Short Communication

A Case of Congenital Folliculitis Caused by *Pseudomonas aeruginosa* in a Preterm Neonate

Koichiro Matsui, Kaoru Okazaki*, Yuho Horikoshi, Ryota Kakinuma, and Masatoshi Kondo

1Division of Neonatology and
2Division of Pediatric Infectious Diseases, Tokyo Metropolitan Children’s Medical Center, Tokyo 183-8561, Japan

SUMMARY: Intrauterine infections are associated with life-threatening neonatal conditions such as sepsis, intracranial hemorrhage, and chronic lung disease. Herein we present a case of generalized congenital folliculitis caused by *Pseudomonas aeruginosa* in a preterm neonate of 27 weeks gestational age successfully treated with antibiotics. Folliculitis is an important manifestation of intrauterine *P. aeruginosa* infection, and prompt, effective treatment is crucial to ensuring a good prognosis.

*Pseudomonas aeruginosa* is a common pathogen involved in nosocomial infections and accounts for 4% of cases of late-onset neonatal sepsis (1). The rate of mortality due to *P. aeruginosa* infection among neonates is reportedly as high as 56% (1). Thus, the clinical management of neonatal *P. aeruginosa* infections is crucial. Intrauterine infections, including bacterial infections, lead to various neurological impairments due to an overproduction of cytokines/chemokines, resulting in a fetal inflammatory response syndrome. Thus, it is important to detect and treat *P. aeruginosa* infections in neonates as early as possible. Herein we report a case of congenital *P. aeruginosa* infection presenting as multiple small red papules soon after birth, which was successfully treated with antibiotics. A written informed consent was obtained from the parents for the use of patient data in this case report.

An extremely preterm male neonate weighing 1,030 g was delivered at a gestational age of 27 weeks by emergency cesarean section due to an intrauterine infection, which was diagnosed based on the maternal clinical signs (fever and uterine fundal tenderness) and blood test results (leukocytosis and remarkably increased C-reactive protein levels). There was no significant family history of disease. The infant’s mother had gestational diabetes but no history of membrane rupture or neurological sequelae.

Foliculitis was diagnosed because the papules were located in the same sites as the lanugo hair follicles. The skin biopsy could not be performed because of the anatomical and physiological prematurity of the skin of preterm infants. A blood examination disclosed high levels of C-reactive protein (1.24 mg/dL) and a low leucocyte count (3,450/mm³), suggesting the presence of a neonatal bacterial infection. Intravenous ampicillin and gentamicin were started empirically. The skin lesions completely resolved only 2 days after birth. On postpartum day 3, *P. aeruginosa* was detected in all of the cultures except the blood culture, indicating the presence of a neonatal bacterial infection.

P. aeruginosa rarely causes intrauterine infections. However, congenital *P. aeruginosa* infections can lead to the mother in order to prevent bacterial infection for 3 weeks. The amniotic fluid was clear at delivery. The placental pathology showed acute chorioamnionitis, umbilical vasculitis, and chorionic vasculitis.

After birth, the infant was intubated, and surfactant was administered. The Apgar scores were 3 and 8 at 1 and 5 minutes, respectively. In the delivery room, numerous small red papules were observed covering the patient’s entire body (Fig. 1). Folliculitis was diagnosed because the papules were located in the same sites as the lanugo hair follicles. The skin biopsy could not be performed because of the anatomical and physiological prematurity of the skin of preterm infants. A blood examination disclosed high levels of C-reactive protein (1.24 mg/dL) and a low leucocyte count (3,450/mm³), suggesting the presence of a neonatal bacterial infection. Intravenous ampicillin and gentamicin were started empirically. The skin lesions completely resolved only 2 days after birth. On postpartum day 3, *P. aeruginosa* was detected in all of the cultures except the blood culture, including the tracheal aspirate, gastric aspirate, pharynx, skin, stool, and papule cultures. Besides *P. aeruginosa*, no bacterium or fungus was detected in any of these cultures. Based on these findings, folliculitis due to *P. aeruginosa* was diagnosed (2). The antibiotic was then switched to piperacillin for 17 days. The infant was extubated at 8 days of age and discharged at 98 days of age without any complications, such as chronic lung disease or neurological sequelae.

