Intrapartum leprosy

Madeline Smoot, BS^a , Randy Volkmer, BS^a, Casey Roth, MD^b, and Brandon Cornelius, MD^b

^aTexas A&M College of Medicine, Dallas, Texas; ^bDepartment of Obstetrics and Gynecology, Baylor University Medical Center, Dallas, Texas

ABSTRACT

Leprosy is caused by an infection with *Mycobacterium leprae*, an acid-fast, slow-growing, obligate intracellular rod-shaped bacillus. Intrapartum leprosy most commonly occurs in the third trimester of pregnancy. This is likely due to pregnancy decreasing cellular immunity, thus allowing the organism to proliferate unchecked. This case report reviews the current understanding of intrapartum leprosy and details the hospital course of a patient who presented with active intrapartum leprosy at 32 weeks gestation.

KEYWORDS Intrapartum leprosy; lepromatous leprosy; systemic lupus erythematosus; transplacental transmission

In 2015, 210,758 new cases of Hansen's disease were documented worldwide, with the greatest number of cases in India, Brazil, and Indonesia.¹ Of this number, only about 150 were diagnosed in the US.¹ Leprosy is caused by an infection with *Mycobacterium leprae*, an acid-fast, slow-growing, obligate intracellular, rod-shaped bacillus. There are two forms of the disease, paucibacillary and multibacillary. The paucibacillary form is clinically characterized by small numbers of hypopigmented, well-circumscribed skin lesions with few bacilli within the lesion. Among patients in remission, leprosy can reappear in the third trimester of pregnancy,² likely because pregnancy decreases cellular immunity, which allows the organism to proliferate unchecked.³ The method of leprosy transmission has not yet been determined and, as a result, many previous cases of intrapartum leprosy have been delivered via cesarean. There has been concern that the disease could be transmitted during delivery with skin-to-skin contact or droplet transmission with the mom.⁴

CASE PRESENTATION

A 29-year-old gravida 3, para 0, aborta 2 woman from Micronesia at 32 weeks gestation with no prenatal care presented to the emergency department complaining of an itchy, bleeding rash that had spread all over her body. There were no sensory changes at the site of the lesions other than pruritus. She had emigrated from Micronesia in 2013 and had two prior pregnancies that both ended in third-trimester

stillbirths, one at 7 months and the second at 8 months. In both pregnancies, she experienced the same type of rash with similar associated symptoms.

Raised boils with bloody drainage, without pus, were present on the arms, legs, and abdomen. Her hemoglobin was 9.6 g/dL, and her white blood cell count was 6.5 K/ μ L. Ultrasound of the abdomen showed a single, normal intrauterine pregnancy without any placental or uterine abnormalities. The lupus anticoagulant and the immunoglobulin G and immunoglobulin M anticardiolipin were positive and consistent with a diagnosis of systemic lupus erythematosus. She was given aspirin and enoxaparin for her autoimmune disorder. A biopsy of one of the skin lesions showed diffuse granulomatous infiltrates with enlarged epithelioid histiocytes. Use of Gram stain on this sample resulted in no stain retention by the organisms. Use of acid-fast stain on this same sample of skin displayed numerous bacilli within the granulomas (*Figure 1*). The patient was started on 600 mg of rifampin and 100 mg of dapsone once daily. An attempt was made to start the patient on clofazimine from the health department. This was not obtained and the patient was maintained on the two-drug regimen. Rifampin was discontinued on posthospital day 16. She was discharged home on hospital day 12 with directions to continue to take the dapsone.

Labor was induced at 37 weeks gestation, secondary to her autoimmune history and history of two prior term demises, resulting in a successful vaginal delivery of a 2.590 g

Corresponding author: Brandon Cornelius, MD, Department of Obstetrics and Gynecology, Baylor University Medical Center, 3500 Gaston Ave., Dallas, TX 75246 (e-mail: Brandon.Cornelius@BWSHealth.org)

Color versions of one or more of the figures in this article can be found online at www.tandfonline.com/ubmc.

Received March 3, 2019; Revised May 1, 2019; Accepted May 6, 2019.

Figure 1. Acid-fast bacilli staining: (a) positive in the patient's arm lesion and (b) negative in the placenta.

baby boy. The placenta was negative for *Mycobacterium* infiltration (Figure 1b). Other than a total bilirubin of 10.8 mg/dL,

the infant was healthy and had no lesions on his skin. The patient and her child were discharged with instructions to follow up with the child's pediatrician in 2 days and to continue routine care of the infant, including breastfeeding.

DISCUSSION

This patient presentation allows further understanding of how Hansen's disease presents during pregnancy. Additionally, it points to the need for research to determine whether the child will develop the disease in adolescence. This case demonstrated no transplacental transmission, because the placenta tested negative for *M. leprae*. The treatment regimen of rifampin and dapsone provided adequate control of maternal disease during gestation. This case report provides an example of successful management during pregnancy and may be of use in clinical application among those populations.

ORCID

Madeline Smoot <http://orcid.org/0000-0002-2791-9530>

1. Fischer M. Leprosy—an overview of clinical features, diagnosis, and treatment. *J Dtsch Dermatol Ges.* 2017;15:801–827.
2. Duncan ME. An historical and clinical review of the interaction of leprosy and pregnancy: a cycle to be broken. *Soc Sci Med.* 1993;37:457–472. doi:10.1016/0277-9536(93)90281-8.
3. Lockwood DN, Sinha HH. Pregnancy and leprosy: a comprehensive literature review. *Int J Lepr Other Mycobact Dis.* 1999;67:6–12.
4. Gimvosky AC. Leprosy in pregnant woman, United States. *Emerg Infect Dis.* 2013;19:1693–1694. doi:10.3201/eid1910.130463.