

SPONTANEOUS INFECTION OF A CEPHALOHEMATOMA ASSOCIATED WITH SEPSIS: A CASE REPORT AND REVISION OF THE LITERATURE

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Introduction

Cephalohematoma is one of the most common birth injuries and consists of a subperiosteal collection of blood, resulting from the rupture of the superficial veins between the skull and the periosteum. It is found in 2.5% of all live births. It is more commonly seen in instrumented deliveries, occurring in 1% to 2% of spontaneous vaginal deliveries, 6% to 10% of vacuum-assisted deliveries and 4% of forceps-assisted deliveries (1). It generally progresses to complete resorption within 2-8 weeks. The initial size conditions the resorption time. In cases of prolonged resorption, over more than one month, cephalohematomas begin to calcify. The most frequent com-

Objective – This case report presents a child born at 41 weeks gestational age with a cephalohematoma that spontaneously evolved into abscess. **Case report** – We report the case of a newborn Chinese boy, born at 41 weeks by a spontaneous delivery, presented on the 18th day of life with a soft-tissue parietal-temporomandibular mass that spontaneously evolved into abscess. Surgical incision and cleaning of the abscess cavity were performed and systemic antibiotic therapy was carried out with resolution of the infection. The fluid and blood cultures grew *Escherichia coli*. **Conclusions** – Infected cephalohematoma requires all microbiological and biochemical laboratory exams to rule out a systemic associated infection. Double antibiotic therapy should be started immediately.

Key words: Cephalohematoma ■ Birth trauma ■ Sepsis ■ Newborn infants.

plications are anemia, hyperbilirubinemia, fibrous organization, calcification and skull fracture. Infection is a rare complication, usually resulting from the incision of cephalohematoma to aspirate blood collection. Spontaneous infection is extremely rare, but can lead to septicaemia and meningitis.

We describe a child of Chinese nationality, born at 41 weeks' gestational age with a cephalohematoma that spontaneously evolved into abscess.

Case report

An at term Chinese boy was born by a prolonged spontaneous labor, without the assistance of vacuum extraction or forceps and

with no scalp monitoring. The maternal pregnancy was uneventful, without any fever. The rupture of the membranes occurred spontaneously less than 12 hours prior to delivery. At birth the baby presented mild asphyxia. The Apgar scores were 6, 7 and 8 at 1, 5 and 10 minutes respectively. Resuscitation was not necessary. The birth-weight was 4100 g (90th percentile), length was 56 cm (97th percentile) and head circumference was 38.5 cm (97th percentile). The baby was discharged on day of life (DOL) 3 with a large, left parietal mass, diagnosed as cephalohematoma, no fever and no jaundice.

On DOL 18 he was admitted to the emergency department due to enlargement of the mass. Skull radiography at that time (Fig. 1) showed no fractures; the baby was otherwise healthy and was discharged. Three days later he was readmitted to the hospital, because the swelling had increased in size, measuring 19 cm in width and 17 cm in height, and it extended into the left parietal temporo mandibular area of the skull. The skin overlying the mass appeared hyperemic, warm and was surmounted by a pustular lesion.

Laboratory tests demonstrated white blood cells 18,800/mm³ (28% neutrophils, 49% lymphocytes and 19% monocytes), hematocrit 33% and platelets 110,000/mm³. C reactive protein was 9 mg/dl (n.v. less than 0.5 mg/dl). Blood culture, urinalysis and urine culture were done. Cerebrospinal fluid (CSF) samples were obtained for bacterial culture. CSF cells count and chemistry and were not suggestive for meningitis. Suspecting an abscess, antibiotic therapy was started: teicoplanin 10 mg/kg 12 hrly for the 1st 3 doses, then 10 mg/kg/die and amikacin 10 mg/kg/once daily dosage. The abscess evolved into spontaneous rupture on the day after the admission and 400 ml of purulent material was drained. Surgical incision and cleaning of the abscess cavity were then performed. The TC scan of the skull ruled out the pres-

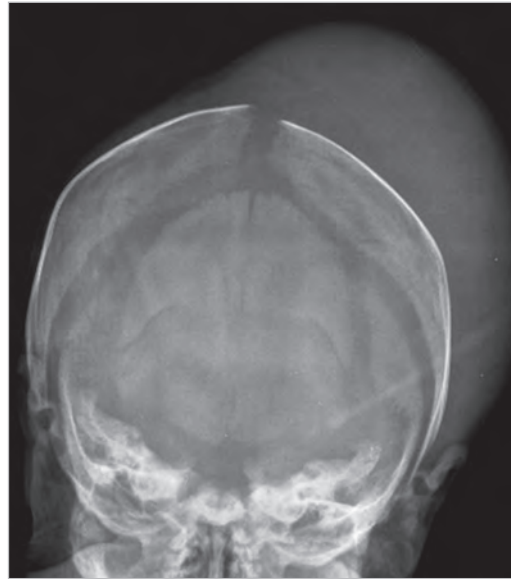


Fig. 1 X-ray of the skull showing significant soft swelling without underlying bone lesions.

ence of osteomyelitis. The blood culture and the culture of purulent material grew *Escherichia coli*. The cultures from other sites were negative. Antibiotics were administered until 15 days iv and then oral amoxicillin 50 mg/kg/die was given for a further 7 days, with complete recovery.

