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CASE REPORT

A subacute epidural haematoma extending over the occipital region and posterior cranial fossa due to a laceration in the transverse sinus

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Abstract A 6-year-old male was found dead on his stomach with massive reddish vomiting from his mouth and nose. Postmortem cranial CT revealed an epidural haematoma in the left occipital region, but the cause and origin of the haematoma were unclear. An autopsy revealed that the epidural haematoma expanded over the left temporal region and the left side of the occipital region and posterior cranial fossa, and its origin was a laceration in the left transverse sinus induced by diastases in the left lambdoidal and occipitomastoid sutures. A pathohistological examination revealed that one portion of the haematoma was an early-stage hemorrhage, while the other portion extended approximately 1 week after the hemorrhage. Moreover, approximately 1 week elapsed after the laceration of the transverse sinus. Thus, we believe that the primary haematoma was induced by the laceration in the transverse sinus approximately 1 week before death, but the haematoma ceased to enlarge due to hemostasis. However, later, the size of the haematoma rapidly increased again due to rebleeding from the laceration, which led to intracranial hypertension. Consequently, we diagnosed the direct cause of death as choking due to vomit aspiration that resulted from intracranial hypertension induced by a subacute epidural haematoma.

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Introduction

In infants and young children, death resulting from traumatic head injury accounts for up to 80% of deaths [1–5]. While the most common cause of severe traumatic brain injury is abuse, accidental head injury causes approximately 20% of accidental deaths in children [6, 7]. Accidental head injury mainly results from motor vehicle accidents and falls; however, abuse must be considered if the history is inconsistent with the injury pattern [1, 3].

In accidental short falls from distances of less than 2 m, the majority of patients exhibit no injury. However, approximately 1% to 3% of short falls causes skull fractures (such as simple linear fractures), less than 1% of which leads to an epidural or subdural hemorrhage [6]. Whereas almost all epidural haematomas (EDHs) may develop due to a laceration in the middle meningeal artery accompanied by a parietal bone fracture, EDHs in the posterior fossa tend to have a venous origin and, even when small in size, may lead to a poor prognosis compared to an ordinary epidural haematoma [8–14]; nevertheless, to our knowledge, there have been no autopsy cases of EDHs in the posterior fossa in the forensic literature.

Here, we report an autopsy case of a subacute EDH in the posterior fossa due to a laceration in the transverse sinus induced by suture diastasis, which may have been caused by a head bruise without a scalp laceration or contusion when the individual accidentally fell down a set of stairs.

Case history

Clinical history

One morning, a 6-year-old male in cardiopulmonary arrest was found by his mother. He was on his stomach, with massive reddish vomiting from his mouth and nose. He was carried to an emergency hospital, and cardiopulmonary resuscitation was vigorously performed. However, he died without recovering his heartbeat or breathing. Immediately after his death, a computed tomography scan of his entire body was performed as postmortem diagnostic imaging, and a subacute EDH was detected in the left occipital region (Fig. 1). A pediatrician suspected choking induced by vomit aspiration due to intracranial hypertension because EDH was the cause of death. However, this proposal was rejected by the cranial neurosurgeon. Consequently, to investigate the cause of death, a medico-legal autopsy was performed 12 h after the estimated time of death.

Autopsy findings

Upon an external examination, the deceased was 120 cm tall and weighed 21 kg. The nail matrices of the upper and lower limbs were cyanotic.

There was a subcutaneous hemorrhage, which was approximately as large as a hen's egg on the left side of the occipital region. In addition, there were a few slight abrasions and subcutaneous hemorrhages on his head, face, chest, lumbar area, and lower extremities. Corresponding to the subcutaneous hemorrhage in the occipital region, a fist-sized hemorrhage spread along the galea on the entire left side of the occipital region and the posterior half of the left temporal region, with intramuscular hemorrhages in the left temporal and nuchal muscles. Moreover, diastases consecutively occurred in the left lambdoidal, occipitomastoid, parietomastoid, and squamoparietal sutures in the deep portion of the subcutaneous hemorrhage on the left side of the occipital region (Fig. 2a).

