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Late-onset Group B streptococcal disease by infected mother's milk detected by polymerase chain reaction

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SUMMARY

Late-onset Group B streptococcal (GBS) disease is a cause of illness, death and neurological sequelae in infancy. The epidemiology and pathogenesis of late-onset GBS disease is poorly defined. Infected breast-milk has been suggested as a source of postnatal infection and invasive disease. We describe a late-onset GBS disease by infected mother's milk in a term newborn in which the detection of GBS in neonatal bloodstream (confirmed by culture) and in the mother's milk was performed by PCR.

KEY WORDS: Group B streptococcus, *Streptococcus agalactiae*, Neonatal infection, Late-onset GBS disease, Breast milk, Polymerase Chain Reaction

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Group B streptococcus (GBS) is recognized as the most frequent cause of severe infection in newborns during the first week of life (early-onset disease), with incidence varying in different countries (Schrag *et al.,* 2002). Intrapartum antibiotic prophylaxis (IAP) has reduced the incidence of early-onset GBS disease, whereas the incidence of late-onset GBS disease (7-89 days) has remained unchanged (CDC, 2005). Maternal milk has been suggested among the possible sources of infection and it may be underestimated because the investigation of the presence of GBS in breast milk is not carried out routinely in late-onset disease.

We describe a case of late-onset GBS disease in a male term infant, weight 3800 g, delivered by planned caesarean section (CS) to a gravida 1 para 2 Caucasian mother. Pregnancy developed normally. Although GBS had been isolated from vagino-rectal swabs performed at 36 weeks of pregnancy, IAP was not done in agreement with CDC's 2002 guidelines about planned caesarean delivery performed before onset of labor in a woman with intact amniotic membranes (Schrag *et al.,* 2002). Newborn Apgar scores at 5 and 10 minutes were 9 and 10 respectively but in the following first hour the infant presented tachypnea and grunting. There were no laboratory markers of sepsis and a blood culture was obtained before empirical antibiotic therapy with ampicillin/sulbactan intravenously was started. The therapy was stopped four days later, since clinical the picture and blood culture were completely negative. Enteral feeding with maternal expressed breast-milk was started on the second day of life, with full feeding achieved within 1 week. The newborn was discharged from the hospital at five days of life apparently healthy. On day 26 poor sucking and lethargy was noted by

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his mother and within a few hours abnormal crying, irritability on being touched and fever appeared. He was admitted to the neonatal unit in serious condition with greyish colour, poor perfusion with *cutis marmorata*, lethargy, muscular hypotonia, tachycardia, tachypnea, grunting, arterial hypotension and fever (38.2°C). No bulging fontanelle nor other signs of meningeal involvement were present. The mother showed no signs of mastitis, but she referred mild left breast tension and pain in the third week post partum; exclusively breast-feeding had been continued, without assumption of any therapy. Laboratory data at admission revealed a WBC count of $8.7x10^9$/l with a significant left shift (34% band forms and 65% neutrophils), CRP serum concentration was 8.7 mg/dl returning to normal level within 6 days. Microbiological and biochemical analysis of cerebrospinal fluid (CSF) showed no abnormalities.

Blood, urine and CSF baby's cultures were taken and empiric broad-spectrum therapy was started with ampicillin and netilmicin intravenously. Blood cultures and Real-Time Polymerase Chain Reaction (RT-PCR) were scored positive for the presence of *Streptococcus agalactiae*, which was susceptible to ampicillin. All the remaining cultures were negative. Maternal breast milk was aseptically collected and submitted for culture.

Results of breast milk bacterial cultures were negative whereas the same milk sample tested by the RT-PCR method recently described (Lanari *et al.,* 2006) showed the presence of *Streptococcus agalactiae.* The mother was given oral amoxicillin for seven days until PCR on milk became negative and she was suggested to continue to breastfeed her infant. The baby was treated for 10 days with ampicillin whereas netilmicin was stopped when antimicrobial susceptibility data become available. At the end of this period he recovered, with normalization of laboratory findings and he was discharged from hospital in good health. There were no sequelae at six months of life.

The carriage of GBS in the mother's vagino-rectal tract makes perinatal transmission possible, but this was highly improbable in this case, since delivery was performed by CS, before onset of labor and amniotic membrane rupture. Moreover the age of the infant at the onset of sepsis, after a period of good health, the antibiotic therapy administered for four days after birth and the negativity of the blood culture and laboratory parameters performed at the first day of life rule out a recurrent infection following an early colonisation. In agreement with other authors (Godambe *et al.,* 2005, Arias-Camison, 2003) we prove that breast-milk is a potential source of GBS infection of child. We believe that the implication of infected milk in determining late-onset GBS disease may be underestimated and we suggest that bacteria investigation on milk should be performed whenever a breast-fed infant presents with late-onset sepsis and preventively for expressed breastfed preterm infants, that are more susceptible to invasive infections. In our case the only test able to identify the presence of GBS in milk was PCR, likely due to the higher sensitivity of this method. The result obtained by PCR linked the late-onset GBS disease of the baby with the *S. agalactiae* excretion in the mother's milk. Consequently we suggest PCR should be preferred to identify GBS in breast milk.

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