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Fatal late-onset group B streptococcal sepsis on a special care baby unit

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Summary

A 3-week-old preterm baby developed late-onset group B streptococcal sepsis and died, despite therapy with penicillin, granulocyte transfusions and fresh frozen plasma. Serotyping and phage typing results strongly suggested late acquisition of the organism from the mother. The baby had previously developed and recovered from an Escherichia coli bacteraemia.

Introduction

The epidemiology and immunology of early-onset group B streptococcal (GBS) disease have been extensively studied, although many questions remain unanswered. The background to the late-onset form of the disease is much less clear. Here we report on a 3-week-old preterm baby who developed late-onset GBS disease following successful treatment of an Escherichia coli bacteraemia.

Case report

A 16-year-old was admitted at 31 weeks gestation with a life-threatening ante-partum haemorrhage. Placenta praevia had been diagnosed at 28 weeks. A baby girl weighing 1-8 kg was delivered by emergency caesarian section. At birth she required endotracheal intubation and ventilation for 1 hr, then only minimal ventilatory assistance for the next 12 hr. The baby was of 33 weeks maturity by Dubowitz assessment and had an uneventful first week. Following a period of temperature instability and poor weight gain, an infection screen (surface swabs, blood cultures, urine culture, and blood count) was performed on day 9. The total white blood count (WBC) was 9-7 x 10^9/litre, of which 40% were neutrophils; pending culture results, the baby was started on penicillin (60 mg/kg/day) and gentamicin (9 mg/kg/day). An E. coli, sensitive to gentamicin, was isolated from the blood cultures. The E. coli had not, however, been isolated from previously taken surface swabs. Gentamicin was continued for 7 days. Weight gain improved, the baby was moved into a cot, and breast-feeding started. Group B streptococci were not isolated from the surface swabs taken both before and after this episode of infection.

On day 18, 2 days after the withdrawal of antibiotics, the baby collapsed. She was hypotensive, cyanosed, had a profound metabolic acidosis (pH 7-01; base excess -20) and required resuscitation with intubation, ventilation, bicarbonate and plasma expanders. The blood count showed a marked neutropenia, the total leucocyte count being 2-8 x 10^9/litre with only 20% mature neutrophils and 12% band forms giving a band/total neutrophil of 0-54. Infection was presumed and therapy commenced with fresh frozen plasma, buffy coat transfusion and antibiotics (penicillin 45 mg/kg/day; gentamicin 9 mg/kg/day; metronidazole 22-5 mg/kg/day). Despite these measures, acidosis and shock persisted and the baby began to have convulsions. A GBS serotype IIIIR, phage type 8 was isolated from both blood culture bottles, but not from the cerebro-spinal fluid. A chest X-ray showed changes in the right upper lobe. At this time the penicillin dosage was increased to 240 mg/kg/day. The organism was sensitive to penicillin with minimum inhibitory and bactericidal concentrations of 0-03 mg/litre. The baby died on day 21 and a subsequent post-mortem showed widespread GBS infection with cerebral and meningeal involvement.

A vaginal swab from the mother grew GBS. She had been actively involved in caring for the baby, including some attempts at breast-feeding. Four
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members of the Special Care Baby Unit staff and one other baby were also found to be carrying GBS. However, only the maternal isolate was found to be the same serotype and phagetype as the infecting strain (Table 1). There was no evidence of spread of this strain within the unit.

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<th>Table 1. Details of GBS isolates</th>
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A sample of the baby’s serum was obtained on day 21. Using the technique of luminol-dependent chemiluminescence (Hastings and Easmon, 1981) a lack of serum opsonins to both the infecting isolate and a laboratory GBS type III strain was demonstrated. Complement activity was low with a CH50 of only 44% of control and there was no detectable antibody to GBS. Latex agglutination (Wellcome Reagents) showed the presence of GBS antigen in the serum to a dilution of 1 in 8.

Discussion

There is little published data on the source of the infecting strain in late-onset GBS disease. Non-maternal acquisition has been demonstrated (Baker, 1977) but in the present report the mother was almost certainly the index case. Surface swabs, taken from the baby on admission to the Special Care Baby Unit and on two occasions before the E. coli bacteraemia, did not grow GBS. Earlier transmission of the organism was probably avoided by the operative delivery, the initial period of intensive care and antibiotic treatment. Although late-onset GBS disease may result from transfer of the organism in maternal breast milk (Kenny and Zedd, 1977) it was not possible to obtain a milk sample from the baby’s mother to investigate this possibility. The mother did not have overt mastitis.

The first episode of sepsis involving E. coli was unremarkable. Clinical signs were minimal, the course was mild and, more specifically, there was no neutropenia. In contrast, the onset of the subsequently fatal GBS sepsis was severe and sudden. The clinical picture of ‘shock’, acidosis, and respiratory involvement is more typical of the early-onset form of the disease (Boyer et al., 1980). This type of bacteriogenic shock, although more usually associated with Gram negative infections, has been described in streptococcal, staphylococcal and pneumococcal bacteraemia. Neutropenia and a raised immature to mature neutrophil ratio are poor prognostic signs in neonatal sepsis, particularly when associated with depletion of the neutrophil storage pool (Christensen, Bradley and Rothstein, 1981). Marrow was not examined and we cannot therefore comment directly on the latter. However, extrapolation from previous work suggests that an immature to mature neutrophil ratio of about 0.5 would be associated with a neutrophil storage pool of between 15% to 20% (normal 30% to 50%) (Christensen et al., 1981).

E. coli and GBS together are responsible for the majority of cases of neonatal sepsis in this country. The K antigen of E. coli which is regarded as a marker for pathogenicity (McCracken et al., 1974) is rich in the carbohydrate 2-D-N-acetylneuraminic acid (sialic acid). Interestingly, this same carbohydrate has been found to be an integral component of the type III GBS antigen (Kasper, Goroff and Baker, 1978) and recent work has suggested it may play some part in pathogenicity (Hastings and Easmon, 1981).

Unfortunately, the serum sample used for the various immunological investigations was obtained only late in the course of the disease. This, and the presence of antigenaemia, make interpretation of antibody and complement levels difficult. However, the chemiluminescence results do demonstrate that the baby’s serum was unable to opsonise the invading organism.

This case underlines the difficulties of the prophylaxis and treatment of GBS disease. It has been suggested that interruption of transmission of the organism during labour, by giving penicillin to the mother, might cut down the incidence. However, this approach would not interfere with transmission at a later date as appears to have occurred in this particular instance. Rapid identification of the pathogen, using latex agglutination or countercurrent immunoelectrophoresis, might have resulted in an earlier increase in penicillin dosage, but whether this would have had any effect on the outcome is uncertain. It could also be argued that the use of fresh plasma from a donor with high titres of antibody to GBS might be an improvement on fresh frozen plasma in the treatment of GBS disease. However, whether more specific treatment might have saved a baby in whom acidosis, shock and cerebral involvement were so sudden, must be open to question.

Acknowledgments

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References


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