Growing Skull Fracture: A Clinical Study of 15 Children

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Abstract

Fifteen cases of growing skull fractures (GSF) were surgically treated in our department during a period of 4 years. GSF is an entity observed mainly during infancy. The main feature for deciding surgical intervention was enlargement of the skull defect with development of a leptomeningeal cyst revealed by CT or MRI. Surgery should include: Enlarging of craniectomy to find normal borders of dura, adhesiolysis and resection of cerebro-periosteal scar, tight closing of dura and cranioplasty. No post operative seizures or neurological deterioration were seen.

KeyWords: Traumatic leptomeningeal cyst – Growing skull fractures – Diastatic skull fractures – Dural tear.

Introduction

Growing skull fractures are rare complications of head injury, occurring almost exclusively in infants and children under the age of three [8]. Although growing skull fractures have been well described, their pathogenesis remains uncertain [8,12,16]. Characteristic features of this unusual entity include a skull fracture with an underlying dural tear that with time is noted to enlarge, producing a cranial defect [7,8]. Rapid growth of the brain and skull are usually concurrent with the pathologic events due to the population subset [13]. While these elements are typically associated with the growing fracture, other factors including intracranial pressure [3,6,7], leptomeningeal cysts [16], parenchymal injury [5,15], and alterations of cerebrospinal fluid circulation [5,12] have been incriminated as contributing to growth of fractures. The aim of this study is to evaluate the role of early diagnosis and surgical repair in prevention of complications and improving the outcome of growing skull fractures.

Abbreviations:

GCS: Glasgow coma scale.
GSF: Growing skull fractures.

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Patients and Methods

The study included fifteen infants aged 1 to 36 months (seven girls and eight boys; mean age of 17 months) with growing skull fracture were treated from 2008 to 2011 at Neurosurgery Department, Suez Canal University Hospital. Eleven fractures were due to falls and four were involved in a motor vehicle accident. The time range between the initial head trauma fracture ranged from 3 weeks to 6 months. All 15 patients were admitted to the hospital following the initial trauma and growing skull with 8 out of the 15 patients having a GCS of 11 or less.

All patients presented with marked scalp swelling mostly involving the parietal bone, 6 patients presented with seizures, 2 patients presented with contralateral weakness, and 1 patient presented with squint. All patients were examined with skull X-ray. Anteroposterior and lateral skull X-ray revealed wide diastatic fractures. CT scan brain including bone window was done for all patients. MRI brain was performed for all patients without contrast.

Surgical technique included: Enlarging of bony edges on craniotomy in order to find normal borders of dura, lysis of adhesion, resection of cerebro-periosteal scar, exposure of the cyst, opening of the cyst to subarachnoid space or ventricle, closure of the dura and duraplasty with or without cranioplasty. 12 patients were managed with duraplasty only, after separation of herniated cortex from dural edges, skull and surrounding soft tissues. Duraplasty was done using epicranium, and artificial dura. Duro- and cranioplasty were done for 3 patients aged 11 and 16 months with wide diastatic skull fracture. Cranioplasty were done using (autologous bone). Four burr holes around the fracture...
sites and the bones reconstructed and fixed by sutures. One patient had associated hydrocephalus and was treated with ventriculo-peritoneal shunt.

**Results**

In 4 year period from 2008 to 2011, 15 patients underwent surgical repair for growing skull fractures by duroplasty using epicranium, and artificial dura. Clinical data, imaging characteristics, and operative findings in the 15 infants with growing skull fracture were used.

The study included fifteen infants aged 1 to 36 months (seven girls and eight boys; mean age of 17 months). 50% of cases were 1 year of age or less. Eleven fractures were due to falls and four were involved in a motor vehicle accident. All 15 patients were admitted to the hospital following the initial trauma with 8 out of the 15 patients having a GCS of 11 or less. The time range between the initial head trauma and GSF fracture ranged from 3 weeks to 6 months. All patients in this study presented with scalp mass: Six patients (40%) presented with seizure, five (33%) with neurological deficit, four (26%) with loss of consciousness, and one (7%) with hydrocephalus.

