

Case Report



Growing Skull Fracture in the Primary Motor Cortex in a 50-day-old Child: A Case Report

Inkyeong Kim

Department of Neurosurgery, Kangwon National University Hospital, Kangwon National University School of Medicine, Chuncheon, Korea



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Address for correspondence:

Inkyeong Kim

Department of Neurosurgery, Kangwon National University Hospital, Kangwon National University School of Medicine, 156 Baengnyeong-ro, Chuncheon 24289, Korea.
E-mail: deargrape@naver.com

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ORCID iDs

Inkyeong Kim

<https://orcid.org/0000-0002-7761-9456>

Conflict of Interest

The author has no financial conflicts of interest.

ABSTRACT

Growing skull fracture (GSF) is a rare complication of skull fracture in children. We report a case of GSF, also known as leptomeningeal cyst with significant damage in the motor cortex in a 50-day-old child, but the motor function was preserved. A 50-day-old male baby visited our hospital after trauma in the left side of the head. His level of consciousness and motor function were normal. Brain computed tomography (CT) scan revealed gapped skull fracture of the left parietal lobe with underlying contusion and subdural hemorrhage. During hospitalization, bulging in the left parietal scalp had progressed, and follow-up magnetic resonance imaging revealed increased skull defect with enlarged leptomeningeal cyst at the left motor cortex. Cranioplasty and duroplasty were performed. Intraoperatively, a dura tear, brain tissue herniation and fluid collection around the motor cortex were observed. One-year follow-up CT revealed cystic encephalomalacia in the left motor cortex. During the 30-month follow-up, nearly normal gross motor function was observed except for few fine motor impairments. We report a case of GSF with significant damage on the motor cortex in an early infant, but with the preserved motor function during the postoperative developmental process.

Keywords: Motor cortex; Skull fractures; Neuronal plasticity

INTRODUCTION

Growing skull fracture (GSF) is a rare type of pediatric skull fracture, with an incidence rate of <1%, and it commonly occurs in children aged below 3 years.^{8,10,12} The mechanism of the GSFs is due to the herniation and enlargement of gliotic brain tissues through the site of dura tear.^{1,7,12} Neurologic symptoms and signs of GSF may vary based on the site of injury and the hemiparesis can occur with the parietal predominance of GSF.^{1,7,12} Herein, we report a case of GSF, also known as leptomeningeal cyst, characterized by a significant damage in the motor cortex in a 50-day-old child. However, the motor function was preserved during the postoperative developmental process.

CASE REPORT

A 50-day-old male baby visited the emergency room due to fussing and crying after hitting the left side of his head on a wall while in his mother's arms. The patient presented with a normal level of consciousness, prompt reaction, bilateral pupil reactivity, and symmetric motor function. Skull radiography (**FIGURE 1**) and brain computed tomography (CT) scan (**FIGURE 2A**) revealed left parietal skull fracture, acute subdural hemorrhage, and hemorrhagic contusion of the left frontoparietal lobe and falx. The diastasis of the skull fracture site was 13.95 mm (**FIGURE 2B**). The parents were counseled, and there were no signs of child abuse. Moreover, no other sites of trauma were found. Within the first 24 hours, the patient presented with partial seizure on the right arm and leg within several seconds for 7 times, and the seizure was controlled after the administration of levetiracetam. Cranioplasty was planned to perform after the patient's condition stabilized. However, during hospitalization, swelling on the left parietal area gradually progressed. Brain magnetic resonance imaging at 12 days after the injury revealed worsening of skull defect, with a diastasis of 19.91 mm (**FIGURE 2B**), and progression of brain tissue herniation and gliotic changes in the fracture site at the left motor cortex (**FIGURE 2B-D**). Due to his family's hesitation, surgery was conducted on the 18th day of insult, and a worse scalp bulging was noted (**FIGURE 3**). Detailed sensorimotor assessment cannot be conducted because the patient was too young. However, there was no evidence of hemiparesis, and normal muscle tone, posture, and reflexes were noted at the time of surgery. Cranioplasty using the bicoronal approach with autologous split calvarial grafts was performed. Operative findings showed extensive dura tear at fracture edge, brain tissue herniation, and fluid collection around the motor cortex. After removing the contused and degenerated brain tissue, watertight duroplasty was performed using an artificial dura with skull graft fixation with an absorbable 1-0 Vicryl. The patient discharged 11 days after surgery without any complication and neurologic deficit. At 1-year follow-up, CT scan revealed cystic encephalomalacia in the left motor cortex (**FIGURE 4**). The patient underwent persistent rehabilitation during the 30 months, and his muscle strength and cognitive function were evaluated using the Denver Developmental Screening Test⁵⁾ and the Gross Motor Function Classification System.²⁾ At the age of 25 months, the patient could go up the stairs with support but had slight resistance with right ankle dorsiflexion. At the age of 32 months, he could run and go up and down the stairs without support but with right heel lifting when he rises to his feet, fine motor

