A Fatal Case of *Mycoplasma hominis* Meningoencephalitis in a Full-Term Newborn

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We report the case of a 20-day-old full-term baby, born to a mother who had had an uncomplicated pregnancy and delivery, who died 13 days after the onset of meningitis. *Mycoplasma hominis* was the sole agent repeatedly recovered from cerebrospinal fluid and from postmortem brain tissue.

A 37-week-old male infant weighing 4,020 g was deliveredatraumatically by vacuum extraction a few hours after artificial rupture of membranes. His Apgar scores were 4 at 1 min and 9 at 5 min. His 20-year-old mother had an uncomplicated pregnancy and delivery. Mother and child were discharged after a 10-day uneventful postpartum period. The baby was breast fed and did well until the morning of day 20, when he developed fever and irritability and refused to be fed. That afternoon he was admitted to the hospital, where a lumbar puncture confirmed the diagnosis of meningitis. Cerebrospinal fluid (CSF) contained 650 leukocytes per mm³ (9% neutrophils and 91% lymphocytes), 915 erythrocytes per mm³, 175 mg of protein per dl, and 9 mg of glucose per dl (blood glucose, 112 mg/dl). No organism was detected by Gram staining. The peripheral leukocyte count was 24,000 cells per mm³ (64% neutrophils and 20% lymphocytes), and the concentration of CRP was 7.4 mg/dl. The baby was given an initial regimen of ampicillin (200 mg/kg of body weight per day) and amikacin (15 mg/kg/day), which was later found to be sensitive to tetracycline (ATB Mycoplasmes, API system, La Balme Les Grottes, France). For the following 5 days, the child continued to deteriorate, with development of intracranial hypertension and coma, and he died at the age of 33 days. Repeated bacterial and viral cultures from CSF were all negative. *M. hominis* was still recovered from CSF obtained on day 11. Postmortem examination was unremarkable, except for the brain, which showed diffuse necrosis and yielded a pure culture of *M. hominis*. Cultures for bacteria, including mycobacteria, were negative. Postmortem viral cultures were not performed.

*Mycoplasmas* are small pleomorphic bacteria 0.3 to 0.8 μm in diameter that lack cell walls and are bounded only by a cell membrane with a significant sterol content. At least 15 species have been isolated from humans; among these 15, only 3 (*Mycoplasma pneumoniae*, *M. hominis*, and *Ureaplasma urealyticum*) are well-established human pathogens. Most of these species are fastidious and/or slowly growing, making their routine diagnostic testing not readily available and justifying the development of hybridization techniques for their detection (16). However, *M. hominis* and *U. urealyticum* grow rapidly on specific media and can be identified from clinical specimens within 2 to 5 days (2). Moreover, in some instances, *M. hominis* will be discovered by observing pinpoint colonies on conventional unsupplemented media providing an adequate (1-week) incubation period, thus allowing its detection even when mycoplasmal culture is not initially requested. These tiny colonies, which have a typical fried-egg appearance on arginine agar media when examined under a stereomicroscope, are made of organisms which fail to stain by the Gram stain.

*M. hominis* is a frequent colonizer of the genital tracts of pregnant women (9, 14). This organism, which may be transiently isolated from the blood of asymptomatic women immediately after normal vaginal delivery, is responsible for some cases of postpartum fever, presumably by causing endometritis. In newborn infants, acquisition of the organism usually occurs by passage through the colonized birth canal. Rare cases of intrauterine infection and postnatal transmission have also been described elsewhere (10).
Although several reports have focused on the association between maternal *M. hominis* colonization and the outcome of pregnancy (3, 6, 9), the pathogenic role of this organism in neonates with suspected infections has not been clearly demonstrated. In particular, the significance of isolation of *M. hominis* from CSF is not obvious. In a prospective study of 69 neonates who underwent a diagnostic workup for suspected sepsis, Valencia et al. (15) isolated *M. hominis* from CSF in 9 cases. Eight of these babies were asymptomatic, and the biochemical findings of their CSF analyses were within normal limits for newborns. These authors suggested that the presence of these organisms in the CSF could be tied to a blood-brain barrier thought to be more permeable in the very early neonatal period and concluded that their pathogenic role remains to be assessed. Waites et al. prospectively investigated two cohorts of newborn infants undergoing lumbar puncture for suspected sepsis and/or meningitis. In their first study (19), involving 100 predominantly preterm infants derived from a high-risk obstetric population, *M. hominis* was isolated from the CSF of 5 babies, of whom only 1 had neurological signs and CSF alterations suggestive of congenital meningitis. In the second study (18), *M. hominis* was isolated from CSF of 9 of 318 infants belonging to a general population. Lack of inflammatory response in the CSF was the rule. Most of the infants had a benign course of infection and made an uneventful recovery without specific treatment. Waites et al. proposed both the amoebic fluid and the respiratory tract of the newborn as likely sources of mycoplasmal CSF infection via bloodstream spread; they considered *M. hominis* as a common cause of neonatal meningitis, probably underdiagnosed because of minimal neurological signs, the unreliability of CSF pleocytosis as a marker for mycoplasmal infection, and the difficulty of obtaining positive cultures on routine bacteriological media. By contrast, in a prospective study of 203 infants with suspected sepsis, Likitnukul et al. (7) did not isolate *M. hominis* from any of the 191 blood and 199 CSF samples tested. They confirmed their findings in an additional study of 35 infants aged 7 to 90 days with aseptic meningitis (8) and concluded that these organisms were rare causes of sepsis and/or meningitis in young infants. There are, however, several case reports describing isolation of *M. hominis* from the CSF of infants with both obvious clinical and biological signs of meningitis. Most of these cases were observed either in premature babies born to a high-risk maternal population (1, 4, 5, 11, 12, 13), in full-term newborns with neurological defects (11), or in hydrocephalic children with ventriculoperitoneal shunts (10, 17). The organism can persist for weeks in the CSF, with occasional spontaneous clearance (20).

The present case is unusual in that it occurred in a full-term baby with normal gestation and birth who developed meningitis at the age of 20 days with a fatal outcome 13 days later. It corroborates the suggestion of Waites et al. (16) that only severely affected infants with mycoplasmal meningitis would be identified by currently available routine diagnostic procedures. Nevertheless, suspicion of *M. hominis* infection must be raised when neonates and young infants have clinical signs of meningitis but negative CSF cultures. Indeed, although routine use of appropriate culture media for *Mycoplasma* recovery is not worthwhile, the ability of this organism to form small colonies in standard bacteriological media providing that incubation is extended for some days should allow its isolation in many instances and lead to initiation of effective antimycoplasmal therapy, which is not the case for antibiotics usually recommended for the treatment of meningitis in children.

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REFERENCES