Case Report

Mycoplasma hominis as a cause of septic hip arthritis in a neonate

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Introduction: Mycoplasma hominis frequently colonizes the adult genitourinary tract and causes significant diseases such as chorioamnionitis in adults. It also causes systemic infections such as pneumonitis and meningoencephalitis in neonates via vertical transmission. Here, we present a case of septic hip arthritis in a full-term neonate born to a woman who had intrapartum fever with premature rupture of the membranes.

Case presentation: A 0-day-old newborn female, whose mother had a significant history of intrapartum fever and 52 h of premature rupture of the membranes, developed a fever after birth. Empiric antimicrobial therapy was initiated for sepsis; however, the fever did not subside. Subsequently, she developed a subcutaneous scalp abscess and right hip joint arthritis despite continued cell-wall-active antimicrobial therapy. Blood and scalp lesion samples were found to be positive by bacterial culture, and pinpoint non-haemolytic colonies were observed on blood agar plates; however, no organisms were observed by Gram staining. Microbiological analyses using Mycoplasma-specific growth plates demonstrated that the samples were positive for Mycoplasma hominis, and PCR analysis was positive for 16S rRNA in blood, abscess and joint fluid samples. The patient was treated with surgical debridement and a 3-week course of clindamycin and was discharged from hospital without sequelae.

Conclusion: To the best of our knowledge, this is the first report of M. hominis causing septic hip arthritis in a neonate. Therefore, M. hominis should be included as a causative organism when treatment with empiric antimicrobial therapy is not effective in septic neonates.

Keywords: clindamycin; Mycoplasma hominis; neonate; scalp abscess; septic arthritis.

Introduction

Mycoplasma hominis frequently colonizes the adult genitourinary tract and is present in large numbers in the vagina of most women with bacterial vaginosis (Waites et al., 2005). Therefore, vertical transmission occurs frequently during birth. Ureaplasma is also often observed and might be involved in bacterial vaginosis; however, the precise relationship between these organisms and infection remains unclear (Waites et al., 2005). M. hominis has also been implicated as a cause of urinary tract infection, cervicitis and chorioamnionitis (Taylor-Robinson, 1996; Waites et al., 2005). In contrast, non-genitourinary-tract diseases including central nervous system infections, wound infections and arthritis have been reported in immunocompromised individuals who have received solid-organ transplantation, or who were diagnosed with hypogammaglobulinaemia (Taylor-Robinson, 1996). In addition, M. hominis is a potential cause of pneumonitis and meningoencephalitis, as well as bacteremia, and subcutaneous and brain abscesses in neonates (Abdel-Haq et al., 2002; Rao et al., 2002; Taylor-Robinson, 1996; Waites et al., 2005). To the best of our knowledge, this is the first report of M. hominis causing septic arthritis in a neonate. The infection was confirmed by the typical appearance of
colonies on special medium and by PCR analysis of different samples including blood, abscess and synovial fluid.

Case report

A term, female neonate, who was born to a 27-year-old primigravida woman by vacuum-assisted vaginal delivery because of prolonged labour, was presented to our neonatal care nursery soon after birth because of fever. Her prenatal history was notable because of maternal fever and premature rupture of the membranes at 52 h before delivery. Her birth weight was 3990 g and Apgar scores were 9 and 9 at 1 and 5 min, respectively. Upon physical examination, she was not acutely distressed and appeared well. Her temperature was 38.1 °C, with a respiratory rate of 65 min⁻¹, heart rate of 150 min⁻¹, blood pressure of 66/46 mmHg and percutaneous oxygen saturation of 97 % in room air. Her physical examination was unremarkable and she had no skin lesions or joint swelling. Laboratory findings showed a white blood cell count of 15 080 μl⁻¹ consisting of 71 % neutrophils and 19 % lymphocytes, and serum C-reactive protein levels were 0.01 mg dl⁻¹. Given the maternal history of fever and premature rupture of membranes, intravenous ampicillin and third-generation cephalosporin were initiated for possible early-onset sepsis pending blood culture results. Although her general condition was stable, her routine white blood cell counts and C-reactive protein increased after several days to 21 590 μl⁻¹ and 8.36 mg dl⁻¹, respectively, and her low-grade fever persisted. The clinical course indicated a systemic infection, and we performed a lumbar puncture on day 6, which showed no pleocytosis with normal levels of glucose and protein, an absence of organisms by Gram staining and negative cerebrospinal fluid culture. On the same day, the patient developed a soft-tissue swelling on the right occipital area of the scalp measuring 1 cm in diameter in addition to a cephalohaematoma that had been present on the left parietal area of the skull for a few days. Over the subsequent few days, serial blood, urine and cerebrospinal fluid samples were obtained and the antimicrobial therapy was continued. Although the antimicrobial treatment was changed to vancomycin and meropenem on day 10, her clinical condition did not improve. On day 11, the right occipital lesion became more erythematous, fluctuant and larger; therefore, drainage of the lesion was performed. Purulent fluid and blood were drained from the right scalp region and left cephalohaematoma, respectively, and both were sent for aerobic and anaerobic culture. Gram staining of the samples did not show any organisms present. On day 13, pinpoint colonies were observed on blood agar plates after inoculation with specimens obtained from the scalp; however, no organisms were seen by Gram staining. These findings indicated *M. hominis* as the causative micro-organism (Waites et al., 2005). In addition to the scalp lesions, we observed that the patient cried and had reduced movement of her right leg during diaper changes. On the same day, magnetic resonance imaging confirmed the diagnosis of arthritis with findings of right hip effusion, and contrast enhancements within the adjacent muscles without bone involvement. Shortly after the diagnosis, surgical drainage was performed and the antibiotic was changed to clindamycin on day 13. The collected synovial fluid had increased opacity because of the large numbers of nucleated cells present, but no organism was detected by Gram staining; however, pinpoint non-haemolytic colonies were present on blood agar plates on the subsequent day as shown in Fig. 1(a). *M. hominis* was detected in specimens including blood from the cephalohaematoma, scalp abscess aspirates, umbilical cord and synovial fluid by PCR analyses targeting the 16S rRNA gene (Lane, 1991). Pinpoint non-haemolytic colonies also grew on blood agar plates inoculated with multiple specimens including urine on day 5, fluid from scalp lesions and cephalohaematoma, blood on day 11, and synovial fluid on day 13. Colonies