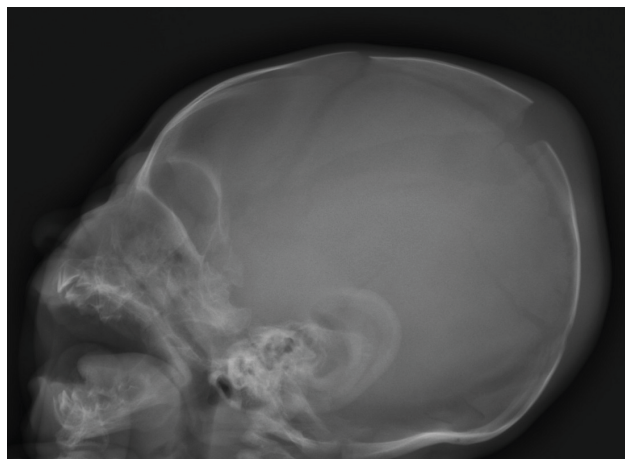


FIGURE 1. Skull lateral radiography of a 50-day-old patient with a growing skull fracture at the time of admission. A left parietal skull fracture is seen with a widely gapped and scalloped edge.

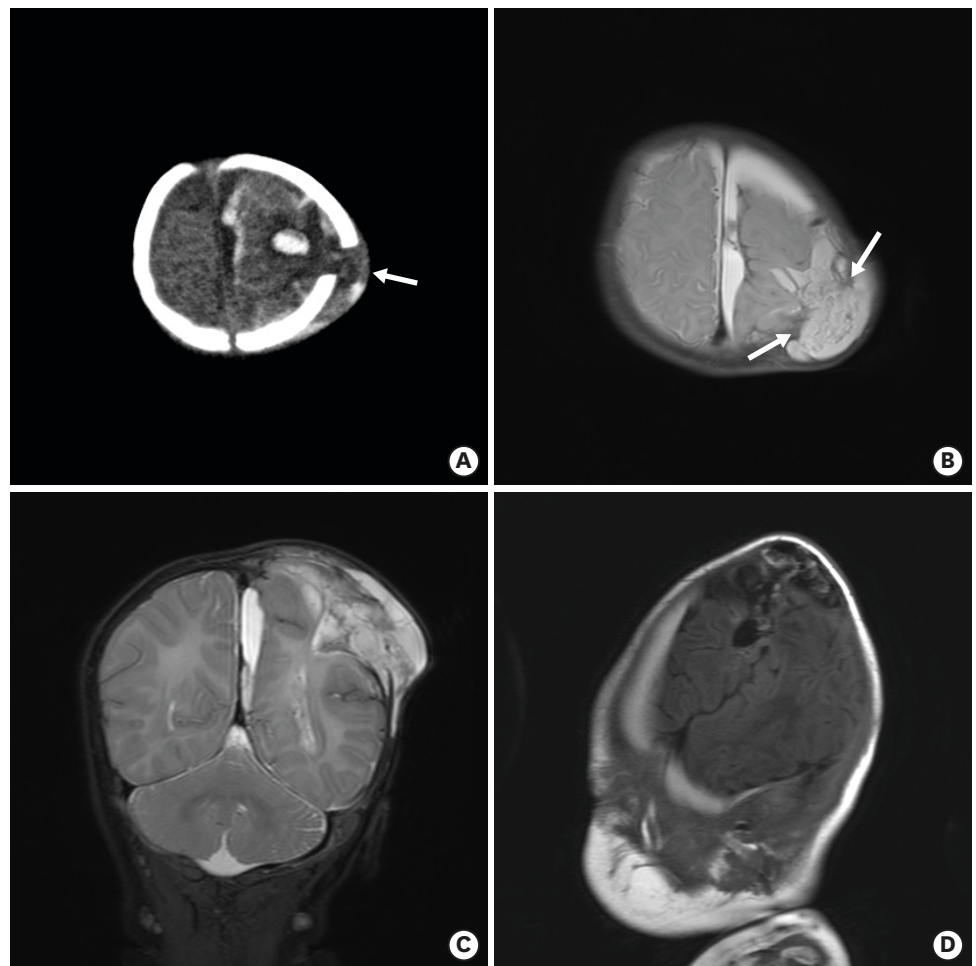


FIGURE 2. (A) Brain computed tomography scan of a 50-day-old patient at the time of admission showing a left parietal bone fracture (13.95 mm-width gap) with underlying hemorrhagic contusion and subdural hemorrhage in the left frontoparietal lobe and falx. The white arrow indicates a zone with mixed density, the parenchymal cerebral contusion, protruding from the fracture into the subgaleal space. (B) T2-weighted axial, (C) T2-weighted coronal, and (D) T1-weighted sagittal magnetic resonance images obtained 12 days after the initial injury presented growth of the fracture edge (white arrows) to a width of 19.91 mm and progressed herniation of the brain tissue through the skull defect, demonstrating progression of the leptomeningeal cyst.

clumsiness of the right fingers. No differences were observed between the side of the arm/calf muscles, foot size, and arch shape. Although a large portion of the motor cortex was damaged on imaging, the patient had an almost intact gross motor function in the right side, with slightly poor fine motor function and ankle dorsiflexion. The intelligence and linguistic levels of the patient were normal. Moreover, the patient did not present with seizure with the use of oral levetiracetam. Consent for publication was obtained from the guardian.

