Growing skull fractures: classification and management

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Abstract
Seven patients with growing skull fractures treated between 1983 and 1993 are described. These growing fractures constituted 1.6% of all the cases of skull fractures seen during the period (a total of 449 cases). Based on aetiopathogenesis, computed tomography (CT) appearances, operative findings and management strategies required, three main types of growing skull fractures were recognised. In type I (n = 3) a leptomeningeal cyst, in type II (n = 3) damaged and gliotic brain, and in type III (n = 2) a porencephalic cyst extended through the skull defect into the subgaleal space. A combination of type I and type III co-existed in one patient. Initial head injury and neurological deficit were judged to be mild to moderate in all the seven cases. Continued growth of skull fractures correlated closely to the increasing neurological deficit in five cases. In two patients natural arrest of fracture growth at 5 and 7 months after trauma was accompanied by arrest in progress of neurological deficit. Available surgical options are discussed and general guidelines for the management are given.

Key words: Trauma, leptomeningeal cyst, porencephalic cyst, meningocele spuria, progressive neurological deficit, skull fracture

Introduction
Linear or non-linear skull fractures in children that enlarge with time are termed growing skull fractures. Although these lesions are much more common in children, and 90% occur under the age of 3 years; the process may occur following a skull fracture in an adult. The incidence of 'growth' as a delayed complication of skull fracture is rare and occurred in only 0.6% of the cases in one large series. It is important to realize that the lesion expands not only between the fracture edges, but also intracranially and, thus, may cause atrophy of underlying cerebral tissue with resulting progressive neurological deficit; as was seen in most of our cases. Because of the diverse clinical, radiological (CT) and operative findings and variable temporal course, there is controversy concerning the terminology, aetiopathogenesis and management of growing skull fractures. Classification of the growing skull fractures into three types (Fig. 1) suggested here was found to be helpful in explaining these diversities and planning the treatment. Similarly, based on the clinical presentation and temporal course, two forms of the growing skull fractures could be distinguished. An active form with evidence of raised intracranial pressure (ICP), mass effect on CT, progressive separation of bone edges with a tense bulge between them; and an ar-
rested or 'burnt out' form with a normal ICP, and a slack/sunken gap between the bone edges often showing some evidence of bone regrowth and healing at the fracture site. Duro-cranioplasty was the correct treatment for type I fractures without raised intracranial pressure; whereas a shunting procedure was required as an initial or definitive management for type I and III fractures with raised intracranial pressure.

**Report of cases**

A summary of the seven patients is presented in Table I and management options summarized in Table III.

**Clinical Data**

Six patients were children while one was 39 years old. In the paediatric group, the age at the time of diagnosis ranged from 4 months to...
Growing skull fractures

6 years (mean: 24.6 months). Six patients were female and one male. The interval between the history of head injury and diagnosis of growing skull fracture ranged from 2 months to 1 year (mean: 7 months). Location of the skull fracture was parietal in three patients, frontoparietal in one, occipitoparietal in one, and occipital in the remaining two children.

Two children (cases 1 and 2) had experienced permanent remissions after a period of active fracture growth. The remaining five showed evidence of raised intracranial pressure and more or less progressive neurological deficits. Five children presented with swelling in the region of the fracture site, while in two (cases 1 and 7), the area between the gaping bone edges was slack and depressed. Seizures and hemiparesis were the next common presenting symptoms and occurred in four patients. Blindness or progressive visual deterioration was present in two children with occipital fractures, while retardation was seen in one child.

Radiological evaluation

Plain radiographs of the skull and CT were performed on all patients. Skull radiographs correctly demonstrated the location and extent of separation of bony edges at the fracture site. Extensive bony resorption with widely gaping everted fracture margins, seen in three children, correlated well with the size of the underlying cyst and brain damage on a CT scan.

