Neurosurg Rev (2000) 23:139–144

ORIGINAL ARTICLE

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Growing skull fractures (craniocerebral erosion)

Received: 5 July 1999 / Accepted: 22 September 1999

Abstract The incidence of growing skull fractures ranges from less than 0.05% to 1.6%. We reviewed 22 growing skull fracture patients retrospectively. There were 15 boys and seven girls ranging in age from newborn to 6 years (mean: 12.4 months) at the time of injury. Falling was the most frequent cause of injury. In total, 17 patients presented with a scalp mass. The scalp was sunken over the bone defect in three patients. Other symptoms and signs were seizure in five patients, hemiparesis in four, recurrent meningitis in one, and pulsatile exophtalmus in one. The most common location was the parietal region. The extent of dural defect was always greater than that of bony defect, except in one case that had been previously shunted for hydrocephalus. In another patient with a growing fracture in the posterior cranial fossa, the dural edges could not be exposed, although a wide craniotomy was performed. Therefore, a cystoperitoneal shunt was inserted. Gliotic tissue was present in all the patients. Cyst or cystic lesions were observed in only nine patients, duraplasty was performed in 21, 16 were neurologically intact, and six had minor deficits. All patients under the age of 3 years with a diastatic skull fracture should be closely followed up. A sustaining diastatic fracture and brain herniation through the skull defect shown on CT or MRI imply a growing skull fracture.

Key words Head injury · Growing skull fracture · Leptomeningeal cyst · Complication · Diastatic fracture

Introduction

The first report of growing skull fracture (GSF) was credited to Howship [1] in 1816. The terms "leptomeningeal cyst," first used by Dyke [2] in 1938, and "growing fracture," first used in the German literature by Pia and Tönnis [3] in 1958, have been used to name the same condition. In their review of the literature, Tandon et al. [4] found many different terms for the same condition, such as meningocele spuria, cephalhydrocele, traumatic ventricular cyst, cerebrocranial and craniocerebral erosion, fibrosing osteitis, cranial malacia, and expanding skull fracture. We reviewed the 22 patients with GSF treated in our institution between 1981 and 1997.

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Patients and methods

In our retrospective review of these patients, age, gender, cause of injury, symptoms and signs, radiological and operative findings, interval between injury and the development of GSF, and outcome were evaluated. Dural and bony defects were calculated by multiplying the maximum length and width. When possible, postoperative EEG and neurological examination were performed in long-term follow-up.

Results

Age and gender

There were 15 boys and seven girls. The patients ranged in age from newborn to 6 years (mean: 12.4 months) at the time of the head injury. The intervals between injury and the development of GSF ranged from 1 to 36 weeks (mean: 7.7 weeks). This interval was not noted in the record of one of the patients, who was operated on for GSF 7 years after the injury.

Cause of injury

Falling was the most frequent cause of injury (17 patients) (Table 1). The other types were motor vehicle accidents (two patients), birth injury (one patient), and penetrating transorbital injury(one patient). In one pa-

tient who had been operated on for traumatic intracerebral hematoma, the GSF was due to a dural tear.

Presenting symptoms and signs

Of the 22 patients, 11 were seen at our institution at the time of injury and, except for the one with transorbital penetrating injury, all had cephalhematomas over the skull fracture. Cephalhematoma was described in the five patients who were referred from other hospitals. In ten of the 17 patients presenting with a scalp mass, the mass was pulsatile. The scalp was sunken over the bone defect in three. The other symptoms and signs were seizure in five patients, hemiparesis in four, recurrent meningitis in one, and pulsatile exophtalmus in one. The GSFs located in the frontal, occipital, parieto-occipital, and temporal regions were not associated with neurological deficits.

Table 1 Etiologies of growing skull fractures in 20 patients

Type of injury	No. of patients
Fall	15
Motor vehicle accident	2
Birth injury	1
Transorbital penetrating injury	1
Postoperative	1