Discussion

Our patient was suffering from a spontaneous *Escherichia coli* infection of a parietal cephalohematoma, that had never been incised for blood aspiration. This is a rare and dangerous event, following prolonged spontaneous labour, that can happen even without the use of surgical instruments. Although rare, cases of infected cephalohematomas are described in the literature, with possible complications such as meningitis or osteomyelitis. Recently, Van Helleputte (2) reported a case of infected cephalohematoma with secondary osteomyelitis due to a wound caused by foetal monitoring, that required drainage and long term antibiotic therapy. In 1993, Blom and

Vreede (3) reviewed 27 cases occurring between 1818 and 1993; the first 2 cases (in 1818 and 1907) were complicated by incision of the cephalohematoma, the others were consequent to well known risk factors such as aspiration of blood, scalp infection, forceps delivery and sepsis. Cases reported after 1975 were related to the use of electrode foetal monitoring.

In non-instrumental, prolonged labours cephalohematoma is associated with cephalopelvic disproportion or with a deviant position of the head (occipital posterior, occipital transverse). These risk factors contribute to the impact of the head against the pelvic bones during the labour. The continuous trauma of the skull, compressed against the mother's bone, generates the lesion of the nutritive vessels of the periosteum, with bleeding into the subperiosteal layer of the skull. The bleeding lifts the periosteum away from the skull. In the case of forced delivery or vacuum extraction, subperiosteal hematoma usually results in two soft masses at the vertex of the head, separated by a deep sulcus along the sagittal suture.

Cephalohematoma generally does not cross cranial sutures. Only three cases have been described in the literature where the lesion crosses the sagittal suture (4-6). They have to be differentiated as caput succedaneum and subgaleal hematoma. Caput succedaneum is defined as a collection of serosanguineous fluid between the scalp and the epicranial aponeurosis. It generally crosses the cranial sutures. Generally it is evident immediately after delivery, on the vertex of the head. It has a soft, boggy feel with irregular margins, and it may have haemorrhagic manifestations (petechiae, purpura, ecchymosis). A subgaleal hematoma is a collection of blood between the epicranial aponeurosis and the periosteum. It generally crosses the sutures, such as the caput succedaneum. It is potentially life threatening (7). The most

frequent complications of cephalohematoma are shown in Box 1.

Box 1 Complications of cephalohematoma

- Skull fractures: 5% of unilateral and 18% of bilateral cephalohematoma. They are not correlated with the dimension of the cephalohematoma and generally they require no treatment.
- Calcification: this is a rare instance and may cause cranial deformity. It is caused by the incomplete resorption of the hematoma, with the formation of fibrous tissue and then (about four weeks later) with the calcification of the mass. The long-term natural history is unknown (8).
- Anemia: this is caused by the collection of blood in the mass. It is generally present in more large lesions (9).
- Hyperbilirubinemia: this occurs when the red blood cells in the cephalohematoma are destroyed (10).
- Infection: the predisposing factors to infection are similar to those associated with the development of a scalp infection in a monitored infant. These include the presence of ruptured membranes, vacuum extraction, high-risk indications for monitoring and the presence of amnionitis.

It has been reported that the incidence of a scalp abscess at the site of electrode application is highly increased when a scalp electrode has been used. It may reach 4.5 per cent of newborn infants following continuous, direct fetal heart rate monitoring during labour (11-12). The most frequent bacteria is *Escherichia coli*, as in our patient, but other bacteria, such as *Staphylococcus aureus*, *Staphylococcus epidermidis*, *Pseudomonas*, *Proteus*,

Bacteroides, *Salmonella*, *Gardnerella*, group B *Streptococcus* and *Escherichia hermannii* may be involved (13). These data indicate that a scalp abscess complicating intrapartum foetal monitoring may be relevant as a nosocomial infection, and infants monitored should be closely observed in order to prevent more serious infectious complications.

If an infected cephalohematoma is suspected, the diagnostic approach should be directed both locally and systemically. If possible, an aspirate should be considered. The systematic approach includes performing blood and CSF culture. Imaging of the skull should be obtained to discriminate the scalp abscess from osteomyelitis, epidural abscess or subdural empyema. Head ultrasounds may be used as the first line diagnosis. Considering the most frequent bacteria responsible for the infection, antibiotics against *Escherichia coli* and *Staphylococcus aureus* should be administered. Moreover, meningitis could be a complication of infected cephalohematoma. Therefore combined treatment with ampicillin and an aminoglycoside should be started immediately, upon suspicion of an infection. In some clinical settings the proportion of *Escherichia coli* resistant to ampicillin may be high, especially when preterm infants catch this infection. In these cases, gentamicin may be a useful option as a second antibiotic. When meningitis has been excluded, ampicillin, aminoglycoside or cephalosporins (i.e. Cefotaxime) may be started as monotherapy and the therapy has to be continued for 10-14 days, but in the presence of meningitis it should be continued for at least 21 days. When *Staphylococcus aureus* is the bacteria responsible for the infection, Vancomycin therapy should be started and continued for four weeks or more. Once diagnosed, if there is no spontaneous drainage, the infected cephalohematoma should be incised and drained and, if underlying osteomyelitis or

epidural abscess are found, surgical debridement should be performed (14-17).

