The pediatric team is called to the vaginal delivery of a 31-year-old gravida 3, para 2-0-1-2 mother at 40 weeks and 1 day of gestation because of a category II tracing. The pregnancy is uncomplicated, including a negative group B Streptococcus test result, and clear amniotic fluid after artificial rupture of membranes 4.5 hours before delivery. Nuchal cord/C2 is noted at the delivery of a female infant with 1- and 5-minute Apgar scores of 8 and 9.

Examination reveals a birthweight of 3,625 g, caput over the left occiput without bruising, generalized edema and bruising of the frontal scalp (Fig 1A). The infant exhibits discomfort with care. She is neurologically normal and breastfeeds well. Phototherapy is begun for a total bilirubin of 8.7 mg/dL (148.77 μmol/L) 25 hours after birth.

On day 2 after birth, she becomes more irritable and less active, with mildly decreased tone and a weak cry. A sepsis evaluation is performed, and treatment is started with ampicillin and gentamicin.

The initial white blood cell count is 33,000/μL (3×10⁹/L) with an absolute neutrophil count of 1,000/μL (1×10⁹/L), and no bands. Blood culture is positive for Escherichia coli at 8 hours on polymerase chain reaction (PCR)–based blood culture identification panel. Cerebrospinal fluid (CSF) is clear, with glucose of 76 mg/dL (4.22 mmol/L), protein 71 mg/dL (0.71 g/L), white blood cells 10/μL (10/10⁶L), and red blood cell count of 0. Gram stain and PCR-based meningitis/encephalitis panel are negative. Antibiotic therapy is broadened to include ceftazidime, and the neonate is transferred to the NICU.

Upon arrival in the NICU, the neonate is well appearing and alert. The anterior fontanelle is soft and flat, the scalp has scattered bruising, diffuse edema, and a 3.0×3.0-cm triangular dark crusted macule slightly and uniformly depressed on the right posterior occiput (Fig 1B). A complete review of the obstetrics records reveals delivery in occiput posterior position and application of fetal scalp electrodes (FSE). Blood culture from day 2 after birth confirms pan-susceptible E coli. Repeat blood culture and CSF and urine culture remain sterile. On day 4 after birth, the absolute neutrophil count is 4,900/μL (4.9×10⁹/L) and the C-reactive protein (CRP) reaches 31.3 mg/dL (313 mg/L). Antibiotic treatment continues with ampicillin monotherapy.

AUTHOR DISCLOSURES Drs Dias Maia, Niermeyer, Palau, and Cataldi have disclosed no financial relationships relevant to this article. This commentary does not contain a discussion of an unapproved/investigative use of a commercial product/device.
On day 6 after birth, the infant develops a fever to 101.1°F (38.4°C). A third blood culture and blood specimen for herpes simplex virus (HSV) PCR are sent, and treatment is broadened to cefazidime and acyclovir. On day 7, the scalp exhibits indurated erythema around the dark crusted macule (Fig 1C) and CRP is 5.5 mg/dL (55 mg/L). On day 8, the infant remains febrile and the CRP level rises to 8 mg/dL (80 mg/L). The scalp shows worsening erythema, and the indurated area becomes fluctuant (Fig 1D). Needle aspiration yields 30 mL of purulent serosanguinous fluid from 2 puncture sites. The pediatric surgeon enlarges both puncture sites to 3 to 4 mm and irrigates the wound. Aerobic and anaerobic culture specimens are sent and antibiotic coverage is broadened to vancomycin and meropenem.

**DISCUSSION**

**Diagnosis/Management**
The infant remained clinically and hemodynamically stable. Blood cultures and HSV PCR did not yield any organism. Acyclovir was discontinued. *E. coli* was isolated from the purulent fluid and showed the same antibiotic sensitivity as the isolate from blood culture, so antibiotic coverage reverted to ampicillin alone. Brain magnetic resonance imaging revealed a subgaleal posterior scalp abscess with no intracranial or intraspinal extension (Fig 2). Echocardiography showed no evidence of vegetation. During hospitalization, the CRP normalized and neutropenia resolved, but direct hyperbilirubinemia prompted abdominal ultrasonography, which showed normal results. Thrombocytopenia with normal coagulation profile required multiple platelet transfusions. The first newborn screen was negative for galactosemia but flagged for severe combined immunodeficiency which subsequently was negative on the second newborn screen. TBNK and T-cell–naive/memory panels were normal. The patient remained afebrile and continued to improve clinically (Fig 3A,B), though prolonged drainage was noted from the surgical sites (Fig 3C,D). She was treated with ampicillin until 35 days after birth, when she was

Figure 1. A. Initial physical examination: generalized edema and bruising of the frontal scalp and forehead. B–D. Evolution of the scalp lesion before surgical intervention.

