GBS and the newborn infant

Group B streptococcus (GBS) is the most common cause of infection in the newborn period. It colonises more mothers and babies than it infects. However when GBS does cause infection, this can be devastating. Infection can be relatively 'silent' in the early stages. Prevention in the UK is a much debated issue: in the USA all pregnant mothers are screened for GBS and given antibiotics in labour if GBS is detected. Use of broad spectrum antibiotics in labour has however resulted in the emergence of other resistant bacteria and may contribute to allergy in later life.

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Key points

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1. There is a need for increased awareness of the risk factors and the signs and symptoms of GBS infection amongst healthcare workers and parents.
2. Support from parents and healthcare workers is required to carry out studies to improve detection of GBS.
3. Babies with GBS infection need to be treated promptly.

The newborn infant is vulnerable to infection from organisms acquired from the birth canal. The most commonly identified organism causing infection in the first week of life in the USA and Western Europe is called 'Group B streptococcus' or 'GBS'.

GBS colonisation

GBS lives in the lower gut and vagina in 10-25% of mothers – such women are described as being 'carriers' of GBS or 'colonised' with GBS. GBS is passed from one person to another by skin to skin contact and can be passed on through sexual contact. However, there are no known harmful effects of carriage itself and, since the GBS bacteria do not cause genital symptoms or discomfort, GBS is not a sexually transmitted disease. Neither is GBS carriage a sign of ill health or poor hygiene.

Being colonised with GBS in the vagina means that GBS is in the birth canal but is causing no harm to the mother. Babies too may be colonised with GBS. This is where GBS can be detected on the baby's skin, but it is not invading the blood stream and is causing no harm.

GBS infection

Babies can become very unwell when GBS causes infection – when GBS invades the blood stream, lungs or cerebrospinal fluid. In the UK a minimum of 0.7/1000 babies are infected per year[1]. These figures are based on 'culture-proven' GBS infections whereby GBS is grown in blood or cerebrospinal fluid. Because organisms cannot always be grown, even when they are present and causing infection, the actual number of babies affected by GBS could be as high as 3.6/1000[2].

A baby may have very subtle signs of infection in the early stages. They may simply not be feeding well or possibly be excessively sleepy. Some have a more rapid breathing rate and some may just stop breathing. If infection is suspected in a baby, the baby should have tests (including blood tests for organisms and possibly a chest X-ray) to investigate infection and be given antibiotics. Penicillin is the antibiotic of choice for GBS. Any organisms present in blood have to have a minimum of 48 hours to grow in a laboratory. During this time babies are treated with antibiotics until the results of the cultures are known. It is not uncommon for a baby to have signs of infection but for the tests on blood, swabs or cerebrospinal fluid to be negative. It is common practice to treat such babies with antibiotics for longer because of the difficulty in being certain about whether or not bacteria are causing their illness.

Studies are being conducted on new methods of detecting GBS in body fluids (blood or cerebrospinal fluid). These methods, using PCR (polymerase chain reactions), detect specific bacterial proteins in body fluids and swabs[3]. Such methods do not depend on having to grow the bacteria in culture. In animals these methods have been shown to result in a significantly more rapid detection rate of organisms than conventional culture methods[4,5]. In this way PCR may become a tool which will lead to more rapid and more reliable diagnosis of GBS infection in the newborn.

GBS infection can be very serious, especially for preterm babies. Some babies die from GBS infection[1] and some survive but have problems with their development later on, as do any babies subject to infection[6,7]. Mercifully most babies survive and grow up to be normal children, but this depends on the infection being detected early and treated promptly.

The big dilemma in the UK is what is the

best way of detecting GBS infection as early as possible in these babies. In the USA, where GBS infection was very much more common than in the UK, all mothers have a vaginal swab to look for GBS carriage late in pregnancy. If the swab is positive for GBS, the mothers are treated with penicillin in labour. This has resulted in a big reduction in babies infected with GBS. However many more mothers are treated with antibiotics than who actually need them. There is increasing concern that unnecessary use of antibiotics increases the risk of infections with resistant bacteria[8]. There is also some speculation that antibiotics may bring about changes in the baby's immune system as a result of changing the organisms living in the baby's gut[9]. Giving antibiotics may therefore be useful in the short term, but in the long term may bring about different problems.

There is ongoing debate about the best approach to preventing babies in the UK being infected with GBS. Screening mothers for GBS carriage would help, but it may result in an excessive use of antibiotics in mothers. What is undoubtedly true is that heightened awareness of GBS amongst healthcare workers – midwives, obstetricians, nurses and doctors – as well as parents is very important. Both groups should be aware of what situations in labour are associated with a higher risk of infection in the baby. For 40% of babies there are, however, no apparent risk factors before delivery. Signs that a baby may be unwell include a baby not feeding well, being excessively sleepy, and having a rapid breathing rate. Midwives, nurses and doctors need to be well aware of these early signs of GBS infection and investigate and treat infection promptly and properly. In the future better diagnostic methods with PCR will be instrumental in helping decide whether or not a baby is infected.