*P. aeruginosa* rarely causes intrauterine infections. However, congenital *P. aeruginosa* infections can lead to...
serious complications including sepsis and pneumonia and may have neurological sequelae or result in neonatal death.

In the present case, how the neonate was infected with *P. aeruginosa* remained unknown as *P. aeruginosa* was not detected in the vaginal discharge one week before delivery. *P. aeruginosa* is known to account for approximately 2% of the commensal vaginal flora (3) and therefore might have been undetected in the vaginal discharge. Furthermore, several days before delivery, a urethral catheter was placed in the mother in order to enable complete bed rest and prevent premature delivery. Indwelling urethral catheters are often associated with nosocomial urinary tract infections caused by *Escherichia coli* or *P. aeruginosa* (4). In addition, the prolonged use of broad-spectrum antibiotics (3 weeks) and complications with insulin-treated diabetes mellitus might have rendered the mother more susceptible to resistant bacterial infection (5). We speculated that *P. aeruginosa* may have infected the urinary tract and spread from there to other genitourinary areas like the birth canal, resulting in the infection of the amniotic membrane after its protrusion into the vagina. Indeed, the day before delivery the mother showed some signs of urinary tract infection, including a mild fever, cloudiness of urine, leukocyturia (about 250 cells/μL), and microhematuria. However, a urine culture was not ordered. Shortening of the cervical canal or silent cervical dilatation with protrusion of the amniotic membrane into the vagina may also have led to a secondary intrauterine infection. In particular, a cervical length of less than 25 mm before 24 weeks gestation indicates a 51.1% probability of a positive amniotic fluid culture for microorganisms (6). Thus, an intraamniotic infection may be caused by the transmission of *P. aeruginosa* from the vagina.

*P. aeruginosa* prefers a moist environment with a high temperature (optimum temperature 37.0°C) and a wide pH range. Amniotic fluid provides a suitable environment due to the relatively high temperature and mildly alkaline environment containing fewer antimicrobial peptides (i.e., defensins, calprotectins, and bacterial/permeability-increasing protein) during early gestation (7). Given these conditions, amniotic fluid in early gestation may enhance the rapid growth of *P. aeruginosa*.

Bacterial folliculitis is generally caused by gram-positive bacteria (e.g., *Staphylococcus aureus*, coagulase-negative *staphylococci*) and occurs in areas with abundant hair follicles, such as the scalp, beard, axilla, buttocks, and extremities (8). However, in this case, the folliculitis occurred on the trunk, arm, and abdomen, which are typically areas with fewer hair follicles. *P. aeruginosa* is known to cause folliculitis owing to contact with contaminated circulated water in heated swimming pools, whirlpools, hot tubs, and foot tubs, as evidenced by its unique distribution (9–13). Circulating water contaminated by *P. aeruginosa* may become trapped, or enter the skin in occluded body sites, giving rise to folliculitis. The characteristic position of the fetus in utero may trap contaminated amniotic fluid on the trunk, arms, and abdomen, resulting in *P. aeruginosa* folliculitis. Indeed, *P. aeruginosa* folliculitis reportedly developed even on the abdominal skin, which typically has fewer hair follicles (9, 14).

In utero exposure to amniotic fluid contaminated by *P. aeruginosa* is likely to have caused the folliculitis in the present case. Congenital folliculitis may thus serve as an indicator of intraterine *P. aeruginosa* infection and serve as an indication for rapid treatment.

Acknowledgments We would like to thank Mr. JR Valera for his help with editing this manuscript. We would also like to thank Dr. Kenta Ito for his advice on the diagnosis and treatment of *P. aeruginosa* infections.

Conflict of interest None to declare.

REFERENCES