In our case the cephalohematoma was evident on initial presentation, but the infection remained subclinical until late in the course, no risk factors were present, the labour was without complications, and no invasive manoeuvres were performed. It was not possible to establish if the bacteraemia was subsequent to or causative of the infection of the cephalohematoma.

Conclusions

A cephalohematoma associated with persistent fever, erythema of the overlying cutis, tenderness and fluctuation of the mass may be suggestive of an infected lesion. In this case all microbiological and biochemical laboratory tests should be performed to exclude a systemic associated infection. Double antibiotic therapy should be started immediately, considering the frequent serious infectious complications of infected cephalohematoma in infants.

Conflict of interest: The authors declare that they have no conflict of interest.

References

1. Silverman FN, Byrd CR, Fitz CR. The skull: Traumatic lesions. In: Silverman FN, Khun JP (eds): Caffey's Pediatric X-Ray Diagnosis: An Integrated Imaging Approach, 9 ed. St Louis: Mosby; 1993. p. 43-57.
2. Van Helleput C, Dupont V, Barthels S, Aeby A. *Escherichia coli* meningitis and parietal osteomyelitis in an infant: a rare complication of cephalohematoma. Rev Med Brux. 2010;31(1):57-9.
3. Blom NA, Vreede WB. Infected cephalohematomas associated with osteomyelitis, sepsis and meningitis. Pediatr Infect Dis J. 1993;12(12):1015-7.
4. Currarino G. Neonatal subperiosteal cephalohematoma crossing a synostosed sagittal suture. Pediatr Radiol. 2007;37(12):1283-5.

5. Merlob P, Grunebaum M, Reisner SH. Crossed sagittal-suture cephalhaematoma. *Br J Radiol.* 1985;58(694):1007-8.
6. Cohen MM Jr. Sutural pathology. In: Cohen MM Jr, MacLean RE (eds) *Craniosynostosis, diagnosis, evaluation and management*, 2nd edn. New York: Oxford University Press; 2000. p. 51-88.
7. Parker, LA. Early recognition and treatment of birth trauma: Injuries to the head and face. *Advances in Neonatal Care.* 2005;5(6):288-97.
8. Petersen JD, Becker DB, Fundakowski CE, Marsh JL, Kane AA. A novel management for calcifying cephalohematoma. *Plast Reconstr Surg.* 2004;113(5):1404-9.
9. Mangurten HH. Birth injuries. In: Martin RJ, Fanaroff AA, Walsh MC editors. *Neonatal-perinatal medicine: Diseases of the fetus and infant*, 8th ed. Philadelphia: Mosby; 2006. p. 531-535.
10. Wong RJ, De Sandre GH, Sibley E, Stevenson DK. Neonatal jaundice and liver disease. In: Martin R, Fanaroff AA, Walsh MC, editors. *Neonatal-perinatal medicine: Diseases of the fetus and infant* 8th ed, Philadelphia: Mosby; 2006. p. 1419.
11. Brook I: Microbiology of scalp abscess in newborn. *Pediatr Infect Dis J.* 1992;11:766.
12. Okada DM, Chow AW, Bruce VT. Neonatal scalp abscess and fetal monitoring: factors associated with infection. *Am J Obstet Gynecol.* 1977;129(2):185-9.
13. Dahl KM, Barry J, DeBiasi RL. *Escherichia hermannii* infection of a cephalohematoma: case report, review of the literature, and description of a novel invasive pathogen. *Clin Infect Dis.* 2002;35(9):e96-8.
14. Chang, HY, Chiu NC, Huang FY, Kao HA, Hsu CH, Hung HY. Infected cephalohematoma of newborns: experience in a medical center in Taiwan. *Pediatr Int.* 2005;47:274-77.
15. Fan HC, Hua YM, Juan CJ, Fang YM, Cheng SN, Wang CC. Infected cephalohematoma associated with sepsis and scalp cellulitis: a case report. *J Microbiol Immunol Infect.* 2002;35:125-28.
16. Kao HC, Huang YC, Lin TY. Infected cephalohematoma associated with sepsis and skull osteomyelitis: report of one case. *Am J Perinatol.* 1999;16:459-62.
17. Huang CS, Cheng KJ, Huang CB. Infected cephalohematoma complicated with meningitis: report of one case. *Acta Paediatr Taiwan.* 2002;43:217-19.