There are some promising studies of GBS vaccines which may prevent GBS infection, but these studies are in early phases and at present there is not currently a GBS vaccine that is routinely available.

References

1. **Heath P.T., Balfour G., Weisner A.M. et al., PHLS Group B Streptococcus Working Group.** Group B streptococcal disease in UK and Irish infants younger than 90 days. *Lancet* 2004; **363**(9405): 292-94.
2. **Luck S., Torry M., d'Agapeyeff K., Pitt A., Heath P., Breathnach A., Russell A.B.** Estimated early-onset Group B streptococcal neonatal disease. *Lancet* 2003; **361**(9373): 1953-54.
3. **Natarajan G., Johnson Y.R., Zhang F., Chen K.M., Worsham M.J.** Real-time polymerase chain reaction for the rapid detection of Group B streptococcal colonization in neonates. *Pediatrics* 2006; **118**: 14-22.
4. **Straka M., Cruz W.D., Blackmon C. et al.** Rapid detection of Group B streptococcus and *Escherichia coli* in amniotic fluid using real-time fluorescent PCR. *Infect Dis Obst Gynecol* 2004; **12**: 109-13.
5. **Heininger A., Binder M., Schmidt S., Unertl K., Botzenhart K., Doring G.** PCR and blood culture for detection of *Escherichia* coli bacteremia in rats. *J Clin Microbiol* 1999; **37**: 2479-82.
6. **Jacobsson B., Hagberg G.** Antenatal risk factors for cerebral palsy. *Best Practice Res Clin Obs Gynae* 2004; **18**: 425-36.
7. **Inder T.E., Volpe J.J.** Mechanisms of perinatal brain injury. *Semin Neonatology* 2000; **5**: 3-15.
8. **Stoll B.J., Hansen N., Fanaroff A.A. et al.** Changes in pathogens causing early-onset sepsis in very-low birthweight infants. *N Engl J Med* 2002; **347**: 240-47.
9. **Bedford Russell A.R., Murch S.H.** Could peripartum antibiotics have delayed health consequences for the infant? *BJOG* 2006; **113**: 758-65.

Case study – Léon

Our beautiful baby, Léon, was born on 7 December 2005 at 14:47 at St. Thomas' Hospital, London. My labour lasted just over 15 hours.

My waters were broken by the midwife only about 3 hours before Léon was born. I did not have a high temperature or show signs of any other risk factors to indicate my baby was at higher risk of developing Group B streptococcus infection. Léon started to show signs of distress and there was meconium present when my waters were broken, so I was given an epidural to enable the doctors to assist me to deliver him with forceps.

Within a couple of hours of delivery, Léon was taken to the special care baby unit because his blood glucose was too low. By 12:00 on the 8 December they moved him to the neonatal intensive care unit as he was struggling to breathe and they suspected an infection.

At 02:00 on 9 December, a midwife woke me and told me to phone my partner, Johan, at home and ask him to hurry to the hospital as Léon was in trouble. I got to his bedside and the consultant on call took me to one side and told me his oxygen level had dropped to only 30%. It was still dropping and she was going to try everything she could to save him. We asked them to call the chaplain to have him baptised and spent the whole night waiting to see what would happen. By 09:00, they said that he'd been stable for a few hours, but that his oxygen levels had started dropping again and his only chance of survival would be to go to Great Ormond Street Hospital to be treated on the ECMO machine. This would give his heart and lungs a rest while his body fought the infection.

By 19:00 he was at Great Ormond Street Hospital and stable, but extremely critical. He had neonatal sepsis, caused by two bacterial infections: Group B streptococcus and *E.coli*. The infections had caused multiple organ failure (lungs, kidneys and liver) and brain damage. He was kept on ECMO for 12 days, then he was transferred back to St. Thomas' Hospital neonatal intensive care unit on the 2 January 2006, still in an extremely critical condition.

Unfortunately, despite all their best efforts, Léon couldn't be saved and we lost him at 22:10 on Monday, 9 January 2006.

We are so thankful to the dedication of the medical teams at both St. Thomas' Hospital neonatal intensive care unit and Great Ormond Street Hospital cardiac critical care ECMO unit who fought continuously to try and save our boy, and for all the specialist treatment and assessments that he received during those 33 days of his short life when he put up such a brave fight. We, as parents, were also looked after by the wonderful staff at both hospitals and we will always be touched by the caring and kindness shown to us and Léon during this extremely difficult time.

Liesl Booysen