Neonatal Group B Streptococcal Septicemia Transmitted by Contaminated Breast Milk, Proven by Pulsed Field Gel Electrophoresis in 2 Cases

To the Editors:

Group B streptococcus (GBS) is a leading cause of sepsis and meningitis in newborns and infants.[1] Breast milk has been suggested as a route of transmission for GBS and as a cause for late-onset GBS infection.[2–5] We report 2 cases of premature infants, who developed severe septicemia caused by GBS-contaminated breast milk. The route of transmission was verified using pulsed field gel electrophoresis performed on the GBS-strains found in stored breast milk samples and in blood cultures of the infants.

The first infant was a premature female of 27+4 weeks gestational age. At 46 days of age, the child showed signs of septicemia. Antibiotic treatment was started immediately with vancomycin and meropenem. The blood culture showed GBS. Cefotaxime was added to the antibiotic regimen and antibiotic treatment was completed after 7 days. On day 64 of life, septicemia was diagnosed again. Antibiotic treatment with vancomycin and meropenem was started. Again GBS infection was proven by positive blood culture. The antibiotic regime was changed to cefotaxime and gentamicin was added. Antibiotic treatment was continued for 10 days. With onset of the second sepsis, the mother mentioned having mastitis. Because of a positive maternal cytomegalovirus status, mother's milk had been stored and frozen to reduce the cytomegalovirus count. In 2 of these frozen breast milk samples, GBS was found. The GBS-strains isolated from the infant's blood cultures and mother's milk were sent to the German National Reference Center for Streptococci (Nationales Referenzzentrum für Streptokokken, Institut für Medizinische Mikrobiologie der RWTH Aachen, Pauwelstrasse 30, 52074 Aachen, Germany), pulsed field gel electrophoresis was performed on all samples (see Figure, Supplemental Digital Content 1, http://links.lww.com/INF/B754) and the isolates were found to be identical.

The second patient, a premature infant of 27+0 weeks of gestational age, developed infection at 46 days of life. The infant was treated with vancomycin and meropenem. The blood cultures were positive for GBS. Cefotaxime was added to the regime and treatment was for 11 days. The mother had mastitis. Cultures of the milk yielded GBS. The GBS isolates in the blood culture and breast milk were identical by pulsed field gel electrophoresis.

We believe that breast milk is not a common, but a possible route of GBS transmission. We suggest culturing breast milk for GBS in mothers, whose children have recurrent GBS infections or who have mastitis.

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The authors have no funding or conflict of interests to disclose.
Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's website (www.pidj.com).

ISSN: 0891-3668/14/3304-0429
DOI: 10.1097/INF.0000000000000206

Growth Failure in Pediatric Tubercular Meningitis

To the Editors:

In regions where incidence rates of tuberculosis are low, like Italy, extrapulmonary manifestations of diseases are seen primarily in adults with reactivation infection, and the dominant form of central nervous system disease is tubercular meningitis (TBM). Despite adequate therapy, TBM patients have about 30% mortality and 50% of TBM survivors have neurologic sequelae.[1]

There have been very few studies concerning growth abnormalities in tuberculosis.[2–5] Reports on growth consequences in pediatric patients affected by TBM are particularly rare and do not include a detailed patient follow up.[3]

We verified the growth pattern in surviving children hospitalized at the Bambino Gesù Children Hospital, Rome, Italy, for TBM between April 1, 2006, and April 1, 2013 (cases). Bambino Gesù Children Hospital is a third-level pediatric hospital with approximately 200 beds. The control group was composed of children affected by tuberculosis but without meningeal localization. Patients over 18 years of age as well as children with immunodeficiency disorders were excluded. In the children with TBM, as well as in the control group, anthropometric measurements were collected at onset and at any follow-up visit by a specialized nurse and expressed in standard deviation score (SDS) according to sex and age.

We reviewed 8 prepubertal cases of TBM with a mean age of 4.13 years. There were more females than males (62% vs. 38%). The control group was composed of 27 prepubertal patients with a mean age of 4.18 years, of whom 66.6% were females and 33.4% males. At admission, height SDS was normal and similar in both groups of patients (0.60 SDS for cases and −0.01 SDS for controls).

At 1-year follow up, despite prompt therapy, height velocity significantly decreased in the TBM group (height −2.65 SDS; $P < 0.01$). Conversely, it remained within the normal range in the control group (height 1.39 SDS).

One limit of our study is that it was carried out retrospectively, so that we cannot exclude a potential concomitant endocrinologic cause of growth failure. We did not evaluate the GH-IGF1 axis and the secretion of hormones such as ACTH, TSH and

The authors have no funding or conflicts of interest to disclose.

ISSN: 0891-3668/14/3304-0429
DOI: 10.1097/INF.0000000000000247

GH. However, we could speculate that these children do not have any endocrinologic disorders influencing growth, because at the time of admission for TBM their height was within the normal ranges for age and sex. Consequently, we suggest that growth failure in TBM, as well as in other pathologies, is mainly due to inflammation.

Our results suggest that TBM patients may experience auxologic consequences. The mechanisms involved in growth failure in TBM are unknown and the reduced growth rate observed in these patients requires both long-term follow up and further case studies. A limit of this report is that it included just 8 patients because TBM is rare among children in Italy and many children affected by TBM did not survive the acute infection.

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Erratum

Changes in Infectious Disease Mortality in Children During the Past Three Decades: ERRATUM

In the article appearing on page e355, volume 32, number 9, several errors in the text occurred. Percent signs were incorrectly used when per mille signs were appropriate in some instances magnifying the mortality rates significantly. Below are the corrected sentences in which percent signs are replaced by per mille signs.

Abstract, Results, 1st sentence: "Childhood mortality due to infectious diseases decreased by 89%, from 0.12‰ in 1969 to 0.013‰ in 2004, and neonatal mortality by 69%, from 0.50‰) to 0.16‰."

Materials and Methods, 3rd paragraph, 1st sentence: "Mortality rates were calculated in proportion to those at risk of dying (ie, all living children in the same age group) and rounded to deaths per 1000 children (‰)."

Results, Childhood Mortality due to Infections, 1st paragraph, 2nd sentence: "Infection mortality declined by 89%, from 0.12‰ (95% CI:0.10–0.15‰) in 1969 to 0.013‰ (95% CI: 0.007–0.023‰) in 2004."

Results, Neonatal Mortality From Infections, 1st paragraph, 2nd sentence: "…it did show a significant general decline by 69%, from 0.50‰ (95% CI: 0.35–0.70‰) in 1969 to 0.16‰ (95% CI: 0.07–0.30‰) in 2004."

Discussion, 1st paragraph, 3rd sentence: "…and infection mortality among the same age group in the United States declined by 30% between 1980 and 1992, from 0.30‰ to 0.21‰."

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Lantto M, Renko M, Uhari M. Changes in Infectious Disease Mortality in Children During the Past Three Decades. *Pediatr Infect Dis J*. 2013;32:e355–e359.