DISCUSSION

GSF is a rare type of skull fracture that commonly occurs in children aged below 3 years and more than 50 percent of it occurs under 12 months.^{8,10,12} The incidence of GSF is reported from 0.05% to 0.6% in pediatric skull fractures.^{8,10,11} The condition is caused by falling, vehicular accident, and child abuse.^{4,10} It is accepted that the most important factors for



FIGURE 3. Preoperative photograph obtained 18 days after injury showing progression of the left parietal scalp bulging.

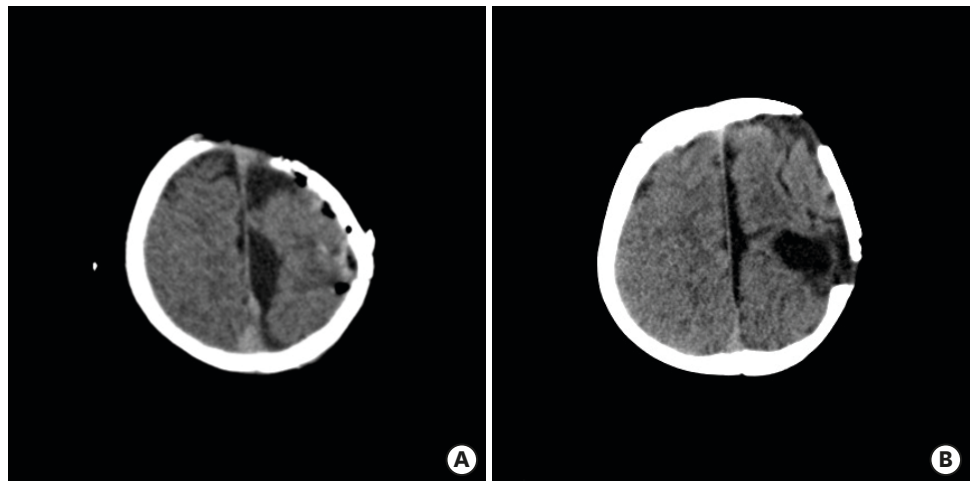


FIGURE 4. (A) Immediate postoperative CT scan showing slightly low density around the left motor cortex after removing the contused and degenerated brain tissue. (B) Postoperative one-year follow-up brain CT scan showing cystic encephalomalacia at the left motor cortex, which was the site of the initial hemorrhagic contusion and gliosis.
CT: computed tomography.

pathogenesis of GSF are the skull fracture with underlying dural tear and the entrapment of the arachnoid membrane with brain tissue through the fracture margin.^{7,12)} The standard treatment of GSF is the surgery that includes cranioplasty and dura repair with a graft.^{4,8,12)}

The symptoms of GSF may include seizure and hemiparesis, impaired vision, scalp swelling at the fracture site, and they may vary based on the site of injury.^{4,8,13)} GSF commonly occurs in the parietal lobe.^{1,7,12)} Thus, the motor cortex and surrounding eloquent area are at risk of damage. Then, extremely young infants have difficulty reporting the subdivided degree of sensorimotor and fine motor activities. However, some reports have shown that the outcome of neurologic deficit may be favorable with treatment in individuals with less than or equal to

stage II disease (I: injury before fracture enlargement; II: within the first 2 months of fracture enlargement; III: after 2 months of fracture enlargement) and ≤type II GSF (I: leptomeningeal cyst herniated to the subgaleal space in the skull defect; II: accompanied by brain damage or gliosis; III: porencephalic cyst).^{7,8)}

Our patient presented with stage II and type II GSF. However, he had significant damage and tissue loss in the left primary motor cortex (M1). Moreover, severe contralateral hemiplegia was expected. But nearly normal activity was possible with almost intact right gross motor function but with slightly poor fine motor function and ankle dorsiflexion during the 30-month follow-up. This result might be attributed to neuroplasticity that occurs before a major transition in motor development and shifting to focal pattern in M1 activity during the early age of infants.^{3,6,9)} Therefore, motor function preservation and a favorable motor outcome can be expected in early infants compared to old infants and adults with motor cortex injury. However, if the leptomeningeal cyst progresses and hemiparesis occurs, a patient is less likely to recover. Thus, prompt and safe surgical treatment is required with collaboration between pediatricians and pediatric anesthesiologists.

CONCLUSION

Herein, we report a case of leptomeningeal cyst on the motor cortex in a 50-day-old child. Although there was a significant damage in the motor cortex, the patient presented with a near normal motor function, except for few fine motor impairments. This result could be attributed to neuroplasticity prior to the complete development of the motor cortex. During this period, a good motor outcome could be expected after a quick surgery.

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