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CASE REPORT

# Neonatal Infected Subgaleal Hematoma: An Unusual Complication of Early-onset *E. coli* Sepsis



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PEDIATRICS and NEONATOLOG

Hung-Yang Chang <sup>a,b,\*</sup>, Kun-Shan Cheng <sup>a</sup>, Yu-Peng Liu <sup>c</sup>, Hsiao-Fang Hung <sup>b,d</sup>, Hua-Wen Fu <sup>d</sup>

<sup>a</sup> Department of Pediatrics, Mackay Memorial Hospital, Hsinchu Branch, Hsinchu, Taiwan

<sup>b</sup> Department of Medical Technology, Jen-Teh Junior College of Medicine, Nursing and Management, Miaoli, Taiwan

<sup>c</sup> Department of Radiology, Mackay Memorial Hospital, Hsinchu Branch, Hsinchu, Taiwan <sup>d</sup> Institute of Molecular and Cellular Biology, Department of Life Science, National Tsinghua University,

Hsinchu, Taiwan

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Key Words infected subgaleal hematoma; neonatal sepsis; newborn Subgaleal hematoma (SGH) is an uncommon but potentially lethal medical emergency in newborns. Delay in diagnosis may lead to mortality and morbidity. Infection of an SGH is extremely rare. We report an infected SGH with abscess formation as a complication of early-onset *Escherichia coli* sepsis in a term neonate. The patient was discovered to have SGH soon after birth. Early-onset *E. coli* sepsis developed on Day 3 of life. The SGH became infected, with abscess formation 1 week later. The infected SGH was probably due to direct hematogenous spreading of sepsis. The patient was successfully treated without complications. Clinicians should be aware that SGH is a potential site of infection and infection may be caused either by direct hematogenous extension or from traumatic scalp lesions. Appropriate antibiotic treatment and surgical debridement are necessary when an infected SGH occurs. Copyright © 2013, Taiwan Pediatric Association. Published by Elsevier Taiwan LLC. All rights reserved.

\* Corresponding author. Department of Pediatrics, Mackay Memorial Hospital, Hsinchu Branch, 30071, Number 690, Section 2, Guangfu Road, Hsinchu City, Taiwan.

E-mail address: a4583@ms7.mmh.org.tw (H.-Y. Chang).

### 1. Introduction

Neonatal sepsis remains a potentially lethal condition, especially in preterm neonates. Early-onset neonatal sepsis is associated with acquisition of microorganisms from the

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mother. Escherichia coli is well established as a leading cause of neonatal sepsis. Risk factors increasing chances of E. coli infection include low gestational age, intrapartum fever, and prolonged rupture of membranes.<sup>1</sup> Infection may not be only in the bloodstream, but can also spread to the lungs, brain, bones, joints, soft tissues, and other organs in the body. Subgaleal hematoma (SGH) (also known as subaponeurotic hemorrhage or subgaleal hemorrhage) is a potentially lethal medical emergency that results from the rupture of the emissary veins. Blood collects in the space between the galea aponeurotica and the periosteum of the skull. Massive hemorrhages may lead to serious complications or death. However, bacterial infection of a neonatal SGH is extremely rare. The route of infection may be direct hematogenous seeding of bacteremia or invasion through scalp monitor or wounds. We report a case of an infected SGH with abscess formation following early-onset E. coli sepsis in a term infant.

### 2. Case report

A term female neonate was born by spontaneous vaginal delivery to a 33-year-old prim gravid mother. The infant's birth weight was 3990 g, and APGAR scores were 8 and 9 at 1 and 5 minutes, respectively. During delivery, scalp monitor, forceps, and vacuum were not employed, and there was no scalp wound noted after birth. A large scalp hematoma over the parietal and occipital area was noted soon after birth. On Day 2, the hematoma extended to the bilateral mandibular area and an SGH was diagnosed. Hemoglobin level, coagulation profile, blood pressure, and other vital signs were within normal limits. However, fever developed on Day 3 of life, and a complete septic workup was performed. The patient's C-reactive protein (CRP) was 17.4 mg/dL. Ampicillin (100 mg/kg/dose every 12 hours) and gentamicin (2.5 mg/kg every 24 hours) were administered intravenously. On Day 5 of life, a Gram-negative rod was recovered from the blood culture, and was subsequently identified as E. coli and found sensitive to ampicillin and gentamicin. The patient's fever persisted intermittently until Day 10 of life. In addition, the previous SGH site over the bilateral parietal and mandibular area became red and tender, and pus began to form gradually

