

CASE REPORT

Nagoya J. Med. Sci. 82. 377–381, 2020
doi:10.18999/najms.82.2.377

Growing skull fracture with an atypical mechanism: a case report

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ABSTRACT

Growing skull fractures (GSFs) are well-known but rare causes of pediatric head trauma. They generally occur several months after a head injury, and the main lesion is located under the periosteum. We herein report a case involving a 3-month-old boy with GSF that developed by a different mechanism than previously considered. It developed 18 days after the head injury. A large mass containing cerebrospinal fluid and brain tissue was present within the periosteum. A good outcome was obtained with early strategic surgery. Injury to the inner layer of the periosteum and sudden increase in intracranial pressure might be related to GSF in this case.

Keywords: cranioplasty, duraplasty, growing skull fracture, pediatric, trauma

Abbreviations:

GSF: Growing skull fracture

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INTRODUCTION

Growing skull fracture (GSF) is a well-known but uncommon complication of pediatric head trauma,¹ with the reported incidence ranging from 0.05% to 1.60%.² It was first described in 1816, and its mechanism has been elucidated in several reports.³⁻⁸ Four essential factors associated with the incidence of GSF have been reported: (a) skull fracture during infancy or early childhood, (b) dural tear with an intact arachnoid membrane at the time of fracture, (c) underlying parenchymal injury, and (d) cranial defect resulting from enlargement of the fracture gap.⁴ Most GSF cases occur in children aged less than 3 years, of which almost 50% occur in those aged less than 1 year.^{6,9} The dura is more easily torn at these susceptible ages because of its tight adhesion to the skull as well as the particular features of the skull in young children (i.e., thinner, less stiff, and more deformable than the skull in older children and adults).⁹⁻¹¹ Early diagnosis and prompt surgical treatment are important to ensure a good outcome, whereas delayed treatment leads to poor prognosis.⁹ Here we report the successful treatment of GSF with an atypical mechanism in an infant.

Received: May 8, 2019; accepted: September 20, 2019

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CASE PRESENTATION

A boy fell from the second floor of his house at the age of 2 months and 23 days and was transferred by an ambulance from the nearest hospital to our hospital. On arrival, right parietal to temporal subcutaneous tissue swelling was noted, indicating subgaleal hemorrhage. His Children's Coma Scale score was O4V3M4, and mild to moderate left lower limb motor weakness was seen. Radiological examination showed a linear fracture in the right temporal bone and contusion in the right parietal and temporal lobes (Fig. 1a, b). Hospitalization and conservative treatment were initiated.

Nine days after the injury, at the age of 3 months and 1 day, improvements in the subgaleal hemorrhage and paralysis were seen, and the patient was discharged from the hospital. However, 18 days after the injury, at the age of 3 months and 10 days, the patient's mother noticed sudden swelling of the subcutaneous tissue. At 27 days after the injury, at the age of 3 months and 19 days, a follow-up examination revealed enlargement of the fracture line, remarkable subcutaneous fluid collection, and moderate external hydrocephalus (Fig. 1c, d). A GSF was considered and an emergency surgery was performed.

A large U-shaped incision was marked in the right temporal, parietal, and occipital areas for preservation of the superficial temporal artery and occipital artery (Fig. 2a, b). The skin was cut and the skin flap was turned up, just on the subgaleal layer, revealing a large cystic mass with walls consisting of the periosteum (Fig. 2c). The mass was connected to the intracranial tissue via the fracture line and constituted cerebrospinal fluid and brain tissue (Fig. 2d). The craniotomy was expanded in the ventral and dorsal directions from the bone defect, confirming a dural defect. The dural defect was larger than the initial bone defect (Fig. 2e). Duraplasty was performed using the occipital periosteum (Fig. 2f). Cranioplasty was performed with an autologous bone flap and nylon yarn (Fig. 2g). The bone flap was covered with a pedicled periosteum flap (Fig. 2h), and the skin was closed.

