

# Neonatal group B streptococcal disease associated with infected breast milk

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## Abstract

**Premature triplets each developed late onset group B streptococcal disease over a period of nine weeks. The source of the organism appeared to be expressed maternal breast milk, in the absence of clinical mastitis. Asymptomatic excretion of group B streptococcus in breast milk may be an under-recognised cause of neonatal infection.**

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Group B streptococcus (GBS) is the commonest cause of neonatal meningitis/septicaemia in the United Kingdom. Most cases occur in the first week of life and are related to vaginal carriage in the mother. Late onset GBS disease (more than seven days after birth) is less common, and has been linked to cross infection from the hands of healthcare workers.<sup>1</sup> Alternatively, it may reflect delayed infection after early colonisation. There are also reports of late onset or recurrent disease associated with ingestion of infected mother's milk.<sup>2</sup> We report three cases of late onset disease in a set of triplets, in which the source of infection appeared to be the mother's expressed breast milk, a route of transmission that may not be widely recognised.

## Case histories

Female triplets were delivered by caesarian section at 26 weeks gestation, seven days after membrane rupture. They weighed between 730 and 845 g and were ventilated for five days. Two maternal high vaginal swabs taken before delivery were negative for GBS; the triplets did not have skin swabs taken. They received five days of intravenous benzylpenicillin and gentamicin, until initial blood cultures were confirmed to be negative. Enteral feeding with expressed breast milk was begun in the first week.

Triplet one had increasing apnoeas and bradycardias, with a metabolic acidosis on day 12. Her white cell count was  $28.7 \times 10^9/l$  and C reactive protein concentration 85 mg/l. Cerebrospinal fluid culture was negative, but GBS was isolated from blood cultures. She was treated with benzylpenicillin and gentamicin for seven days and made a good recovery. On day 63 she again became unwell, with fever and increased oxygen requirement. Investigations showed a white cell count of  $2.6 \times 10^9/l$  and C reactive protein concentration of 200 mg/l, and blood cultures were again positive for GBS. Benzylpenicillin and gentamicin were given for

14 days. Immunological investigations including nitroblue tetrazolium test, immunoglobulins, lymphocyte markers, and complement function tests were all normal at this time.

Triplet two developed persistent fever on day 35, with a white cell count of  $1.2 \times 10^9/l$  and C reactive protein concentration of 226 mg/l. Blood and cerebrospinal fluid cultures were positive for GBS. She was successfully treated with 14 days of benzylpenicillin and gentamicin.

Triplet three became pyrexial and bradycardic on day 33 with a white cell count of  $19 \times 10^9/l$  and C reactive protein concentration of 111 mg/l. Blood cultures were positive for GBS, but cerebrospinal fluid culture was negative. She was treated with benzylpenicillin and gentamicin for 10 days and made a good recovery.

On day 68, after the second episode of GBS disease had been diagnosed in triplet one, a sample of maternal expressed breast milk was cultured, yielding a pure growth of GBS ( $>10^5$  colony forming units (cfu/ml)). A repeat sample of freshly expressed breast milk also yielded  $>10^5$  cfu/ml GBS, but there was no clinical evidence of mastitis. Isolates from each baby and the mother were all shown to be serotype III. The mother was treated with oral amoxicillin for seven days. Repeated cultures of breast milk after treatment were all negative, but breast milk feeding was not reintroduced for other reasons. The triplets subsequently remained well and were discharged from hospital at 3 months of age.

## Discussion

Early onset GBS infections are normally acquired from the mother's genital tract. Late onset disease is believed to reflect delayed infection after early colonisation, or cross infection.<sup>1</sup> Early colonisation is less likely in this case because there was no maternal carriage of GBS before delivery. However, superficial and rectal swabs were not taken from the triplets at delivery so this cannot be completely excluded. There were no other cases of GBS infection over the period that the triplets were on the neonatal unit, and no evidence of cross infection. It seems probable that the source of infection in these triplets was the mother's expressed milk.

GBS is a well recognised cause of mastitis in cattle, causing loss of milk production (this is the source of its original name, *Streptococcus agalactiae*). Only one study has examined the breast carriage rate in humans, finding an incidence of 3.5% in 1132 samples from lactating mothers.<sup>3</sup> However, there have been several

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previous reports of transmission of GBS from mother to baby through breast milk, and recent cases have been confirmed using restriction fragment length polymorphism and pulsed field gel electrophoresis.<sup>4 5</sup> We believe that cases of transmission of GBS in breast milk may be under-reported. As shown in our case, GBS may be present in breast milk without causing clinical mastitis.

Rifampicin has been used successfully to eradicate carriage of the organism in mother and neonate when there was recurrent GBS infection, but these are isolated case reports.<sup>5 6</sup> We treated the mother of the triplets with amoxicillin, which successfully eradicated the organism from her milk.

#### CONCLUSION

Transmission of GBS through breast milk is unusual but should be considered in cases of late onset or recurrent GBS disease. Excretion

of GBS in breast milk can be eradicated with antibiotics.

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