CT clearly demonstrated the bony defect, the nature of intracranial contents herniating through it (leptomeningeal cyst = 3, contused cerebral tissue = 3, porencephalic cyst = 2); and the extent of the underlying intracranial lesion. Thus, a leptomeningeal cyst with CSF density was seen in three patients, extensive low density areas of brain damage and encephalomalacia in three patients and porencephalic cysts communicating with dilated ventricles in two children.

Operative findings

Five of the seven patients underwent surgery. One child (case 4) had a ventriculoperitoneal shunt for tense porencephalic cyst and dilated ventricles. In the remaining four children the defect was repaired by duroplasty using fascia lata or pericranial graft, and an acrylic cranioplasty. Of these four children who underwent craniotomies at the fracture sites, two (cases 2 and 4) showed that the scalp and pericranium were densely adherent to the underlying congested, gliotic and herniated brain. The dura was totally absent under the cranial defect. More or less extensive nibbling of the margins of bone defect was required to find a reasonable dural edge. Dissection and elevation of the scalp flap from the underlying congested gliotic brain was tedious and accompanied by much bleeding. In case 5, operation was performed for a large tense occipital swelling through a growing fracture of the skull. Elevation of a large occipital scalp flap revealed a porencephalic cyst on the right side and a leptomeningeal cyst overlying a congested and softened left occipital lobe. The occipital cyst was aspirated and an acrylic cranioplasty carried out. Durocranioplasty was straightforward in the seventh case.

Surgical results

Complete resolution of the raised intracranial pressure, extracranial swelling and progressive closure of skull defect was obtained in the child who underwent shunt surgery (case 3). Follow-up at 3 and 6 months has shown reduction in the size of cranial defect with evidence of bone regrowth at the fracture site. Complete obliteration of the cranial defect was achieved in three out of four patients who underwent duro-cranioplasty (mean follow-up: 4 years). In the fourth patient, the acrylic plate loosened and bulged 2 years after surgery. Reoperation was refused. All the five patients who underwent surgery recovered without any increase in the pre-existing neurological deficits and definite improvement was recorded in four patients.

Case 1. This 30-month-old girl was well until 10 months before admission when she was
<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age/sex*</th>
<th>Location of fracture</th>
<th>Type &amp; temporal classification</th>
<th>Presentation and clinical examination</th>
<th>Skull radiography</th>
<th>CT appearance</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 months</td>
<td>30 months</td>
<td>Parietal</td>
<td>I Arrested (burnt-out)</td>
<td>Slack depression in the region of fracture improving left hemiparesis, Retardation</td>
<td>Gaping bone defect with everted margins</td>
<td>Atrophic (drained) right parietal leptomeningeal cysts with ipsilateral pulling of midline</td>
<td>Conservative</td>
<td>Being followed</td>
</tr>
<tr>
<td>72 months</td>
<td>F</td>
<td>Parietal</td>
<td>II Arrested</td>
<td>Slack swelling right parietal region seizures, minimal left hemiparesis</td>
<td>Gaping defect right parietal</td>
<td>Low density, atrophic cerebral tissue underlying skull defect</td>
<td>Duroplasty and acrylic cranioplasty</td>
<td>Cured!</td>
</tr>
<tr>
<td>4 months</td>
<td>F</td>
<td>Occipital</td>
<td>II Active</td>
<td>Tense, increasing bulge overlying left occipital bone defect, RICP, vomiting papilledema, ?Blindness</td>
<td>Large, wedge-shape bone gap with extensive retraction</td>
<td>Large left occipital porencephalic cyst communicating with the dilated lateral ventricles and herniating through the occipitoparietal defect</td>
<td>Cysto-ventriculoperitoneal shunt</td>
<td>Cured!</td>
</tr>
<tr>
<td>36 months</td>
<td>F</td>
<td>Frontoparietal</td>
<td>II Active</td>
<td>Tense swelling left forehead, occasional headaches and vomiting, seizures, Q progressive) right hemiparesis</td>
<td>Extensive gaping fracture left fronto-parietal involving anterior cranial fossa</td>
<td>Low density atrophic cerebral tissue herniating through gaping fracture</td>
<td>Duroplasty and acrylic cranioplasty</td>
<td>Recurrence! after surgery</td>
</tr>
<tr>
<td>15 months</td>
<td>F</td>
<td>Occipital</td>
<td>I &amp; III Active</td>
<td>Tense enlarging swelling occipital region, blindness, progressive neurologic deficits</td>
<td>Gaping, biocipital bony defects</td>
<td>Bilateral occipital cephalomalacia, large right occipital porencephalic cyst communicating with dilated lateral ventricles and herniating through bone defect or left leptomeningeal cyst</td>
<td>Acrylic cranioplasty followed by recurrence, followed by repeat duro-cranioplasty</td>
<td>Improved! Followed-up to 3 years has defective vision V.E.P. = poor response = cortical blindness</td>
</tr>
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TABLE II. Management options