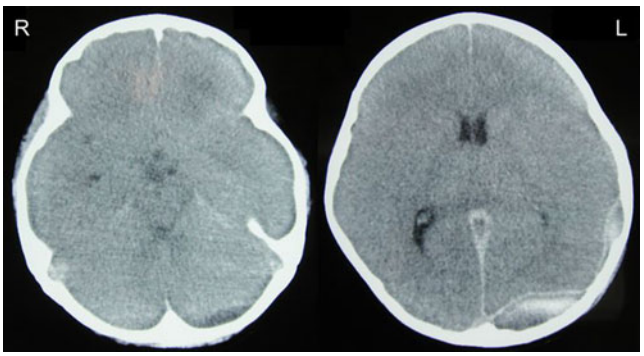


Fig. 1 Postmortem cranial CT reveals a subacute epidural haematoma expanding over the left occipital region and left posterior cranial fossa. The perimesencephalic cisternae tend to be obliterated, and the left inferior horn of lateral ventricle is completely compressed due to the epidural haematoma, which indicates intracranial hypertension

Intracranially, there was a partially organised EDH (more than 10 g) expanding over the left temporal area and the left side of the occipital area and posterior cranial fossa (Fig. 2b). Within the EDH, the skull sidewall of the left transverse sinus was slightly lacerated, and the EDH was medial with respect to the laceration (Fig. 2c).

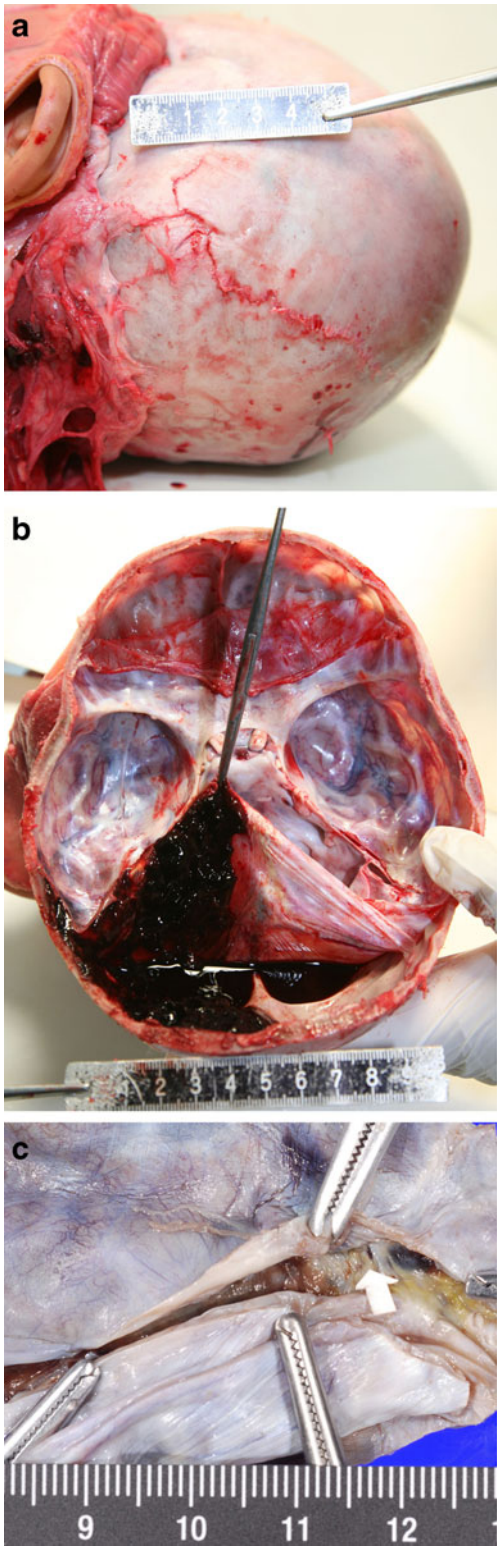
The cerebrum was oedematous and, in combination with the cerebellum and the brain stem, weighed 1,400 g; thus, the gyrus was compressed by the dura mater and became flat. In particular, the left occipital lobe was compressed by the epidural haematoma. The lungs were bilaterally congested and oedematous without pneumonia (lung weights, left, 223 g; right 271 g). Moreover, there was substantial saburra with gastric fluid in the trachea and bilateral main bronchi. The other organs showed macroscopically no significant findings, except for congestion and dark-red blood flowing from the heart without clotting.

