A case of congenital folliculitis caused by *Pseudomonas aeruginosa* in a preterm neonate

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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* (*P. 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.
Text

*P. aeruginosa* is a common pathogen in nosocomial infections and accounts for 4% of cases of late-onset neonatal sepsis (1). The mortality rate of *Paeruginosa* infection among neonates is reportedly as high as 56% (1). Thus 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 (FIRS). Thus it is important to detect and treat *Paeruginosa* infections in neonates as early as possible. Herein we report a case of congenital *P. aeruginosa* infection presenting numerous small red papules soon after birth, which was successfully treated with antibiotics. Written informed consent was obtained from the parents for the use of patient data in this case report.

An extremely preterm male neonate weighing 1030g was delivered at 27 weeks gestation 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 remarkable increased C-reactive protein). There was no significant family history of disease. The infant’s mother had gestational diabetes but no history of membrane rupture during pregnancy. At 23 weeks of pregnancy, she was hospitalized for the management of threatened premature delivery due to a cervical length of 15mm and cervical dilatation with protrusion of the membrane into the vagina. A maternal vaginal culture obtained at 26 weeks of pregnancy showed the presence of *Candida glabrata*, *Citrobacter koseri*, *Gardneralla vaginalis*, and *Enterococccus* spp. but not of *P. aeruginosa*. Both cefmetazole and sulbactam/ampicillin were administered to the mother in order to
prevent bacterial infection for three 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 a 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 (Figure 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/mm$^3$), suggesting the presence of a neonatal bacterial infection. Intravenous ampicillin and gentamicin were started empirically. The skin lesions completely resolved only two days after birth. On postpartum day 3, $P. \text{aeruginosa}$ was detected in all of the cultures except the blood culture, including the tracheal aspirate, gastric aspirate, pharynx, skin, stool, and papule cultures. Besides $P. \text{aeruginosa}$, no bacterium or fungus was detected in any of the cultures. Based on these findings folliculitis due to $P. \text{aeruginosa}$ 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.

$P. \text{aeruginosa}$ rarely causes intrauterine infections. However, congenital $P. \text{aeruginosa}$ infections can lead to serious complications including sepsis and pneumonia, and may have neurological sequelae or result in neonatal death.
How the neonate in the present case was infected with *P. aeruginosa* remains 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 gone undetected in 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 (three 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 per µL), and micro hematuria. 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 lead 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, bactericidal/permeability-increasing protein) during early gestation (7). Given these conditions, amniotic fluid in early gestation may enhance rapid growth of *P. aeruginosa*.

Bacterial folliculitis is generally caused by gram positive bacteria (i.e., *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, typically areas with fewer hair follicles. *P. aeruginosa* is known to cause folliculitis by contaminating 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 and so give 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 intrauterine *P. aeruginosa* infection and serve as an indication for rapid treatment.
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References


Figure legend:

Figure 1. Many small red papules can be seen covering the patient’s body (A), including the lateral and lower regions of abdomen (B), axilla, and inguinal areas.