(Figure 1). The infant began to experience respiratory distress and tachycardia and was irritable, with excessive crying whenever the SGH was touched. Approximately 10 mL of vellowish pus was aspirated from the bilateral mandibular area on Day 14 of life. The organism isolated from the pus was verified as E. coli, and showed the same antibiotic sensitivity as the isolate from the blood culture. Computed tomography (CT) revealed diffuse abscess formation over the bilateral parietal, occipital, bilateral mandibular, and neck regions (Figure 2). Surgical incision and drainage were repeated three times afterwards until the size of the infected hematoma and abscess decreased. CRP returned to normal, and she was discharged after treatment with ampicillin for 3 weeks. The patient was followed up at clinics and showed normal neurologic development.

### 3. Discussion

SGH occurs most commonly after vacuum delivery. Other reported risk factors for SGH include macrosomia, prematurity, primigravida delivery, dystocia, and precipitous labor. Massive hemorrhage may extend from the orbital ridges to the nape of the neck. The most common findings of SGH are increasing head circumference, ecchymosis of the hematoma, respiratory distress, tachycardia, poor activity, pallor, and jaundice.<sup>2</sup> Progression of SGH may be associated with serious complications and death. Complications associated with SGH include anemia, jaundice, hypovolemic shock, renal failure, and neurologic morbidity. Bacterial infection in an SGH is extremely rare. Currently, only two cases of infected SGH in neonates have been reported; neither of these cases was associated with sepsis.<sup>3,4</sup>

The common risk factors for infection of the scalp area in a newborn include electrode insertion during fetal monitoring, traumatic scalp lacerations, needle aspiration of a hematoma, sepsis, or meningitis. The relatively immunocompromised condition and the increased laxity of the loose connective tissue of the subgaleal space in a newborn may contribute to the spread of infection. It's difficult to determine whether an infected SGH is the source of a primary infection or a second infection



**Figure 1** Elongation and increasing head circumference were consistent with clinical findings on subgaleal hematoma (white arrow). The bilateral parietal, mandibular, and post-auricular area became red, tender, and pus formed after sepsis (black arrows).



**Figure 2** (A) Head computed tomography revealed abscess formation over bilateral mandibular and neck regions (white arrows). (B) Hematoma over the bilateral parietal scalp was discovered (black arrows). The wall enhancement of the hematomas was consistent with secondary infection.

following another infection. Our patient developed localized symptoms of infected SGH 1 week after the onset of *E. coli* sepsis. Therefore, in the present case, the SGH became infected due to hematogenous spreading of *E. coli* sepsis.

Several conditions should be differentiated from neonatal infected SGH, including infected cephalohematoma, subgaleal abscess, and scalp abscess. Cephalohematoma is a subperiosteal hemorrhage and is generally a benign condition. Infected cephalohematoma is not uncommon, and E. coli infection has been reported as the leading pathogen.<sup>5</sup> Subgaleal abscess is pus accumulated in the subgaleal space due to progression of other infection.<sup>6,7</sup> It can occur without the presence of SGH and has been associated with meningitis, surgery, trauma, or sinusitis in adults.<sup>8</sup> However, infected SGH may develop into subgaleal abscess as the infection process progresses. Scalp abscess is a solitary infection that is primarily limited to the scalp tissue. Although infected cephalohematoma, subgaleal abscess, and scalp abscess differ from each other structurally, they share the same risk factors and common pathogens as infected SGH.

In newborns with suspected sepsis, a complete sepsis workup should be performed. A diagnostic tap and culture should be carried out in patients with local infectious signs of SGH, including erythema, tenderness, and pus formation. The antibiotic treatments and duration for treating infected SGH have not been well documented. Treatment is mainly based on antibiotic sensitivity results derived from pus cultures and clinical improvements in clinical practice. A CT scan is often required to determine the size of infection or abscess formation. CT can reveal the amount of hematoma, the degree of bone destruction, and the extent of intracranial injury. CT should also be performed if other complications, such as osteomyelitis and subdural empyema, are suspected. For these scenarios, or if the clinical condition does not improve with antibiotic therapy, repeat surgical incision, drainage, and debridement are often required.

In conclusion, clinicians should be aware that SGH is a potential site of infection and may be secondary to systemic infection. Appropriate diagnostic and therapeutic measures should be promptly implemented. Proper image studies, surgical debridement, and prolonged antibiotics are essential to the successful treatment of an infected SGH.

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