ASYMPTOMATIC CHRONIC EPIDURAL HEMATOMA IN A CHILD AS A RESULT OF EXTRACRANIAL DECOMPRESSION

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Abstract—Background: Epidural hematoma (EDH) in children is a diagnostic challenge due to its nonspecific clinical presentation. Asymptomatic chronic epidural hematoma is a very rare entity. Reports of spontaneous decompression into the subgaleal spaces are limited with acute epidural hematomas in the literature. Objective: We report a child presenting with chronic epidural hematoma at 15 days after a head trauma. She remained asymptomatic, owing to spontaneous decompression via a skull fracture. We intend to remind emergency physicians to be alert about epidural hematomas in asymptomatic children in the presence of, even minor and distant, trauma. Case Presentation: An 8-year-old girl presented to the Emergency Department with a swelling in the right parietal region. She had fallen at the playground and struck her head on the ground 15 days prior. Computed tomography showed a mixed-density subacute-chronic parietal epidural hematoma with a linear fracture overlying it. There was no evidence of midline shift or ipsilateral ventricular compression. Conclusion: An initially minimal but expanding EDH in a child can remain asymptomatic even in the later phases, owing to the spontaneous decompression through a skull fracture. © 2014 Elsevier Inc.

Keywords—chronic; epidural hematoma; decompression; asymptomatic; child

INTRODUCTION

Epidural hematoma (EDH) is a rare, yet life-threatening, complication of head trauma in children. It often results from a relatively minor trauma (1). An EDH is referred to as chronic when it trails a trauma by 2 to 13 days (2,3). However, there is not an exact definition of chronic EDH regarding the interval between the trauma and the diagnosis. Although there are several reports associating chronic EDH with a variety of symptoms, the asymptomatic presentation of a chronic EDH is very rare (3–5).

Spontaneous decompression of EDHs into the subgaleal space has been reported within 2 days after traumas. We report a child presenting with chronic epidural hematoma 15 days after a head trauma. She remains asymptomatic owing to the spontaneous decompression through a skull fracture.

CASE PRESENTATION

An 8-year-old girl was admitted to the Emergency Department (ED) with a swelling in the right parietal region. Although there was no history of a recent head trauma, her parents stated that she had experienced a ground-level fall at the playground and had struck her head on the ground 15 days prior. She was taken to the ED of a state hospital in another city. Relying on her and the family’s reports, she did not have any complaints suggesting injury. She revealed normal neurological...
examination with no external signs of trauma. She had been observed for 6 h and discharged with head injury instructions because her neurological examination was normal. The patient and her parents denied headache, vomiting, fever, seizures, or any change in her mental status by then. She had remained asymptomatic for 13 days. Two days prior to the ED presentation, a swelling had occurred on the right parietal region, expanding slowly. This concerned the family and they brought her to the ED seeking further evaluation. She had no known disease or allergy and was not taking any medication. She had no other complaints. On presentation, the patient was appropriately alert and interactive with her parents and the ED staff. She had no obvious signs other than a large hematoma on her right parietal region over the scalp. The neurological examination showed normal cranial nerves with clear fundi. Motor strength was 5/5 with no pronator drift. Sensory examination was unremarkable. There were no pathologic reflexes. On cerebellar examination, she had no dysmetria on finger-to-nose testing. Her gait was normal, and Romberg testing showed no abnormality. She had no neck pain or tenderness.

The detailed history and physical examination suggested no exact diagnosis. Considering the large and still-growing isolated head swelling with a distant trauma in conjunction with the parental preference, we obtained a cranial computed tomography (CT) scan. The CT scan showed a mixed-density subacute-chronic EDH involving the parietal region of the brain (Figure 1) that was associated with an overlying linear parietal fracture (Figure 2). There was no evidence of midline shift or ipsilateral ventricular compression (Figure 3). The EDH, which consisted of organized clots, was surgically evacuated via a craniotomy. The source of the hemorrhage was not identified upon surgery. The patient was discharged after an uneventful 4 days. No symptoms and neurological findings relevant to the injury were identified on control visits at the first and third months after neurosurgery.