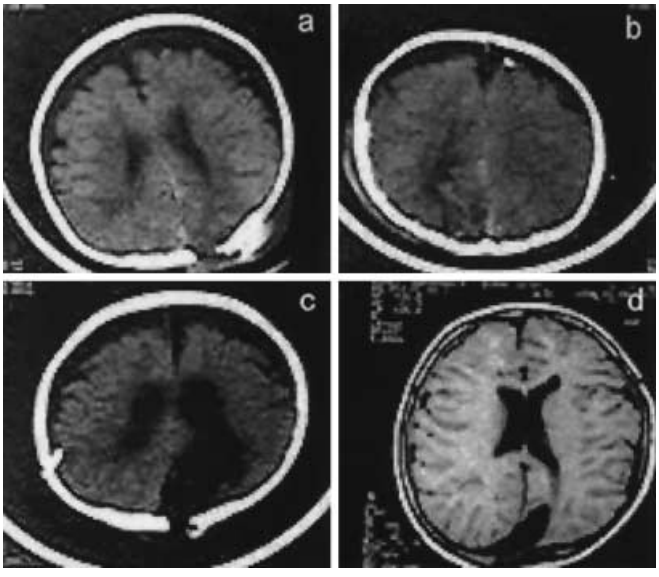

Fig. 1 **a** CT scan showing an occipital diastatic fracture and subdural hematoma. **b** Diastasis of the linear fracture was significantly decreased after subdural drainage. **c** Enlargement of the fracture after the development of hydrocephalus. **d** MRI scan showing a porencephalic cyst beneath the skull defect that had been stable after VP shunting

Radiology

All patients had skull X-rays carried out at presentation. However, skull X-rays obtained at the time of the head injury were available only in the 11 patients who came directly to our institution. The separation of linear fracture on the first X-rays ranged from 4 mm to 12 mm (mean: 6.5 mm). A GSF or skull defect was detected on the plain X-rays of all patients at the time of presentation, except for the case with penetrating orbital injury, whose plain X-rays were not sufficient to disclose the bone defect in the orbital roof.

Computed tomographic (CT) and magnetic resonance imaging (MRI) scans were available in 13 and four patients, respectively. A soft-tissue mass beneath the scalp (five cases), encephalomalacia (ten), cystic parenchymal lesions (four), porencephaly (three), unilateral ventricular dilatation (four), and hydrocephalus (one) were seen on CT scans in addition to the calvarial defects (Fig. 1). MRI scans displayed the parenchymal injury in all four patients. In the patient with a frontoethmoidal GSF, MRI revealed both transfrontal and transethmoidal brain tissue herniation (Fig. 2)

Side and site of the growing skull fractures

GSFs were left-sided in 13 patients and right-sided in nine. The most common location was parietal (ten patients). The other locations were parieto-occipital in three patients, frontoparietal in two, parietotemporal in

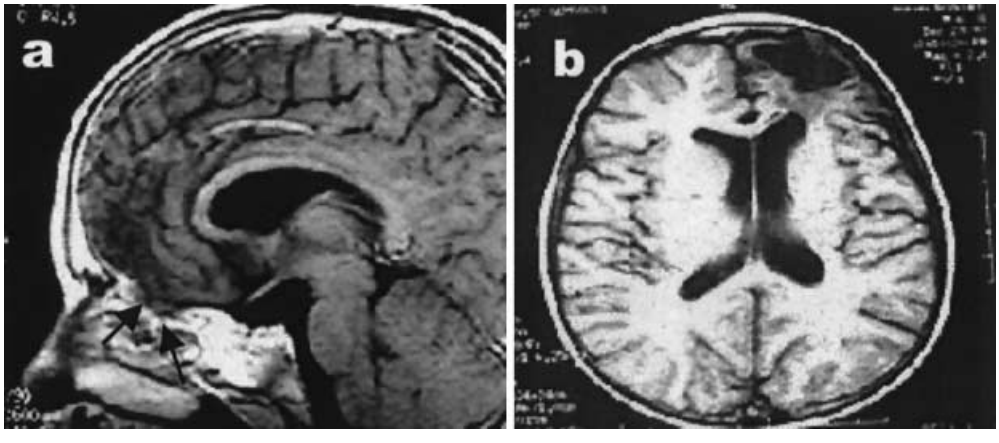

Fig. 2 **a** Sagittal MRI showing transethmoidal brain tissue herniation (*arrows*). **b** Axial MRI showing brain parenchyma herniation through the left frontal skull defect, underlying encephalomalacia, and slight dilatation of the ipsilateral ventricle