Frontoparietal and parietal sites of GSF were (80%). Intraoperatively, the fissure fracture involved the parietal bone alone in 9 cases (60%), the frontal bone alone in 1 case (7%), the frontoparietal bones in 2 cases (15%), and the temporo-parietal bone in 2 cases (15%). The dural defects seen intraoperatively ranged from 4-9cm and in all cases the dural defect extended some distance beyond the bony defect. A dural cyst herniating through the defect without herniating cerebral tissue was encountered in 6 cases (40%).

The patients were followed at out-clinic after discharged from the department on regular visits ranged from 1 month to 12 months. Post-operative status epilepticus occurred in one patient (7%) and was controlled by IV phenytoin. One patient (7%) presented with associated hydrocephalus and was operated upon with ventriculo-peritoneal shunt. Three patients (22%) had post operative superficial wound infection and were controlled with repeated wound dressing and IV antibiotics. No recurrence of leptomeningeal cyst or neurological deterioration occurred. Post-operative skull X-ray and CT scan brain were performed to all patients before discharge (Figs. 1,2).
Discussion

Traumatic leptomeningeal cysts are rare in pediatric populations. Lende and Erickson described four essential features in the diagnosis of growing fractures: 1) A skull fracture in childhood, 2) A dural tear, 3) Underlying brain injury, and 4) Enlargement of a fracture [8]. Taveras and Ransohoff in 1952 proposed that the essential factor was the rupture of dura underlying the fracture site, allowing for the leptomeninges herniating into the defect under the mechanical force of brain pulsation [16]. Localized subarachnoid hemorrhage (SAH) from the initial trauma then allows for arachnoid adhesions to form, causing local cyst formation. This leptomeningeal cyst then augments the mechanical stress on the overlying bone and underlying cortex by providing a more effective pathway for the transmission of brain pulsation.

A dural rupture as the central factor in the development of leptomeningeal cysts is supported widely. In 1941, Penfield stated that absence of the dura will promote erosion of the bone at any age, analogous to the skull erosions associated with arachnoid granulations [10]. In children, the adherence of dura to the skull likely promotes dural rupture more often. In 1967, Goldstein confirmed that a dural tear is important in growing fractures, but also showed that the underlying cyst is important in the pathogenesis [2]. However, extradural cyst formation or encephalomalacia is not always encountered and herniated brain may be present as described by Penfield [10] and Banerji & Tandon [1].

The usual clinical presentation of leptomeningeal cysts in children is one of a non tender, pulsatile scalp mass, or follow-up X-rays showing progressive enlargement of a skull fracture following head trauma. Patients often have neurologic findings consistent with cortical injury underlying the old fracture site. The parietal region is the most common location.

A preoperative diagnosis is often made with clinical evaluation and imaging studies. The findings of progressive enlargement of a previous skull fracture, or bony erosion on radiographs and a pulsatile mass on examination, with a history of trauma is pathognomonic.

It is generally accepted that repair should be undertaken, as the skull and dural defects and leptomeningeal or cerebral herniation tend to enlarge over time [14,17,18]. Others have argued that not all the cases should be repaired, since the associated neurologic findings such as epilepsy and weakness will not be improved by surgery, as they represent the underlying cortical injury [11].

The treatment for leptomeningeal cysts is amputation of the herniated brain or leptomeninges and repair of dural tear, followed by a cranioplasty to cover the skull defect. Most authors advocate amputating or decompressing the cyst as a method to decrease the mechanical forces that facilitate the bone erosion [4,6,9,17,18]. The material used most often for the graft is free pericranium, but Halliday has proposed mobilizing the pericranium circumferentially around the edges and reflecting this tissue over the dural defect [4]. Generally, the erosion of the skull involves the inner table more than the outer table, and the dural tear extends beyond the margins of the bony defect [8]. Therefore, most authors advocate performing a craniectomy until normal dura is obtained. The other option is to perform a craniotomy around the lesion, and to use the craniotomy plate to perform a split thickness bone graft to cover the skull defect.
It could be concluded that all patients under age of 3 years with diastatic skull fracture should be closely followed-up. Children with linear skull fractures should be examined 2-3 months later to look for evidence of a growing skull fracture. Linear fractures and burst fractures in an infant with a scalp swelling must be corrected early to prevent a growing skull fracture. Early management can avoid difficult surgical dissection and progressive neurological sequel seen with delayed intervention.

References