

Case Report

Rapidly Calcified Epidural Hematoma in a Neonate

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We report a very rare case of a rapidly calcified chronic epidural hematoma (EDH) in a neonate. A 26-day-old female infant was referred to us from a regional hospital because of drowsy mentality and a seizure attack. She was delivered through caesarian section because normal spontaneous vaginal delivery was prolonged and failed. At birth, mild scalp swelling was found on the right frontal area. Scalp swelling was spontaneously resolved and she was discharged without any problems. On the 25th day after her birth, the baby presented with drowsiness and hypotonia following a generalized tonic-clonic seizure. Magnetic resonance imaging (MRI) and a computed tomography (CT) scan revealed a chronic EDH that had a thick layer of calcification. A small burr-hole trephination was performed and a single silastic drainage catheter was inserted. After the operation, a total of 12 ml of liquefied hematoma was drained, and the patient's mentality improved from drowsiness to alertness. The patient was asymptomatic when discharged.

KEY WORDS : Ossification · Neonate · Epidural hematoma.

INTRODUCTION

Ossification or calcification of a chronic EDH has rarely been reported^{1,3,7,9,10}. Furthermore, an EDH in neonates is a rare clinical entity^{2,5,6,12}. In the pediatric population, only five cases of ossified EDH have been reported^{1,3,7,9}, and all of these patients were children rather than infants. A calcified EDH in neonates has never been reported. Surgical attention is important for preventing mortality and morbidity in neonatal EDH^{6,11}. We present the case of a 26-day-old female infant who had a rapidly calcified chronic EDH.

CASE REPORT

A 26-day-old female infant was referred from a regional hospital for drowsiness and a seizure attack. According to the medical records of the regional hospital, she was born after a full term with vertex presentation, and her mother was primigravida. She was delivered through caesarian

section because normal spontaneous vaginal delivery was prolonged and failed. At birth, the weight of the baby was 3.68 kg and mild scalp swelling, especially prominent in the right frontal area, was found. At that time, neuro-imaging studies were not performed. The scalp swelling was spontaneously resolved, and she was discharged without any complications. On the 25th day after birth, she presented with drowsiness and hypotonia following a generalized tonic-clonic seizure. On physical examination, she was icteric and drowsy, while her anterior fontanel was slightly distended. Her pupils were equal and reacted to light, and all limbs moved spontaneously; however, the muscle tone was decreased. Complete blood count, coagulation profile, and serum biochemistry were in the normal range except for the elevation of bilirubin. The serum levels of parathyroid hormone, 1,25-dihydroxy vitamin D, T3, free T4, and TSH were all normal. Brain MRI showed chronic EDH on the right frontal area and subdural hygroma on both frontal areas (Fig. 1A). The cranial CT scan revealed isodense chronic EDH that had a thick layer of calcification (Fig. 1B). A small burr-hole trephination was performed on the right frontal area, and a single silastic drainage catheter was inserted under general endotracheal anesthesia. After operation, a total of 12 ml of liquefied hematoma was drained, and the patient's mentality improved from drowsiness to alertness. A CT scan, which was taken on the first day after the operation, showed that the chronic EDH was nearly

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resolved, but the calcification layer of hematoma and subdural hygroma still remained (Fig. 2A). A follow-up CT scan was taken 2 weeks after the operation. This scan showed that the EDH was completely resolved, but the calcification layer and subdural hygroma still remained (Fig. 2B). We planned close observation and a serial check up of CT scans of subdural hygroma on both frontal areas. The patient was discharged without any problems.

DISCUSSION

Cephalohematoma, skull fracture, subarachnoid hemorrhage, subdural hematoma, and EDH following cranial birth injury have been reported in previous studies^{2,8,11}. However, EDH in neonates is a rare condition and has rarely been reported^{2,6,8,10,12}. The reason for this rarity is the specific anatomic cranial condition⁹. In a newborn, the dura firmly adheres to the overlying skull bone¹¹. The middle meningeal artery is not yet fixed into its cranial groove and moves freely between the skull and the dura. Therefore, the EDH in neonates commonly originates from small venous bleeding points on the dura^{5,10,12}. The process of EDH formation is initiated by the stripping of the dura due to trauma, leading to a small collection at the site that subsequently enlarges due to repeated hemorrhage¹⁰. This is the reason why the majority of neonates with EDH show no deterioration of consciousness at the time of injury and are diagnosed with the chronic form of hematoma^{1,10}. In our case, neurologic symptoms were present on the 25th day after birth trauma and the patient's CT scan showed the chronic form of EDH.

Ossification or calcification of a chronic EDH is a rare clinical entity^{1,9}. In the pediatric population, only five cases of ossified EDH have previously been reported^{1,3,7,9}. The precise mechanism of osseous transformation is still not well understood. Erdogan et al.³ described the mechanism of ossification; damage to vascularized tissues such as bones and the dura matter provokes tissue responses including inflammation, repair, and remodeling.