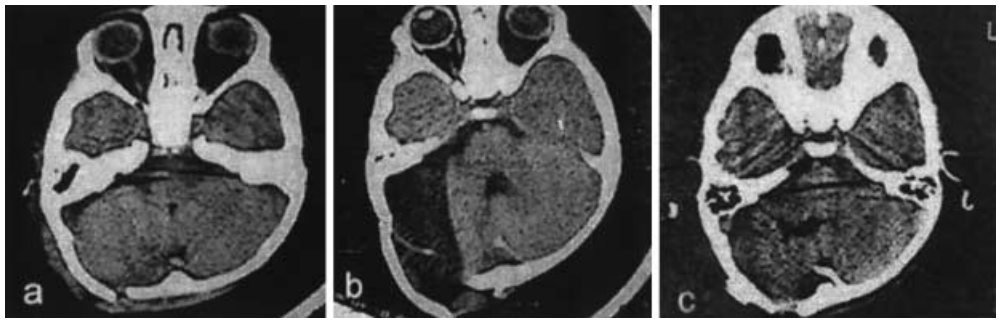

Fig. 3 **a** CT scan showing an occipital diastatic fracture and overlying subgaleal hematoma at the time of injury. **b** CT scan showing a cystic lesion in the right cerebellar hemisphere and the development of growing skull fracture. **c** Postoperative CT scan. Note disappearance of the cyst and expansion of the cerebellar hemisphere after insertion of the CP shunt

Table 2 Location of growing skull fractures

Location	No. of patients
Parietal	8
Parieto-occipital	3
Frontoparietal	2
Parietotemporal	2
Temporal	1
Frontal	1
Occipital	1
Suboccipital	1
Orbital	1
Frontoethmoidal	1

one, frontal in one, temporal in one, occipital in one, suboccipital in one, frontoethmoidal in one, and orbital in one (Table 2).

Operative findings

Craniotomy and craniectomy were performed in 19 and three patients, respectively. The extent of the dural defect was always greater than that of the bony defect, except for the case that had been previously shunted for hydrocephalus ($P<0.0001$), in which the dimensions of the dural and bony defects were found to be the same. In another patient with GSF in the posterior cranial fossa, the dural edges could not be exposed, although a wide craniotomy was performed. Therefore, a cystoperitoneal shunt was inserted. On follow-up CT scan, it was found that the cerebellar parenchyma had expanded and the cyst had disappeared (Fig. 3). Gliotic tissue was present in all patients. Cyst or cystic lesions were observed in only nine (47%). Duraplasty was performed in 21 patients. Pericranium, galea, fascia lata, and cadaveric dura mater (Tutoplast) were used in the repair of dural defects in 13, four, two, and two patients, respectively. Cranial defects were repaired with methyl methacrylate (Codman) in two patients, split calvarial graft in one, and porous polyethylene (Medpor) in one.

Epilepsy and EEG

Of the five patients presenting with seizure, four had late post-traumatic epilepsy. Two of the epileptic patients had associated subdural hematomas at the time of injury. During follow-up, 18 patients received multiple EEG. All of the epileptic patients showed epileptiform discharges on EEG. These were also recorded in two others, even though they had had neither early nor late epilepsy. No significant factor could be determined for the development of epilepsy.

Outcome

There were no deaths in this series. Follow-up of the patients ranged from 3 months to 13 years (mean: 56.8 months). In total, 17 patients were neurologically intact and four had slight hemiparesis. One patient had unilateral blindness due to optic nerve injury from the transorbital stab wound.

Discussion

GSF is a rare complication of head injury. Its incidence reported in the literature ranges from less than 0.05% to 1.6% [5–9], which could have been higher if only infancy and early childhood had been taken into consideration. Lende and Erickson [10] reported that more than half of the GSFs occurred under the age of 12 months and 90% under the age of 3 years. Except for one, all of our patients were 3 years old or younger. However, several cases of GSF have been reported in adults [11–18].

Reasons proposed for why GSFs occur most commonly during infancy and early childhood are that the dura adheres more tightly to the bone in this age group and therefore is more easily torn when the skull is fractured [19, 20], and rapid growth of the brain and skull occurs within the first 2 years of life [21]. Other factors such as intracranial pressure, parenchymal injury, and alterations in cerebrospinal fluid (CSF) circulation may also be contributing factors [15, 22, 23]. In one patient in this series, the diastasis of the fracture decreased following subdural drainage and the increased again with the development of hydrocephalus. The cranial defect remained the same after insertion of a ventriculoperitoneal (VP) shunt. This may also occur with other factors in the development of GSF, such as intracranial hypertension and disturbances in CSF physiology.