Growing skull fracture

<table>
<thead>
<tr>
<th>With normal ICP</th>
<th>With raised ICP</th>
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</thead>
<tbody>
<tr>
<td>Tense porencephalic cyst with hydrocephalus</td>
<td>Tense leptomeningal cyst</td>
</tr>
<tr>
<td>Ventriculoperitoneal shunt</td>
<td>Cysto-peritoneal shunt</td>
</tr>
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<td>I</td>
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Duro-cranioplasty

treated for head injury with a fracture of the skull. Over the next 5 months the child developed bulging of the scalp in the right parietal region which became progressively larger. This was accompanied by progressive left hemiparesis, headache, vomiting and occasional seizures. Five months prior to admission the mother noticed a spontaneous improvement in the child’s condition. The scalp bulge became softer then rapidly disappeared leaving a depressed gap between the bone edges. This was accompanied by resolution of headache and vomiting, and improvement in the left-sided weakness. The child was admitted to the neurosurgical unit with the chief complaints of a soft gap on right side of head, seizures and residual left hemiparesis. Examination revealed a parietal bony defect with a slack depression, in an alert girl walking independently. Neurological examination showed minimal left hemiparesis with pyramidal signs and mild retardation. Skull radiographs on admission showed a gaping right parietal fracture with everted saucerized margins. CT (Fig. 2) showed a leptomeningeal cyst underlying the cranial defect and communicating freely with the perimesencephalic cistern. As there was evidence of regrowth of bone edges (Fig. 2) along with continued improvement in her neurological status, it was decided to observe. At 3-month follow-up the bone gap had narrowed.

Case 2. This 6-year-old girl presented with seizures and a ‘soft area’ over the right parietal region, noticed for the last 4 years. A head injury had occurred when the child was 10 months old. One year after the head injury the parents noticed a soft, gradually increasing bulge in the right parietal region which gradually ‘flattened’ in time leaving a ‘soft area’. There was no history of headache or vomiting. Examination revealed an alert, bright child. A skull defect in the right parietal region measuring about 4 cm and covered by slack scalp could be felt. Neurological examination revealed minimal left hemiparesis with brisk tendon jerks. There was no papilledema. Skull radiograph (Fig. 3) showed a gaping fracture in right parietal region. CT (Fig. 4) showed a low density lesion of the underlying brain with slight dilatation and ipsilateral ‘pulling’ of the right lateral ventricle. At operation, a large,
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Case 1: CT scan showing leptomeningeal cyst underlying cranial defect and its communication with the perimesencephalic cistern (arrow). Note regrowth of fracture edges.

right fronto-parietal scalp flap was raised. Dense adhesions between the scalp and underlying brain were dissected. Fracture edges were nibbled to expose normal dura. Tissues were abnormally vascular and bled excessively. Duroplasty and acrylic cranioplasty was carried out. Postoperatively the patient did very well and was discharged home on phenytoin. Three years later, she is asymptomatic and free of seizures.

