

Calcified Cephalhematoma, Unusual Scenario of a Twin Baby with Spontaneous Vaginal Delivery

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Cephalhematoma is one of the most common neonatal head injuries, especially during assisted vaginal delivery. It usually regresses within a few weeks and occasionally absorbed. They tend to calcify in rare instances. Follow-up of calcified cephalhematoma will either show spontaneous resolution, stationary course or progression which may cause skull deformity and would eventually need surgical intervention.

We report a case of a one-month preterm twin (one boy and one girl), product of spontaneous vaginal delivery found to have a swelling on the skull. Both twins were initially investigated for hypotonia; MRI brain revealed no intracranial abnormality. The skull swelling was diagnosed as cephalhematoma. Two months follow-up brain CT scan confirmed the diagnosis of calcified cephalhematoma. Further follow-up revealed significant regression of the cephalhematoma.

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Cephalhematoma is a collection of blood within the subperiosteal layer. Various causes have been found¹. Documenting good prenatal, postnatal and delivery history is mandatory to find the exact reason². Cephalhematoma is managed according to its location and the connection sutures. It usually resolves by itself, but in rare occasions, it calcifies^{1,2}. The management of the calcified cephalhematoma depends on its outcome and cosmetic effect. Some of them will regress spontaneously with the remodeling of the skull shape; on the other hand, surgery will be performed for cases which did not resolve².

The aim of this presentation is to report a case of a one-month preterm twin baby boy with cephalhematoma, which was managed conservatively.

THE CASE

A preterm twin baby boy was born on 4 June 2018 via spontaneous vaginal delivery (SVD); he was 33+5 weeks with birthweight of 1.980 kg, Apgar score of 6, and 8. The infant was admitted to NICU.

The maternal history was Gravida 2, Para 1, underwent in-vitro fertilization (IVF), hypothyroidism and on long-life thyroxine therapy.

On examination, the baby was on nasal cannula and lying in supine position; he had spontaneous regular breathing and equal bilateral air entry. The heart was normal with regular heartbeats. The abdomen was soft, and the baby passed urine and stool within 24 hours. The swelling was noted on the right aspect of the skull.

The baby was feeding normally. His neonatal screening was normal. The lab investigations were within normal. EEG revealed no epileptic activity or focal abnormalities. Karyotyping revealed normal male karyotype.

MRI brain at the age of one month revealed no brain abnormality, however, an abnormal skull shape was noted, see figure 1.

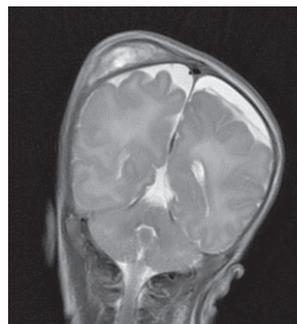


Figure 1 (A): Coronal T2 Image

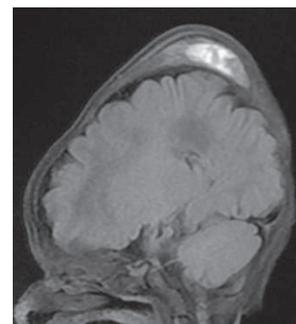


Figure 1 (B): Sagittal FLAIR Image

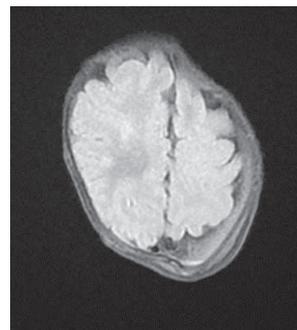


Figure 1 (C): Axial Flair Image

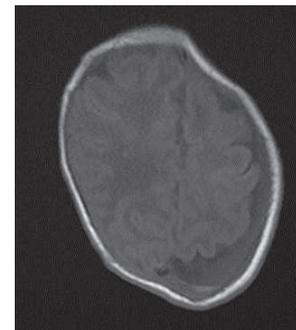


Figure 1 (D): Axial T1 Image

Figure 1 (A-D): MRI Brain (A, B) Revealed Right High Parietal Subgaleal Predominantly Subacute Hematoma; (B, C) Abnormal Skull Outline and Small Left High Parietal Late Subacute Subdural Hematoma

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One month later, follow-up CT brain without contrast revealed calcified high parietal cephalhematoma, see figure 2.

