

Intra-Uterine Death Due To B-Haemolytic Streptococcus Despite Intact Membranes

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Citation

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Abstract

We report a case of a patient presenting with an intrauterine death at 40 weeks gestation. She had felt no fetal movements for 24 hours and her amniotic membranes were "intact". Despite all the routine microbiology tests being negative a diagnosis of group B streptococcus was only discovered in the baby when a post-mortem was carried out. "Intact" membranes does not always exclude the diagnosis of group B streptococcus infection. Obstetricians and midwives should explain to such unfortunate patients that a full diagnosis may not be possible without a post-mortem.

CASE REPORT

A 37 year old lady who had been suffering from primary sub-fertility for a number of years had a successful In-Vitro Fertilization pregnancy after just one attempt. She had a past medical history of coeliac disease but nil else of note and nuchal translucency scan carried out at twelve weeks confirmed that she was at low risk for fetal trisomies (trisomy 21 - 1 in 300 to 1 trisomy 13 18 1 in 655). She had been given low molecular heparin from her assisted reproduction clinic up until 20 weeks gestation and was continued on Aspirin until 32 weeks.

She had an uneventful pregnancy and was normotensive throughout, but unfortunately presented to Delivery Suite at 40 weeks and 2 days, having felt no fetal movements for 24 hours. An ultrasound scan confirmed an intra-uterine death. She had no features suggestive of abdominal pain, vaginal bleeding or spontaneous rupture of membranes and had just begun to experience intermittent tightenings over the past few hours. As she was already contracting, a plan was made for induction of labour and her amniotic membranes were not ruptured until she was late on in established labour (cervix - 9 cm dilated). A 4.2 kilogram male stillborn was delivered within 24 hours of the initial diagnosis.

There was no history of pruritis, diabetes or family history of stillbirths, intra-uterine deaths or venous thromboembolism. Based on the signs and symptoms a diagnosis of abruption was suspected, however there was no evidence of this on close inspection of the placenta after the delivery and the

fetus had a normal macroscopic appearance. The umbilical cord was found to be quite tight around the neck of the fetus, but the post-mortem revealed no evidence of acute asphyxia. The baby was on the 92nd centile weight for gestational age, although a glucose tolerance test at 28 weeks was negative and showed no evidence of gestational diabetes in the mother.

Histology confirmed acute chorio-amnionitis with fetal response and funisitis and microbiology grew Beta-haemolytic Streptococcus from the heart and lung. It was felt that this was the most likely cause of intra-uterine death. A urine sample showed evidence of *Escheria faecalis* but a high vaginal swab was negative and all other blood tests (*Syphilis*, *Listeria*, *Toxoplasmosis*, *Rubella*, *Cytomegalovirus* and *Parvovirus B19*) showed no evidence of any recent infection.

This intrauterine death appeared to be caused by Beta-Haemolytic Streptococcus despite the membranes being "intact" and the negative microbiology from the high vaginal swab before delivery and the placental swab taken after delivery. A *kleihauer* test was negative and a postnatal thrombophilia screen was also negative.

DISCUSSION

Group B Streptococcal (GBS) infection continues to be a major cause of perinatal morbidity and can be a cause of fatal intrauterine infections.

GBS intrauterine infection can follow ascending,

haematogenous, transabdominal or transfallopian pathways (Salafia et al, 2000) and although a history of “ruptured membranes” increases the risks of intrauterine infection, a history of “intact membranes” at the time of delivery does not always exclude the diagnosis.

Chorioamnionitis with “intact membranes” is extremely rare, but the exact incidence of these sources of infection is uncertain, since their absolute identification depends on the sensitivity of microbiological investigations and postmortem examinations being carried out on all intrauterine and neonatal deaths.

Kjaergaard et al (1999) demonstrated that the chorioamniotic membrane constitutes a competent barrier to group B streptococcus in vitro, however our case demonstrates that this may not always be true in vivo.

Cases of preterm intrauterine deaths due to group B streptococci with intact membranes have been reported (Desa et al, 1984, Neri et al, 1984 & Jones et al, 2004), but in almost all cases the deaths have occurred in association with antepartum haemorrhage, preterm labour or rupture of the membranes, or at gestations before 28 weeks (all the 15 cases described by Desa were before 28 weeks). Our case is unusual in that it presented with an intra-uterine death at 40 weeks gestation, despite “intact” membranes.

Jones et al has reported a case of recurrent mid- trimester fetal deaths caused by GBS chorioamnionitis despite “intact” membranes. In a subsequent pregnancy, they used oral amoxicillin 500mg four times daily from 12 weeks gestation and cycled this (2 weeks on and 2 weeks off) through out the rest of pregnancy. This resulted in a successful outcome, however, since recurrence of such intrauterine infections is very unusual even without treatment, the contribution of the antibiotic therapy to the favourable outcome must at best be speculative. In the current climate of concerns about bacterial antibiotic resistance and clostridium difficile, a more conservative regime is probably more appropriate, with antibiotics being reserved for proven infection, of the urinary tract for example.

Baker et al calculated the risk of an infant of a group B streptococci carrier mother contracting an infection to be around 1%. They considered the risk of intrauterine death to

be small, putting the overall risk of intrauterine death due to infection at only 0.13% and the risk of intrauterine group B streptococcal death at only 0.028% (approximately 1 in 4000).

Our case demonstrates the importance of obtaining a post-mortem on infants with an unexplained intra-uterine death and shows that that a negative microbiology screen taken at the time of delivery does not always exclude the diagnosis of chorioamnionitis. Obstetricians and midwives should explain to such unfortunate patients that a full diagnosis may not be possible without a post-mortem, and that a correct diagnosis has important implications for their future reproductive health.

Although routine antenatal screening for vaginal carriage of group B streptococci and antibiotic prophylaxis has greatly reduced the incidence of GBS in the USA, in countries with a lower incidence, such as the UK, its value has yet to be established. Immunoprophylaxis is likely to be the ideal solution in the future, but is not yet available.

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