Histology

In the EDH with the agglutinated dura mater, the haemorrhage was clearly divided into three structures (Fig. 3a–e of the Electronic supplementary materials). In the first EDH structure, numerous erythrocytes were observed. Some anti-CD68-positive macrophages and lymphocytes were scattered among some fibroblasts. The first EDH structure was negative for Fe staining and was observed in the outermost layer of the EDH in the dura mater. We believe that this region exhibited fresh bleeding inside the old bleeding. In the second structure, there were erythrocytes, some macrophages, and lymphocytes within the fibrin fibres. Most likely, this structure existed in the centre of the EDH, which was negative for Fe staining. The third structure in the haemorrhage consisted of erythrocytes, slight neutrophil infiltration, and the infiltration of lymphocytes, macrophages, and fibroblasts. Moreover, the third structure was positive for Fe staining, which was especially strongly observed in the macrophages. The third structure was located between the former two structures.

Upon the laceration of the left transverse sinus in the dura mater, the elastic fibres of the sinus side were entirely disconnected, and the haemorrhage intruded. The Fe staining was negative. The edge of the laceration had become less sharp and was covered with mesothelial cells, as was the surface of the hemorrhage (Fig. 3f of the Electronic supplementary materials).

The cerebrum, cerebellum, and brain stem were congested, and the cerebral gray matter was oedematous. In



◀ **Fig. 2** Macroscopic examination of the skull, dura mater, and cerebrum. **a** Diastases in the left lambdoidal, occipitomastoid, parietomastoid, and squamoparietal sutures are apparent from the external view of the skull. **b** A partially organized epidural haematoma is expanding over the left temporal area and left side of the occipital area and posterior cranial fossa. **c** A small laceration is observed along the skull sidewall of the left transverse sinus (*white arrow*, dura mater fixed by a 4% formaldehyde solution)

addition, microbleeding without inflammatory infiltration was observed in the white matter, which suggested intracranial hypertension. In the bilateral lungs, there were many foreign objects in the alveoli, but no inflammatory infiltration was observed.

Toxicology

The blood alcohol concentration was 0.00 mg/ml. No drugs were detected.

Criminal investigation

The individual had lived with his father, mother, and two siblings. Although his junior brother had been born to the same parents, his senior sister had been born to his mother and her ex-husband. However, there was no criminal evidence suggesting that he or his siblings had suffered from abuse, and he went to nursery school normally 1 day before his death and then played cheerfully with his mother and siblings until going to bed. Moreover, a few weeks after the autopsy, his diary was found in his desk. Ten days before death, he had written that, because he had fallen down stairs and hit his head against the floor of halfpace, he had a left earache. However, afterwards, he had not complained about an earache. In addition, he had neither a congenital disorder nor mental retardation.