This natural healing sequence is more rapid in children than adults. Ossification of an EDH in childhood may be explained by the phenomenon of excess tissue repair following acute injury. Several authors have suggested that the clot may undergo fibrous organization and microscopic ossification, and infiltrated osteoblasts at the junction of the epidural granulation tissue and the dura may initiate ossification¹⁰. Predisposing metabolic, hematologic, and endocrinologic disorders could also contribute to ossification.

Neonatal seizure may be a sign of metabolic disorders, structural injury, or malformations⁴. Therefore, patients with neonatal seizures require screening laboratory tests and neuro-imaging studies. Seizures and hypotonia are the predominant signs of EDH in newborn infants⁶. Similarly, our patient presented with seizure and hypotonia. A CT scan or MRI should be taken in patients with neonatal seizures to exclude surgical conditions such as intracranial hematoma.

Some neonatal EDH may spontaneously resolve, and

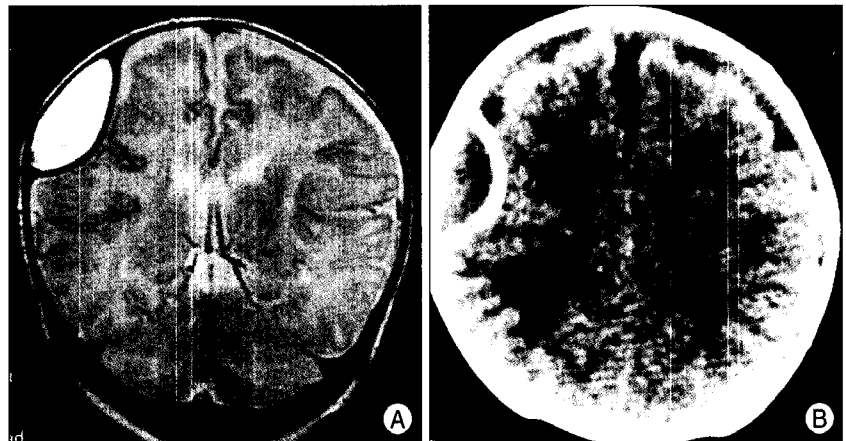


Fig. 1. Preoperative magnetic resonance image showing epidural hematoma (EDH) on the right frontal area and subdural hygroma on both frontal areas (A). A computed tomography scan reveals an isodense chronic EDH that had a thick layer of calcification (B).

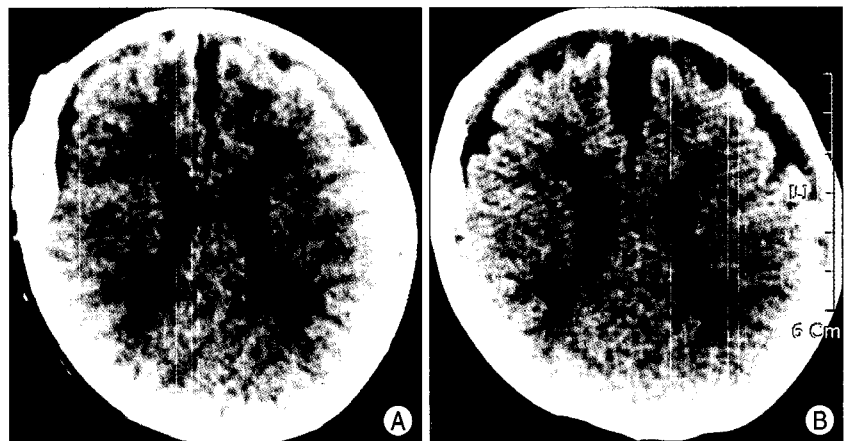


Fig. 2. A computed tomography (CT) scan taken on the first day after the operation show that the chronic epidural hematoma (EDH) is nearly resolved, but the calcification layer of hematoma and subdural hygroma still remained (A). A follow-up CT scan two weeks later show that the EDH has been completely resolved, but the calcification layer and subdural hygroma still remained (B).

surgical treatment is not always necessary⁶⁾. However, we suggest that surgical treatment can prevent neurological deterioration and ossification of EDH in cases of large EDH with a mass effect.

CONCLUSION

We suggest that screening neuro-imaging studies be performed on neonates suspected of cranial birth injury to prevent delayed diagnosis of intra-cranial injuries. Even in the neonatal period, EDH may be rapidly calcified. We propose that if EDH has a mass effect and induces neurologic deficits, surgical intervention should be considered to prevent morbidity and calcification of the EDH. To our knowledge, this is the first case of a rapidly calcified chronic EDH in a neonate.

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