DISCUSSION

Chronic EDH is an exceptional entity in childhood that is not well characterized (6). Unlike adults, children have
unfused cranial sutures, open fontanelles, large extracerebral spaces, and basal cisterns, whereas the dura is relatively firmly attached to the skull (7). Furthermore, the source of hemorrhage is often venous, which produced a later onset mass effect. Therefore, children tolerate an increase in intracranial pressure better than adults (8,9). However, reported chronic EDHs in children are often symptomatic (3,4,6,10). The case presented hereby is unique for being an asymptomatic chronic EDH owing to the latest spontaneous extracranial decompression in the literature.

There are several theories regarding the late onset of symptoms in a chronic EDH. Venous origin and later accumulation of the hemorrhage, the adherence of dura in particular regions causing smaller hematomas or the accumulation of subgaleal hematoma in the epidural space through a skull fracture have all been claimed to be responsible for chronic EDH (4,11,12). Repeated hemorrhage from the hematoma membrane has been emphasized to be responsible for the chronic expansion of initially minimal EDH (13). In support of this theory, Kanamori et al. reported a case of gradually expanding EDH (10). EDH in our patient was evident in the expansive phase (between days 5 and 16) as described by Pang et al. (8). However, there are reports of chronic EDHs that have become clinically evident far later (13–15).

Elevated interstitial pressure in the subgaleal compartment, pulsatility of the intracranial contents or pressure gradient, has been argued as the mechanism leading to spontaneous decompression (16–18). In our case, we believe that the decompression has occurred due to the pressure gradient, because our patient did not initially have a scalp hematoma. The presence of overlying fractures in the previously reported cases of spontaneous rapid resolution of acute EDHs might also point to the pressure gradient mechanism (19–21). The changes in the density of the pericranial soft tissues on CT imaging favor this hypothesis as well (22). Bor-Seng-Shu et al. also reported three asymptomatic children with acute EDH having only head swelling 2 days later. The children all had associated skull fractures (23). Chida et al. reported an asymptomatic infant whose acute EDH slowly drained into the subgaleal space through a fracture within 5 days, as evidenced on CT imaging (24).

In this case, we believe that the leakage from the expanding hematoma through the skull fracture as a result of the pressure gradient prevented the development of the clinical signs of a mass effect. There were no findings suggestive of increased intracranial pressure on her CT scan.

A CT scan had not been obtained on the initial visit to the other ED. Although CT is the reference standard for emergently diagnosing traumatic brain injuries, concurrent with the growing evidence of radiation exposure complications, clinical decision rules (CDRs) have become extremely important. They are anticipated to substitute CT scanning for safe disposition, especially for children with minor head trauma. Relying on the reports of her family, our patient did not require neuroimaging regarding any of the established CDRs for children with minor head trauma such as CATCH (Canadian Assessment of Tomography for Childhood Head Injury), CHALICE (the children’s head injury algorithm for the prediction of important clinical events), or PECARN (Pediatric Emergency Care Applied Research Network) at the time of the initial ED visit outside (25–27). Nevertheless, on the 15th day of the trauma she had a large parietal hematoma indicating CT scan on the basis of physician experience and parental preference according to PECARN, and medium brain injury risk according to CATCH rules, despite the disparity for the time course. Our patient did not suffer any complications from the delay in diagnosis owing to the spontaneous decompression through the skull fracture. This may be attributed to the validity of CDRs. However, even clinically important brain injury is encountered very rarely; in seemingly mild head trauma in practice, potentially severe outcomes should be considered for children. Although our patient survived with no deficits owing to spontaneous decompression, all children with unpredictable brain injuries may not experience such a favorable outcome. Embracing the low-dose radiation exposure principles, research may be focused on an adjunct method such as a marker to help CDRs in decision-making about neuroimaging in children with minor head traumas.

Limitations

Because the patient did not have any signs and symptoms suggestive of cranial injury, neuroimaging studies had not been initially performed. Furthermore, if the source of the hemorrhage could have been identified during surgery, a more accurate interpretation of the mechanism could have been possible.

CONCLUSION

Regardless of the mechanism involved, due to the unpredictable and potentially life-threatening outcome, a high level of suspicion towards EDH is required in children with a history of head trauma, even if they are asymptomatic.

REFERENCES