Discussion

In autopsy cases where infants or children die due to head injuries, it is most important to assess whether the head injury was accidental or inflicted by someone such as a caregiver [3, 5, 7]. In this case, there were no significant injuries suggesting inflicted injuries, such as old subcutaneous haemorrhage, rib fractures, or subdural haemorrhage [8, 9], except for the diastases of sutures and EDH. A few slight abrasions and subcutaneous haemorrhage on the patient's head, face, chest, lumbar area, and lower extremities were considered to have been incurred during daily life including play. Additionally, there was no finding indicating poor nutrition when the autopsy was performed. Thus, it is likely that the head injury was accidental rather than inflicted and that he had fallen down stairs, bruising the occipital area against the flat surface because there was some suture diastases without laceration [15].

From the results of pathohistological examinations of EDH, the first haemorrhage and the last one were estimated to have occurred more than 1 week before and shortly before the death, respectively [16]. However, the left

transverse sinus might have been lacerated approximately 1 week before death, considering its degree of restoration [16]. Thus, it was estimated that the diastases of the left lambdoidal, occipitomastoid, parietomastoid, and squamoparietal sutures were directly caused by contusion on the left occipital area approximately 1 week before death, leading to laceration of the left transverse sinus, which resulted in development of the first EDH. It is possible that the EDH grew gradually due to rebleeding at least twice before death, leading to intracranial hypertension, which caused choking due to the aspiration of vomit, on the basis of findings suggesting sudden death including asphyxia.

Although the mortality rate of all EDHs has been reported as less than 10%, which is extremely low compared with that of traumatic subdural haematoma, it is difficult to make a diagnosis of posterior cranial fossa EDH because its clinical progress is silent and slow and its symptoms are nonspecific, leading to its becoming incurable and characterised by poor prognosis [10–14, 17–19]. Indeed, the criminal investigation after the autopsy revealed that the patient had accidentally fallen down stairs in his apartment 10 days before death. However, there were neither subjective nor objective symptoms such as headache, nausea, or disturbed consciousness until his last sleep before death, except for “left earache” on the day of the accident. Moreover, his mother had not thought much about the earache because the symptom disappeared the next day. If he had gone to see a physician immediately after the accident because of the earache, and if cranial CT had been performed after the accident during the patient's time in the hospital, the posterior cranial fossa EDH might have been diagnosed before death [12–14]. Approximately 60% of EDHs are thought to be of arterial origin, whereas the source of bleeding is venous vessels or diploic veins in bone in 9.7% of all EDHs [10, 11]. Concerning EDHs in the posterior cranial fossa, however, the frequencies of venous sinus and/or vessel origin could be estimated at 25% to 50%, commonly in association with diastatic fractures and/or occipital fractures [12–14]. The mortality rates of EDHs due to dural sinus bleeding are higher than EDHs due to arterial damage [10, 20]. Conversely, with regard to subacute EDH in posterior cranial fossa, the clinical symptoms of which emerge from 24 h to 7 days after injury, all cases had been treated conservatively and were considered to have good prognoses, according to previous reports [13, 14]. Even if the EDH could have been diagnosed precisely, it is unclear whether this patient could have been saved by appropriate treatment because his clinical symptoms had drastically deteriorated, which means that it was an extremely rare case in which it would have been difficult to select the appropriate therapy for subacute EDH in the posterior cranial fossa.

“”One of the concerns is the cause of rebleeding. There are at least three causes by which the occurrence of rebleeding could be explained. First, the patient had suffered from hemostatic disorders [21]; however, none of them had been found during his life, which indicated against the likelihood of this finding. Secondly, elevated venous pressure could have led to rebleeding from the laceration in the transverse sinus [22–24]; however, there were no findings suggesting elevation of venous pressure such as facial or conjunctival petechiae at the autopsy. Thirdly, it is possible that the occipital area had been hit again accidentally a few days before death, but that this could not be detected at the autopsy because the second impact might have been a minimal trauma. Thus, it is considered that the rebleeding can most likely be attributed to either the second or the third explanation, or both.

In conclusion, we investigated an autopsy case of subacute EDH in the posterior cranial fossa. It is considered that, in this case, it would have been clinically difficult to select the appropriate therapy for subacute EDH in the posterior cranial fossa.

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