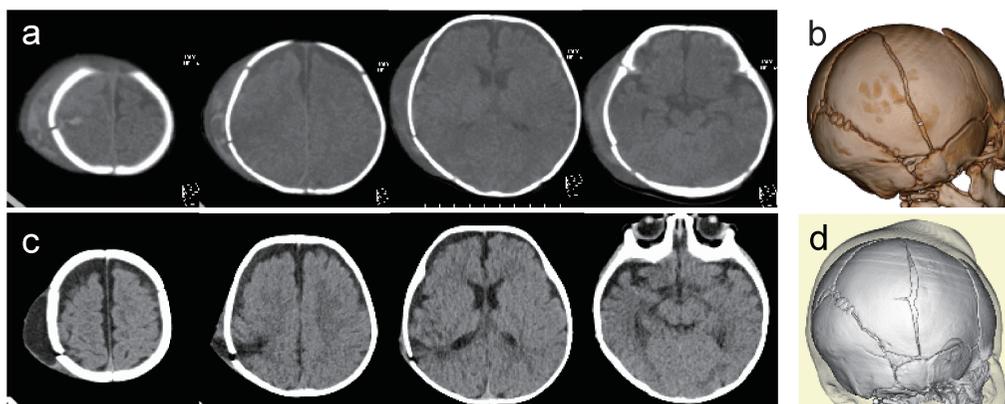


Fig. 1 Computed tomography (CT) performed at the initial visit to our department

Fig. 1a: Right subgaleal hematoma, temporal bone linear fracture, and parietal and temporal lobe brain contusion are observed.

Fig. 1b: A three-dimensional image also shows the linear temporal bone fracture; however, a bone gap cannot be seen.

Fig. 1c: CT performed 27 days after the injury shows subcutaneous fluid collection and mild external hydrocephalus.

Fig. 1d: A three-dimensional image shows deterioration of the bone gap.

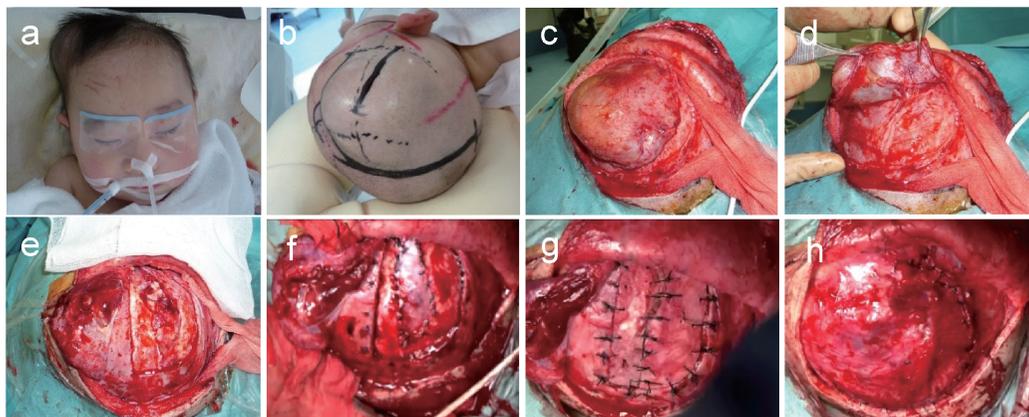


Fig. 2 Intraoperative findings

Fig. 2a: Remarkable skin swelling can be seen in the right temporal and parietal areas.

Fig. 2b: A large U-shaped incision has been marked.

Fig. 2c: A large cystic mass can be seen after dissection of the subgaleal layer.

Fig. 2d: The walls of the mass consist of the periosteum, and the mass is connected to the intracranial tissues via the fracture line.

Fig. 2e: A very large dural defect can be identified after craniotomy.

Fig. 2f: Watertight duraplasty is performed with the periosteum.

Fig. 2g: Cranioplasty is performed with autologous bone and nylon yarn.

Fig. 2h: The bone flap is covered with a pedicled periosteum flap for further ossification of the bone defect.