Figure 2. Magnetic resonance imaging scan of the brain with and without contrast showing a subgaleal posterior scalp abscess with no calvarial signal abnormality (white arrows) and no evidence of intracranial extension.
discharged without oral antibiotics after the scalp wound and drainage showed significant improvement (Fig 3E).

The Condition
Early-onset sepsis remains a significant cause of neonatal morbidity and mortality. *E. coli* is the second leading cause of early-onset sepsis in neonates and is the leading bacterial cause in preterm infants, with a 15% to 40% overall case fatality (1)(2) and rising incidence rate. (3) The clinical features of neonatal sepsis can be subtle and nonspecific, as exhibited by this patient.

Most of these infections occur from vertical transmission of bacteria from the mother during the intrapartum period, through placental transmission after maternal bacteremia, or through neonatal colonization during passage through the birth canal. Factors increasing the risk for *E. coli* infection include low gestational age, maternal fever, prolonged rupture of membranes, meconium-stained fluid, and maternal urinary tract infection. (2) However, this patient had none of these risk factors.

In the literature, there is a paucity of data on FSE application and risk for early-onset sepsis. (2) An association has been described sporadically in case reports, and a small study of 15 culture-proven cases found that prolonged application of FSE (>12 h) was an independent risk factor for early-onset sepsis. No significant association between the use of FSE and early-onset sepsis in a population of term and preterm infants was found in a case-control study that compared 40 neonates who subsequently developed sepsis with 80 controls. (4)

On the other hand, scalp abscesses are acknowledged complications of FSE monitoring, with a reported incidence of 0.1% to 5.2%. They typically develop 2 to 10 days after delivery, and most of the lesions are localized and self-limited. The microbiology of the abscess has been described as a mixture of aerobic and anaerobic vaginal bacterial flora including *E. coli*. (5)(6)

We report a case of *E. coli* early-onset sepsis in a term neonate, without evidence of immunodeficiency or galactosemia. The course was complicated by a sizable subgaleal scalp abscess in the setting of occiput posterior position and intrapartum FSE use. The extensive bruising and caput were evident on initial presentation, but the scalp infection remained subclinical until later in the course, similar to 2 previously described cases. (5)(7)

In our case, it remains unclear whether the scalp became infected during the primary *E. coli* bacteremia by hematogenous seeding, by invasion through the scalp monitor site, or via skin compromised by pressure injury. (8) The scalp edema and localized necrosis from the birth trauma likely contributed to the local infection risk.

Lessons for the Clinician
1. The recognition of subtle and nonspecific clinical changes should raise suspicion of neonatal sepsis in the nursery and prompt early detection and timely intervention.
2. Repeated physical examination can yield surprising and unexpected findings in cases of early-onset sepsis.
3. Carefully documented history can illuminate potential causality for such unexpected findings.

ACKNOWLEDGMENT
We acknowledge the contributions of the Denver Health and the Children’s Hospital Colorado NICU teams in the management of this case.

American Board of Pediatrics
Neonatal-Perinatal Content
Specifications
- Know how infectious agents are transmitted to the neonate.
- Know the clinical manifestations, laboratory features, and differential diagnosis of neonatal sepsis.
- Understand the treatment and complications of sepsis.
- Know the infectious agents that cause neonatal sepsis.
- Know the maternal, perinatal, and neonatal risk factors for neonatal sepsis.
References


Case 2: Early-Onset Neonatal Sepsis in a Term Neonate
Paula Dias Maia, Susan Niermeyer, Mauricio A. Palau and Jessica R. Cataldi
NeoReviews 2021;22;e402
DOI: 10.1542/neo.22-6-e402
Case 2: Early-Onset Neonatal Sepsis in a Term Neonate
Paula Dias Maia, Susan Niermeyer, Mauricio A. Palau and Jessica R. Cataldi
NeoReviews 2021;22:e402
DOI: 10.1542/neo.22-6-e402

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://neoreviews.aappublications.org/content/22/6/e402