acquired or presentation is delayed until much later in infancy.

FOLLOW-UP OF REPORTED CASES

A brief questionnaire requesting further details will be forwarded to clinicians that report a case of GBS to the APSU.

If you have any comments or questions please contact either:

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