Dural tear has been considered the single most important factor in the development of GSF [24–28]. In 1961,

Table 3 Triads in growing skull fractures (*GSF* Growing skull fracture, *MRI* magnetic resonance imaging)

Authors	Indications	Triad
Thompson et al. (1973)	Clinical and radiographic signs	Local scalp swelling Neurological deficit Separation of linear fracture >4 mm
Ito et al. (1977)	Type of tissue in GSF	Granulation tissue type Cyst type Mixed type
Tandon et al. (1987)	Symptoms	Skull defect and/or swelling Persistent or progressive neurological deficit Seizure
Tandon et al. (1987)	Scalp mass	Subgaleal cyst or pseudomeningocele Flattened or sunken scalp Bony swelling or bulge due to eversion of fracture edges or a true intradiploic cyst
Cook and Norman (1988)	Essential features	Diastasis of a skull fracture in infancy or early childhood, commonly in parietal bone Associated dural defect which may be larger than the overlying skull fracture Focal cerebral injury
Scarfò et al. (1989)	Essential features	Head injury with a large gaping fracture Dural tear Occurrence nearly always in infancy
Naim-Ur-Rahman et al. (1994)	Types of GSF	Type I: leptomeningeal cyst Type II: damaged and gliotic brain Type III: porencephalic cyst extending through the skull defect
Havlik et al. (1995)	Mechanism	Cranial bone defect Dural tear Expanding intracranial process (e.g., growth of the brain)
Muhonen et al. (1995)	Pattern of brain herniation on MRI	Solely brain herniating through the fracture Leptomeningeal cyst and brain parenchymal herniation Leptomeningeal cyst herniation
Ersahin et al. (1999)	Predictive signs at time of injury	Cephalhematoma overlying a diastatic skull fracture Separation of linear fracture >4 mm Occurrence during infancy or early childhood

Lende and Erickson [10] emphasized four essential features: (1) skull fracture in infancy or early childhood, (2) dural tear at the time of fracture, (3) brain injury beneath the fracture, and (4) subsequent enlargement of the fracture to form a cranial defect. The linear fracture separation at the time of injury is usually greater than 4 mm [4, 29]. The major enlargement of the fracture occurs about 4 to 8 weeks later. After increasing to a certain size, the bone defect ceases to enlarge further [4, 30]. However, Kutlay et al. [31] and Ziyal et al. [32] reported recently that GSFs may show a progression after a resting period and cause a delayed onset of neurological manifestations. In our patients, the time from injury to the beginning of the enlargement ranged from 1 to 36 weeks (mean: 7.7 weeks). The linear fracture was 4 mm or larger in all 11 patients receiving X-ray immediately following injury. Thompson et al. [29] proposed a triad of clinical and radiographic signs strongly suggesting GSF: (1) local swelling, (2) neurological deficits, and (3) fracture width greater than 4 mm. The majority of the patients in the series of Tandon et al. [4] presented with one or more of the following three complaints: (1) skull defect and/or swelling, (2) persistent or progressive neurological deficit, and (3) seizure. It should be remembered that the scalp overlying a bone defect can appear flat or sunken [4]. The scalp was sunken in three of 21 patients. The swelling can be hard, due to eversion of the fracture edges or a true intradiploic cyst [4, 8, 14, 33]. Interestingly, we have found several triads in the literature regarding GSF (Table 3).

Falling appears to be the most frequent cause of injury. Motor vehicle accidents, child abuse, birth injuries, and previous cranial surgery can also lead to GSF [4, 7, 13, 25, 34–42]. Moss et al. [43] even reported intrauterine GSF. Neurological deficits such as hemiparesis, squinting, and visual field defects may accompany the scalp swelling [4, 7, 16, 27, 30, 35, 37, 41, 44, 45]. Symptoms and signs may also vary with the growing fracture's location. Orbital growing fractures can cause a pulsating exophthalmus, as in one of our patients [11, 46]. Asymptomatic presentation is more common in frontoparietal or frontoparieto-occipital localizations [39]. The patients with GSF in the frontal, temporal, occipital, and parieto-occipital regions had no neurological deficits. A history of seizure was present in almost half of the patients in the series of Tandon et al. [4]. Seizure was common in some series [4, 30, 39]. In this series, almost one fourth of the

patients presented with seizure. Pain is rarely the only symptom and is considered to be due to traction on the blood vessels in the trapped arachnoid membrane caused by the pulsations of the brain [17, 47]. Tandon et al. [4] reported that all their patients had scalp swelling overlying the fracture immediately after the injury. We also observed cephalhematoma overlying the skull fracture in all of our patients admitted at the time of injury.