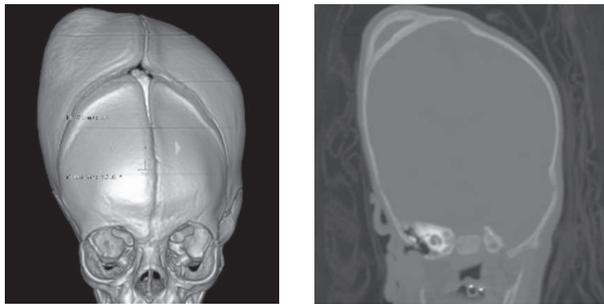


Figure 2 (A)

Figure 2 (B)

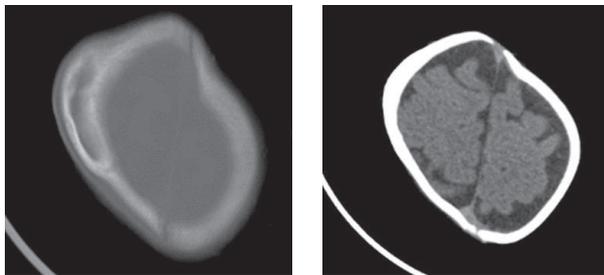


Figure 2 (C)

Figure 2 (D)

Figure 2 (A-D): CT Plain without Contrast (A) 3D Image Showed Abnormal Skull Shape. (B, C) Coronal and Axial Bone Window Images Demonstrated Double Appearance of the Right High Parietal Bone with Isodensity Seen Indicating Calcified Cephalhematoma, No Intracranial Extension. No Craniosynostosis. (D) Resolution of the Left Parietal Subdural Hematoma

Follow-up MRI Brain revealed a decrease in size, see figure 3.

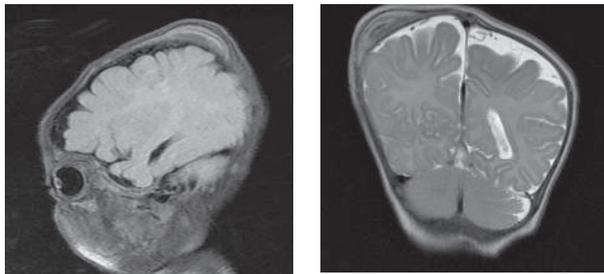


Figure 3 (A): Coronal T2 Image

Figure 3 (B): Sagittal FLAIR Image



Figure 3 (C): Axial FLAIR Image

Figure 3 (A-B): MRI Brain Revealed Decreased Right High Parietal Cephalhematoma Compared to the Previous MRI. (C) No Subdural Hematoma or Other Extra or Intra Axial Blood Collection

Follow-up CT brain after 6 months revealed further regression of the calcified cephalhematoma, see figure 4. Therefore surgical intervention was not considered.

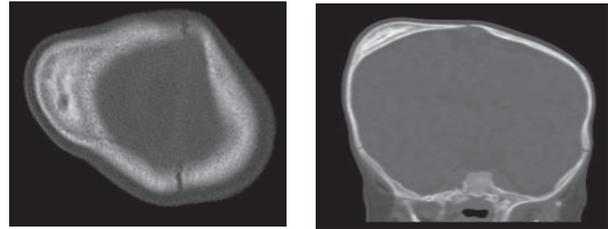


Figure 4 (A)

Figure 4 (B)



Figure 4 (C)

Figure 4 (A-C): Axial, Coronal Bone Window Images and 3D Image Revealed Further Reduction in the Size of the Calcified Cephalhematoma. The Abnormal Skull Shape (Plagiocephaly) was Noted

DISCUSSION

Calcified cephalhematoma is a rare event encountered in infancy¹. Assisted delivery, such as the use of vacuum or forceps extraction is one of the main reasons to develop the cephalhematoma. Other less common causes may include head trauma or coagulation disorders². The cause in some cephalhematomas is not obvious³.

The cephalhematoma is covered by the periosteal layer, which calcifies easily and expands the differential diagnoses, which may include intraosseous lesions, meningoencephalocele, granulomatous lesion or cavernous hemangiomas^{3,6}. Subgaleal hematoma and caput succedaneum are also included in the differentials of cephalhematoma; however, in presence of periosteal attachments, cephalhematoma does not cross the skull suture lines and it is the main distinguishing feature².

Radiological investigations such as CT, MRI or Magnetic Resonance Angiography (MRA) is needed to narrow down the differentials and to diagnose various lesions⁵. CT is usually used initially, though MRI is useful in evaluating the intracranial connection with such lesions⁵. CT scan will demonstrate double skull appearance with hypodense and hyperdense regions^{3,7}. Calcified cephalhematoma is classified as follows: Type I- the inner lamella has its preserved contour, Type II- the inner lamella is depressed⁴. Proper clinical evaluation and history taking for patients with calcified cephalhematoma are essential. Further discussion with the parents regarding treatment options and the outcome is needed².

Calcified cephalhematoma is usually followed up. Cases which undergo spontaneous regression and remodeling will

be managed conservatively². Surgical intervention is indicated when the calcified cephalhematoma causes obvious skull shape deformity.

Spontaneous regression of the calcified cephalhematoma due to the continuous growth of the skull has been illustrated by Daglioglu et al, in which he advised to follow-up the patients up to two years of age².

CONCLUSION

Cephalhematomas usually appear within 1-3 days post-delivery, and rarely ossify. Follow-up of calcified cephalhematoma may reveal spontaneous resolution; yet, failure of regression could result in skull deformity, which ultimately would need surgical intervention.

In our case, Type I calcific cephalhematoma is diagnosed. CT revealed significant regression and was managed conservatively.

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