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PUBLIC HEALTH ALERTS | IN PARTNERSHIP WITH CIDRAP

Paenibacillus dendritiformis as a Cause of Destructive Meningitis in Infants

Danielle Smith, M.D.,[1] Rupa Garikipati, M.D.,[1] Kristen Bastug, M.D.,[2] James R. Broach, Ph.D.,[3] Lisa Schneper, Ph.D.,[3] Kathryn Sheldon, Ph.D.,[3] Jasmine Smith,[3] Marwan Osman, Ph.D.,[4,5] Steven J. Schiff, M.D., Ph.D.,[4,5] and Jessica E. Ericson, M.D., M.P.H.[1]

Abstract

Invasive infections due to *Paenibacillus* species pose a serious risk to young infants and have a high risk of neurologic sequelae. This report describes two infants with severe neurologic manifestations secondary to *Paenibacillus dendritiformis* infection who were recently identified in the United States. Clinicians who care for young infants should be aware of this emerging infection.

The Public Health Alerts section in *NEJM Evidence* is in partnership with the Center for Infectious Disease Research and Policy (CIDRAP) at the University of Minnesota.

Introduction

Invasive infections due to *Paenibacillus* species pose a serious risk to young infants and have a high risk of neurologic sequelae. Following reports of Ugandan infants with infections caused by *Paenibacillus* species,[1,2] similar cases have now been recognized in multiple U.S. states.[3] In this article, we describe two infants with severe neurologic manifestations secondary to *Paenibacillus dendritiformis* infection who were recently identified in the United States. Clinicians who care for young infants should be aware of this emerging infection, as empiric antibiotic regimens for treating bacteremia and meningitis are likely inadequate, and neurosurgical intervention is often needed.

Investigation and Outcomes

A 2-month-old female infant born at 26 weeks' gestation in February 2025 in eastern Pennsylvania developed respiratory distress and seizures. Her previous course in the neonatal intensive care unit (NICU) had been uneventful with no neurologic procedures. Gram-negative rods were identified on blood culture, and blood and cerebrospinal fluid (CSF) cultures grew *Paenibacillus thiaminolyticus,* based on matrix-assisted laser desorption ionization time of flight mass spectrometry (MALDI-TOF MS) analysis. No other pathogens were identified. Brain imaging showed progressive hydrocephalus, encephalomalacia, and abscesses, which required placement of a ventriculoperitoneal shunt (Fig. 1). She was treated with continuous-infusion meropenem for 8 weeks, with vancomycin and rifampin added midway through the treatment course due to ongoing abnormalities of her CSF. She also received supplemental thiamine starting 4 days after symptoms began. At age 8 months, she was able to make eye contact and smile and had no apparent seizures; however,

The author affiliations are listed at the end of the article.

Jessica E. Ericson can be contacted at jericson@pennstatehealth.psu.edu

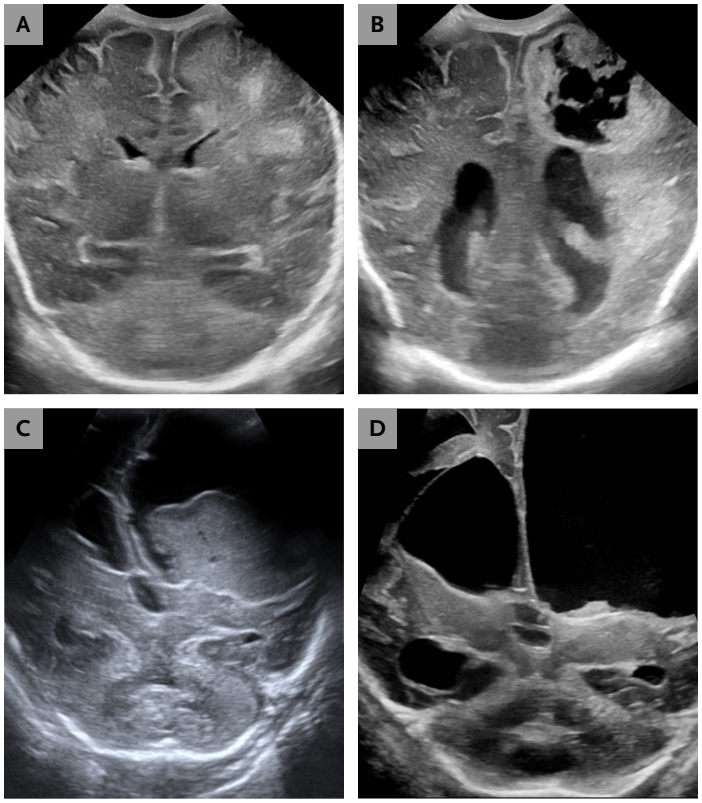

Figure 1. Representative Brain Ultrasound Images from an Infant with *Paenibacillus dendritiformis* Meningitis Cared for at Penn State Health Children's Hospital.