Plain skull X-ray is an inexpensive and invaluable tool in the diagnosis of GSF. Diastatic skull fracture is always shown on the skull X-rays made at the time of injury [4, 22, 25, 27, 37, 44, 45], and bone defects or lucency are shown after a period of time. CT displays cysts, parenchymal injury, and ventricular morphology in addition to the bony defect. Dilatation of the ipsilateral ventricle was also detected in most cases [3, 4, 7, 13, 26, 27, 30, 37, 38, 41, 48]. Porencephalic cysts in communication with the lateral ventricle have been reported in some [4, 13, 23, 30, 37, 38]. On MRI, Mohunen et al. [7] detected patterns in tissue herniation through the skull defect: (1) solely brain herniating through the fracture, (2) leptomeningeal cyst and brain parenchymal herniation, and (3) leptomeningeal cyst herniation. Husson et al. [49] concluded that a zone of the same density as the brain contusion or CSF advancing through the bone margins to the subcutaneous plane was an indirect sign of dural tear.

Growing fractures are usually located in the parietal region [4, 8, 13, 14, 17, 23, 30, 35, 37, 39, 41]. GSFs of the posterior cranial fossa and orbita are very rare [11, 13, 33, 46, 50–52]. To our knowledge, no case of ethmoidal GSF has been reported in children. Stern et al. [53] reported a 3-year-old boy who had been operated on for encephalocele at the age of 7 days and presented with recurrent meningitis, from whom a large leptomeningeal cyst extending into the left naris was removed. Talamonti et al. [54] reported ten adult patients with ethmoidal fracture in whom relatively large defects of the ethmoid bone were found at the time of repair. They speculated that a mechanism like that of growing skull fractures in children could explain the delayed complications of ethmoid injuries.

The standard surgical treatment of GSF involves repairing the dural defect with a graft and cranioplasty [55]. Most cranial defects are irregular and scalloped. The bony edges are markedly thickened [2, 4, 15, 28, 30]. The dural tear almost always extends beyond the margins of the fracture [4, 7, 22, 49]. The extent of dural defect was significantly greater than that of bony defect, except for one case that had been previously shunted for hydrocephalus ($P<0.0001$). The intervening area is usually filled with fibrous tissue [26, 30, 56] and craniotomy is necessary to expose the dural edges.

Tandon et al. [4] and Kingsley et al. [30] reported that they had never observed a leptomeningeal cyst in their patients. The cystic lesions took the form of extracranial pseudomeningoceles or were found in the fibroglial scar bridging the bone defect [26, 4]. The cysts were not histopathologically leptomeningeal [56]. Pericranium, fascia lata, and cadaveric dural grafts have been used in the repair of dural defects [7, 22, 23, 30]. Naim-Ur-Rahman et al. [23] and Kashiwagi et al. [37] treated patients with shunting when standard surgical treatment failed. Sharma and Chandy [57] treated two cases of GSF with shunting and claimed that this averted major surgery. Their patients improved and regrowth of the bone edges was seen. Gupta et al. [13] performed VP shunting alone in five children. We used a cystoperitoneal shunt in one of our patients with a GSF in the posterior cranial fossa. The cerebellar cyst disappeared and the cerebellar tissue expanded after the shunting. The bony defect remained the same following VP shunting in the other patient associated with hydrocephalus and porencephalic cyst. At the time of repair of dural and cranial defects, it was seen that the dural edges did not extend beyond the bony defect. Naim-Ur-Rahman et al. [23] listed the causes of failure of standard surgical treatment as increased intracranial pressure, either local or global, and failure to secure a watertight closure of the dural defect. Rib grafts, transposition of the craniotomy flaps, acrylic, wire mesh, and steel plates, and split calvarial grafts have been used in cranioplasty [13, 22, 30, 55, 58]. We performed cranioplasty with porous polyethylene (Medpor) in one of our patients.

All patients under the age of 3 years with a diastatic skull fracture should be closely followed up. A sustaining diastatic fracture and brain herniation through the skull defect observed on CT or MRI imply a GSF, since all diastatic fractures will heal if the dura is intact [22, 49]. When a cephalhematoma is associated with an underlying linear skull fracture more than 4 mm wide in an infant or young child, he or she is likely to have a GSF. Parenchymal injury was observed at surgery in all patients. A dural tear is crucial for the development of GSF and there will be damage to both skull and brain tissue. Therefore, the term “craniocerebral erosion” seems to be more appropriate than “growing skull fracture” or “leptomeningeal cyst” as suggested by Roy et al. [56] and Tandon et al. [4].

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