Case 3. This 6-month-old girl was seen in the neurosurgical clinic with the history that she fell and struck the back of her head on a concrete floor at the age of 1 month. Shortly afterwards, a bulge was noticed in the left occipito-parietal region. This bulge became progressively larger and tense. On admission the child was irritable, refused feeds and had been vomiting for past few days. Skull radiographs showed a widely gaping wedge-shaped bone defect with extensive resorption in the left occipito-parietal region. CT (Fig. 5a and b) showed a large porencephalic cyst communicating with the occipital horn of the dilated left lateral ventricle and herniating through the wide bony gap into the tense occipital bulge. The child was treated with ventriculo-peritoneal shunt following which the occipital bulge and hydrocephalus resolved. At follow-up examinations at 3 and 6 months, the child was thriving, the occipital bulge had disappeared and the bone defect was much smaller.

FIG. 2. Case 1: CT scan showing leptomeningeal cyst underlying cranial defect and its communication with the perimesencephalic cistern (arrow). Note regrowth of fracture edges.

FIG. 3. Case 2: skull radiography, lateral view, showing a widely gaping parietal fracture.

FIG. 4. Case 2: CT scan showing the right parietal bone defect and the underlying low-density brain lesion.
with definite evidence of bone re-growth at the fracture edges.

Case 4. This 3-year-old girl was originally admitted and treated elsewhere for a linear fracture of left fronto-parietal region. Seven months after the original head injury, she was admitted to our neurosurgical unit because a bulge had developed over the left forehead which became progressively larger. There was a history of occasional headaches, vomiting, seizures and progressive right hemiparesis during the preceding 3 months. On examination there was a tense linear swelling in the left forehead extending from the left fronto-parietal region to the supra-orbital margin and root of the nose. The margins of the underlying skull defect could be palpated. She had mild weakness of right arm and leg but no papilledema. Skull radiograph (Fig. 6) showed a linear defect in the left fronto-parietal region extending to the supraorbital margin. CT (Fig. 7) showed the gaping fracture and an underlying low density brain lesion. At operation, a large frontoparieto-temporal scalp flap was raised. Underneath the flap an adherent linear bulge of gliotic brain was seen between the gaping fracture edges. This herniated cerebral tissue, extending from coronal suture to the supra-orbital margin, was tense, congested and bled easily. Dural repair with fascia lata and cranioplasty with methyl methacrylate was carried out. The postoperative course was uneventful and the child was discharged home, free of headache, but with residual mild right-sided weakness. Follow-up at 2 years showed that the implanted plastic was loose and bulging over a tense cystic swelling. Reoperation was refused.

Case J. The patient was originally admitted to the neurosurgical unit at the age of 9 months following a road traffic accident. On admission, she was drowsy, but without any abnormal neurological findings. A radiograph of the skull showed an obliquely placed fissure fracture of the skull crossing the midline just above torcula and CT showed this fracture with minimal contusion of the occipital lobes (Fig. 8). The baby recovered rapidly from the head injury and was discharged home after 5 days. Six months later (at the age of 15 months) she was re-admitted with a tense swelling in the region of the occipital fracture site and a suspicion of progressive visual loss. The occipital swelling had been gradually enlarging during the weeks prior to re-admission. Examination now revealed that although the
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FIG. 6. Case 4: skull radiograph, AP view, showing a gaping linear defect (arrows) in the fronto-parietal region.

FIG. 7. Case 4: CT scan showing the left frontal growing fracture (arrows) and the underlying low density brain lesion.

child could see, the vision was clearly defective as shown by her inability to fixate and lack of reaction to visual stimuli. Reaction of the pupils to the light was very sluggish but equal. Visual evoked potentials (VEP) confirmed cortical blindness. Plain radiography of the skull on this second admission showed a large, occipital bony defect with scalloped margins (Fig. 9). CT at this stage showed resorption of a portion of the occipital bone and underlying brain lesions (Fig. 10). At surgery, the CSF-containing occipital cyst was aspirated and an acrylic cranioplasty carried out. This child was re-admitted 2 months later, this time with a collection of fluid under the acrylic cranioplasty. A further operation was carried out. A water-tight repair of the dural defect was achieved using a pericranial graft and a fresh cranioplasty was performed. The postoperative course was uneventful and the wound healed satisfactorily. Review 3 years later showed defective vision.