Serial brain ultrasounds demonstrate progression from a subtle area of increased echogenicity on the 2nd day of illness (Panel A) to a circumscribed brain abscess on the 7 th day of illness (Panel B) to bilateral parenchymal destruction and resulting ex-vacuo and obstructive hydrocephalus on the 11th (Panel C) and 25th (Panel D) days of illness.

she was unable to eat by mouth, sit unsupported, or roll independently.

A similar case was previously reported in Minnesota in a 37-day-old male infant born at 33 weeks' gestation who had been doing well following a 22-day NICU stay and 15 days at home.[4] He returned to the hospital with poor feeding and unresponsiveness. Blood and CSF cultures grew *P. thiaminolyticus* as identified by MALDI-TOF MS analysis. No additional pathogens were identified. Brain imaging demonstrated "liquefactive meningoencephalitis." He was treated with 21 days of intravenous ampicillin and required placement of a ventriculoperitoneal shunt. He developed feeding problems and seizures and died at 11 months of age.

Although *P. thiaminolyticus* was identified by MALDI-TOF MS in both clinical laboratories, whole-genome sequencing of isolates from both infants identified *P. dendritiformis*. These isolates had genes encoding the type IV pilus operon, multiple β-lactamases, vancomycin resistance genes, and thiaminase 1. The type IV pilus has previously been identified as an important virulence factor in neonatal paenibacilliosis.[5]

Preliminary Conclusions and Actions

Paenibacillus infection in infants is an emerging concern with devastating consequences; however, the modes of transmission are unclear. Both infants reported here were born preterm, requiring care in a NICU. Other cases of neonatal paenibacilliosis in the United States have occurred in infants with NICU admission and in infants with maternal drug use.[2,3] Members of the *Paenibacillus* genus are commonly identified as part of the soil microbiome, but significant exposure to soil in these patients seems unlikely.[4,6,7-9] An association with rainfall and proximity to large bodies

of water has been noted in Ugandan infants[2]; however, risk factors for infant infection in the United States may be different from those of Ugandan infants.

Paenibacillus are spore-forming gram-positive species that frequently stain gram-variable, a finding that may contribute to delays in diagnosis. MALDI-TOF MS can reliably identify infections as belonging to the *Paenibacillus* genus but cannot accurately distinguish between *P. thiaminolyticus* and *P. dendritiformis*. This distinction is unlikely to be clinically important given that virulence factors and antimicrobial resistance determinants appear to be conserved between the two species. *Paenibacillus* species are facultative anaerobes, a feature that likely contributes to the difficulty growing them in culture, especially in low-resource settings.

Optimal antibiotic therapy is unknown, but narrow-spectrum β-lactams should be used with caution due to the presence of multiple β-lactamase genes. Ugandan isolates have had vancomycin resistance genes (*VanW*); U.S. strains have been phenotypically susceptible to vancomycin, but also harbor these genes. Some U.S. infants have been treated with ampicillin, but among reported cases, those with the best outcomes were treated with meropenem and thiamine.[10] Early thiamine supplementation may be helpful, as both *P. thiaminolyticus* and *P. dendritiformis* produce a thiaminase,[5] and it is possible that some of the brain destruction is due to a local thiamine deficiency in the brain tissue in addition to the direct effects of the infection.[10]

Clinicians who care for young infants should be aware of this emerging pathogen, as empiric antibiotic regimens for treating bacteremia and meningitis may be inadequate, and pediatric neurosurgical expertise for abscess drainage or treatment of hydrocephalus is typically needed.

Disclosures

Author disclosures are available at evidence.nejm.org.

Author Affiliations

[1] Department of Pediatrics, Penn State College of Medicine, Hershey, PA, USA

[2] Department of Pediatrics, University of Minnesota, Minneapolis, MN, USA

[3] Department of Molecular and Precision Medicine, Penn State College of Medicine, Hershey, PA, USA

[4] Department of Neurosurgery, Yale University School of Medicine, New Haven, CT, USA

[5] Department of Epidemiology of Microbial Diseases, Yale University School of Public Health, New Haven, CT, USA

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