Fig. 3 Postoperative CT

Fig. 3a: CT performed 3 months after the surgery shows improvement in the external hydrocephalus.

Fig. 3b: A three-dimensional image shows bone flap fusion.

The postoperative progress was excellent, and the boy was discharged without hangovers. Computed tomography follow-up at 3 months after surgery showed an improvement in the external hydrocephalus and bone flap fusion (Fig. 3a, b).

DISCUSSION

In this report, we presented a case of GSF with an atypical mechanism in an infant. The specific mechanism of GSF has been described. After the development of a linear skull fracture with dural disruption with or without underlying parenchymal injury, herniation of the arachnoid or brain tissue occurs. These tissues press the fracture edge and disturb the bone circulation, causing further erosion of the fracture edge. Gradual advancement and enlargement of the fracture gap occur, and a GSF appears several months to 1 year after the initial head injury.^{3,8} On the

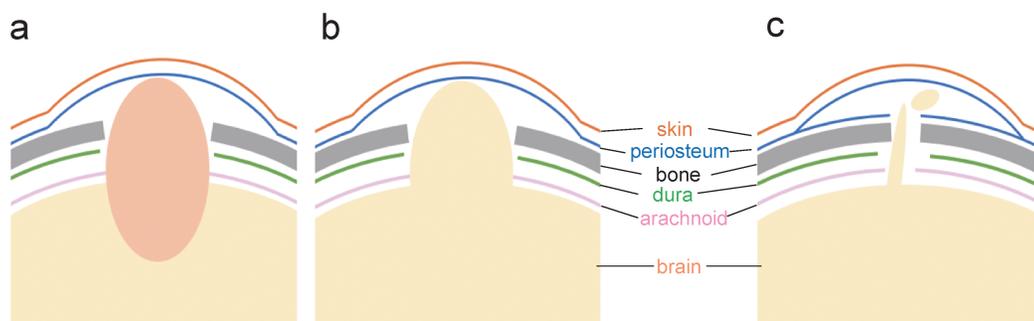


Fig. 4 Classification of growing skull fractures

Classically, growing skull fractures are primarily grouped as a the leptomeningeal cyst type, in which the arachnoid herniates into the subperiosteal layer via the bone defect, and b the brain evacuation type, in which the brain tissue herniates into this layer.

c The present case is not classified as either of these types. The cerebrospinal fluid and brain tissue have herniated into the intraperiosteal space.

basis of this mechanism, GSF is mainly classified as the leptomeningeal cyst type, which forms through herniation of the arachnoid, or the brain evacuation type, which forms through herniation of brain tissue (Fig. 4a, b).^{5,7,8} However, in the present case, GSF occurred only several weeks after the head injury, and the herniated region was not consistent with either of the two types. Instead, cerebrospinal fluid and brain tissue herniated into the intraperiosteal space, suggesting a different mechanism altogether (Fig. 4c). The periosteum consists of an outer “fibrous layer” and an inner “cambium layer”.¹² With respect to the mechanism in the present case, disruption of the inner layer of the periosteum may have occurred at the time of the initial injury, which involved a linear skull fracture, dural disruption, and underlying parenchymal damage. Moreover, a sudden rise in the intracranial pressure due to external hydrocephalus and brain contusion contributed to the injury, and the brain tissue herniation and cerebrospinal fluid leakage into the intraperiosteal space might have occurred in only 3 weeks. If the condition had been neglected, the fracture gap may have continued to enlarge because of fracture-edge erosion.

One of the limitations of this case study is that the pathological tissues were not obtained during surgery. Therefore, we could not prove that cerebrospinal fluid existed between the outer fibrous periosteum layer and inner cambium periosteum layer.

CONCLUSION

In conclusion, we presented a case involving an infant with GSF associated with an unusual mechanism. Although the pathological tissues were not obtained for analysis, the early appearance of GSF after the initial head injury suggested an atypical mechanism.

DISCLOSURE STATEMENT

The authors declare that there is no conflict of interest.

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