Case 6. This child was hospitalized for 3 days for head injury at the age of 5 months. Skull radiography and CT showed a left parieto-occipital fissured fracture of skull with minimal underlying contusion (Fig. 11). Nine months later she presented with a large skull defect and tense scalp swelling at the site of the fracture, along with increasingly frequent seizures and delayed speech. Radiography now showed a large, gaping skull defect at the site of original fracture (Fig. 12) and CT showed the gaping fracture with hypodensity of the underlying brain and dilatation of the ipsilateral ventricle (Fig. 13). Surgery was refused in spite of progressive neurological and radiological deterioration.

Case 7. A 39-year-old woman was seen for increasing right-sided head pain. At the age of 18 years she had suffered a head injury in a car accident. On examination, a large gaping skull defect was present in the right parietal region. Skull radiography showed a widely gaping right parietal defect (Fig. 14). CT showed the skull defect and underlying leptomeningeal cyst (Fig. 15). Durocranioplasty was straightforward, and resulted in resolution of headache and closure of the skull defect.
Discussion

Growing skull fracture has been described in the literature as an entity synonymous to a leptomeningeal cyst. Thus, a posttraumatic extra axial leptomeningeal cyst was assumed to be responsible for the progressive enlargement of the skull fracture. The cases described here suggest that the leptomeningeal cyst is not a consistent feature and that a variety of progressively evolving morphological and pathological changes in the underlying brain may play an important role in the genesis of growing fractures. Morphologically, the predominant factor responsible for fracture growth may lie in the subarachnoid space (a leptomeningeal cyst), cerebrum (herniated brain) or ventricle (dilated underlying ventricle with porencephalic cyst). These events constitute the morphological basis for the fracture types I, II and III, respectively.

At the initial trauma, distortion of the malleable infant skull on impact generates pressure fields within the cranium, leading to a skull fracture and tearing of the tightly adherent underlying dura. Six of the seven patients in our series and most in other series were children. Thus, malleability of the infant skull and tighter adherence of dura to the bone in children may account for the common occurrence of growing fractures in this age group.

Pathogenesis of the leptomeningeal cysts, as seen in our cases 1, 5 and 7, is well described elsewhere. In type II fractures, as seen in our cases 2, 4 and 6, cerebral pulsations erode the fracture edges, and drive the cerebrum through the dural vent and bony gap. With time, the herniating and the underlying brain suffers progressive damage as shown by local atrophy and cephalomalacia. In type III fracture (cases 3 and 5), the underlying ventricle expands into a porencephalic cyst. The entire ventricle may dilate as was seen in case 3 or just the one horn subjacent to the growing fracture may dilate as occurred in case 5. In these cases, the porencephalic cyst extends from ventricular wall through the skull defect and into the subgaleal space, and is roofed by a thin layer of softened gliotic cortex.

In most of the reported cases the initial head injury was a minor one and focal neurological deficits were rare. Thus, at the time of initial skull fracture, only two of our patients showed minimal hemiparesis. However, over the ensuing weeks or months, all of these children showed evidence of more or less progressive neurological deficits. Neurological status and fracture stabilized, after a period of progression and growth, in two children. This
FIG. 10. Case 5: six months later. CT scan showing marked widening of the occipital fracture with scalloping of the margins and right occipital porencephaly and a left occipital leptomeningeal cyst.

resolution was quite dramatic in case 1, and was thought to be due to rupture and drainage of the leptomeningeal cyst into the perimesencephalic cistern (Fig. 2).