![Fig. 1](image_url). *M. hominis* on blood agar and *Mycoplasma* agar plates. (a) Non-haemolytic pinpoint colonies grown on a blood agar plate from specimens obtained from the synovial fluid of the patient. (b) Colonies with a fried-egg appearance grown on a *Mycoplasma* agar plate under ×100 magnification with the centre of the colonies stained dark blue and the periphery of the colonies stained light blue by Dienes’ stain.
obtained after inoculation of the cephalohematoma material were spread over the surface of a *Mycoplasma* agar plate. Following incubation at 37 °C for 10 days, typical fried-egg colonies were seen, enhanced by the application of Diene’s stain as shown in Fig. 1(b). After surgery, the fever subsided and intravenous clindamycin was continued for 3 weeks. The patient was then discharged from the hospital on postnatal day 39 without sequelae.

**Discussion**

Septic arthritis is not a common disease in childhood; however, it occurs frequently in younger children, including neonates (Riise et al., 2008; White & Goldberg, 2012). It is important to note that early diagnosis and appropriate management are vital to prevent permanent disability of the joints. *Staphylococcus aureus* is the most common pathogen that causes septic arthritis in children. In neonates, *Streptococcus agalactiae* and Gram-negative bacilli are the most important pathogens that cause septic arthritis (Morrissy, 1989). A penicillinase-resistant penicillin plus a broad-spectrum cephalosporin or aminoglycoside are the most common antimicrobials used to treat these organisms (Kabak et al., 2002; Morrissy, 1989). Although it is rare, other micro-organisms including other bacteria, viruses, mycobacteria and fungi can be causative agents of septic arthritis in different clinical settings (Morrissy, 1989).

Making a diagnosis of *M. hominis* infection is often difficult because of the need for the use of specialized media and expertise that are not widely available in regular clinical laboratories (Waites et al., 2005). PCR is used to detect *M. hominis* in clinical settings but is not commercially available. Therefore, an in-house PCR method is often used, which has variable sensitivity and specificity depending on the techniques and methodologies of each laboratory. The usefulness of PCR assays for detecting *Mycoplasma* and *Ureaplasma* spp. have been reported previously; however, few studies have compared the sensitivity and specificity of PCR assays with culture methods for detecting *M. hominis* (Abele-Horn et al., 1996; Amirmozafari et al., 2009; Luki et al., 1998; Stellrecht et al., 2004). One study that examined 258 specimens including gynecological and urological samples and samples from newborn infants reported that the sensitivity and specificity of PCR for *M. hominis* were 95 and 99 %, respectively, considering conventional culture as the ‘gold standard’ (Abele-Horn et al., 1996). In this study, samples from newborn infants included oropharyngeal swabs, tracheal aspirates, cerebrospinal fluid and urine specimens; however, detailed information was lacking. Another study using 210 endocervical swab samples reported that the sensitivity and specificity of PCR versus culture for *M. hominis* were 74 and 87 %, respectively (Amirmozafari et al., 2009). In these studies, specimens were obtained from various anatomical sites, not from sterile sites such as synovial fluid or blood, and thus it was difficult to interpret the data. Further studies to evaluate the validity of PCR for these pathogens are warranted. In the current case, bacteremia developed during the late phase of disease, which made it difficult to diagnose the *M. hominis* infection.

*M. hominis* is susceptible to tetracycline and fluoroquinolones but is generally not susceptible to macrolides *in vitro* (Taylor-Robinson & Jensen, 2011; Waites et al., 2005). Because of the toxicity and safety issues of these antimicrobials in neonates, we chose to use clindamycin, which has been reported to be effective *in vitro* (Taylor-Robinson & Jensen, 2011; Waites et al., 2005) and has been successful for treating neonatal scalp and brain abscesses caused by *M. hominis* (Abdel-Haq et al., 2002; Rao et al., 2002).

In conclusion, to the best of our knowledge this is the first reported case of neonatal septic arthritis caused by *M. hominis*. Appropriate antimicrobials were not provided because of a delay in identification of the causative organism. *M. hominis* should be included as a potential causative organism by differential diagnosis when a neonate develops arthritis with maternal genitourinary disease and chorioamnionitis without apparent growth in routine cultures of specimens, for pinpoint colonies with negative Gram staining on blood agar plates, and when empiric β-lactam antimicrobial therapies do not show clinical improvement.

**Acknowledgements**

We express our gratitude to Drs Kimiko Ubukata and Naoko Chiba at Keio University for the identification of *M. hominis*. The patient’s guardian gave informed consent for this publication. The authors declare no conflicts of interest.

**References**


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