The natural history and temporal course of the growing skull fractures varied from case to case. The rapid progression with raised ICP seen in case 3, and spontaneous arrest and healing seen in case 1, appear to be the two ends of a spectrum. Gradually progressive neurological deficits with increasing underlying brain damage was the more common occurrence and was seen in four of our patients.

In active growing skull fractures, a tense overlying scalp bulge may indicate raised ICP which may be caused by a tense leptomeningeal/porencephalic cyst, or posttraumatic hydrocephalus. Slack scalp depression at the fracture site with evidence of bone regrowth and fracture healing, along with the arrest of the previously progressive neurological deficits indicates spontaneous resolution of a leptomeningeal cyst, as was seen in case 1. Gradually progressive focal neurological deficits, seen in four children, coincided closely with the increasing separation of the bone edges at the fracture site and CT evidence of extension of the underlying brain damage. The nature of these focal deficits depended on the fracture site. Thus, the two children with occipital fractures (cases 3 and 5) suffered progressive visual loss, while hemiparesis was seen in the cases with frontoparietal fractures.

Skull radiography and CT were the two most useful investigations for the diagnosis and monitoring of growing skull fractures. Thus, at the time of the initial trauma, all the seven patients had skull radiographs which showed linear fractures. The initial head injury was not considered serious enough to warrant CT in the first instance, except in cases 5 and 6 where CT at the time of trauma confirmed the fissured skull fractures without evidence of significant contusions or damage to the underlying brain (Figs. 8 and 11). Delayed CT showed the fracture growth (Figs. 10 and 13) and, more importantly, appearance of extensive fresh lesions with marked and possibly continuing damage to the underlying brain (Figs. 10 and 13). CT correctly demonstrated the nature and extent of the intracranial pathology including areas of cephalomalacia, subdural, leptomeningeal and porencephalic cysts, as well as ventricular dilatation (complete or only one horn).

Because of the variable pathological findings, the standard surgical procedure described in the literature for the treatment of the growing skull fractures is not justifiable or applicable in all the cases. Standard surgical approach involves resection of the leptomeningeal cyst and herniated brain, repair of the dural defect with a graft and cranioplasty. For the straightforward cases without any evidence of raised ICP, such as our cases 2 and 7, this procedure is curative. We have used pericranium/fascia lata and methyl methacrylate to repair dural and cranial defects in our cases; but others have used split
calvarial bone, ribs, iliac crest and metallic materials with satisfactory results. However, children under 2 years of age have a skull bone thickness that is too difficult to split in order to repair the defect. In some patients, such as our case 4, the dural defect extends well beyond the skull defect, necessitating rongeuring bone edges for long distances in order to expose reasonable dural edges to which the graft can be stitched. In others, such as our case 5, one may be obliged to resect the cortex which roofs the herniating porencephalic cyst. This extensive enlargement of the skull defect and resection of the possibly...
FIG. 15. Case 7: CT scan showing right parietal skull defect overlying a leptomeningeal cyst.

functional and viable areas of the infant brain appear to be somewhat excessive. In these circumstances, the modified method for repairing this defect, as described by Halliday et al., seems more appropriate. This technique involves mobilizing the pericranium circumferentially around the edges of the bone defect and reflecting it over the site of the dural defect to provide tissue for repair.

In growing skull fractures associated with raised ICP, shunt surgery should be considered as an initial or alternative procedure, as it may result in resolution of raised ICP, disappearance of scalp swellings and regrowth of bone edges at the fracture site; as illustrated by case 3.

Recurrence after surgical repair in two of our patients and other reported cases correlated with failure to secure watertight dural closure or failure to address the raised ICP.

Because of the known risk of fracture growth along with the frequently progressive nature of brain damage and neurological deficits, children with linear skull fractures should be examined clinically 2-3 months later to check for evidence of growing fracture. If a scalp bulge or gaping bone defect is found, radiography and CT should be repeated. If growing skull fracture is confirmed, surgical repair should be advised.

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References


