

GROWING SKULL FRACTURES

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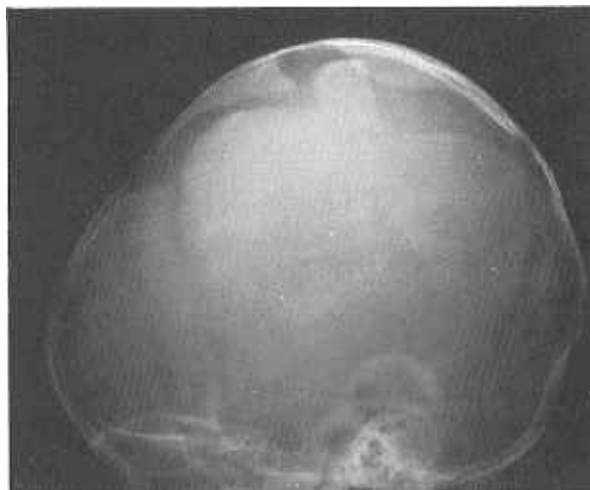
ABSTRACT

Two recent cases of growing skull fractures are presented and the literature reviewed. Skull fractures in children which are at risk of enlarging should be recognised and followed up closely.

Keywords : Skull fracture, growing.

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Fig 1 - Case 1. X-ray skull A-P and Lat. Right parietal fracture.



INTRODUCTION

Growing skull fractures are rare complications of skull fractures in children. They are characteristically associated with encephalomalacia, leptomeningeal cyst, dural defect and enlarging skull defect. Pathogenetic mechanisms have been described and treatment is sometimes controversial.

Case 1

An 8-month-old child was transferred to our department in December 1989 after a motor car accident. He had very mild weakness of the left limbs. There was a large right parieto-temporal cephalhaematoma. Skull X-ray revealed a right parietal fracture 8 cm long with a 3 mm gap (Fig 1). CT head scan

revealed a thin right parietal subdural haematoma and ipsilateral hemispheric oedema with mild midline shift (Fig 2).

He was treated symptomatically for pain and vomiting. A repeat CT scan 7 days later revealed bilateral frontal and right falcine hygromata (Fig 3). This was treated conservatively.

On follow-up, his milestones were normal. A right parietal skull defect was palpable at 6 months post trauma. He returned for review 2 years later by which time the defect had grown to 8 cm by 5 cm (Fig 4). CT scan revealed the bony defect in the right parietal region overlying a 5 cm leptomeningeal cyst which herniated through the defect. The right cerebral hemisphere showed mild atrophy (Fig 5).

The growing skull fracture was repaired in March 1992. A subgaleal parietal scalp flap was raised around the pulsatile mass. The bony defect measured 8 cm by 5 cm. On opening the pericranium over the defect, clear fluid from a cyst drained out. The deep aspect of the cyst was gliotic brain tissue. Dura was deficient over the cyst and had retracted beyond the margins of the skull defect. To minimise bone loss, dura was not traced peripherally and autogenous pericranium was used to cover the cerebral cicatrix. An acrylic cranioplasty was then molded to cover the skull defect.

Post-operative recovery was uneventful. CT scan showed that the cranioplasty had covered the defect and the cyst had obliterated after drainage (Fig 6). The child remained neurologically intact.

Case 2

This was a three-month-old child referred to us from elsewhere. He sustained right parieto-occipital skull fracture at the age of one week when his mother fell onto his head. CT scan showed traumatic subarachnoid, intracerebral and intraventricular haemorrhage. He was treated conservatively. Three months later, he developed a cystic scalp mass in the

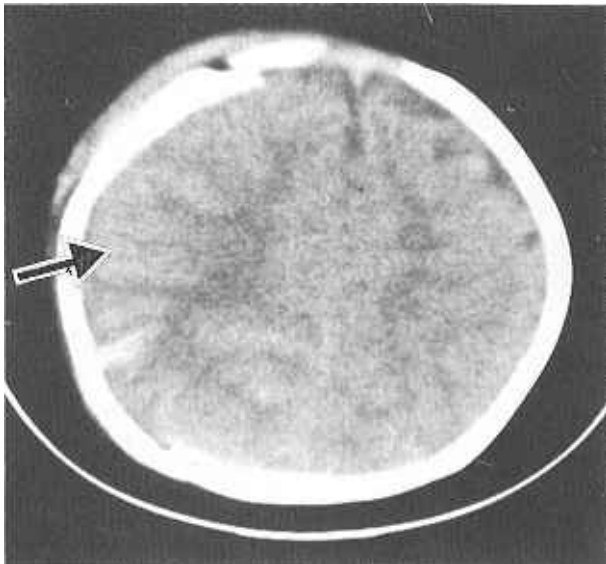
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Fig 2 - Case 1. CT head scan. Thin right parietal subdural haematoma and ipsilateral hemispheric oedema with midline shift.



right parietal region. Skull X-rays showed a large right parietal defect. Operation was carried out by the referring neurosurgeon. A 3 cm diameter dural defect was noted for which duroplasty was done. Post-operative CT scans showed mild atrophy of the R hemisphere with grossly dilated right lateral ventricle in communication with a porencephalic cyst.

At our hospital, a right ventriculo-peritoneal shunt was performed to relieve hydrocephalus. At operation the CSF pressure was not high. He was discharged well. He was due to be seen at our outpatient clinic to assess the need of cranioplasty but he did not turn up.

DISCUSSION

Historical Review⁽¹⁾

Growing skull fractures have been described since the last century. The names given by these early investigators reflected their concept of the pathology of these lesions.

The earliest, "partial absorption of the parietal bone, arising from a blow on the head" in a child aged 9 months, was reported in 1816 by John Howship. He ascribed the lesion to a local debility in cranial circulation.

"Cephalhydrocoele" was the term used by Rokitansky and Weinechner in 1856 to describe a 6 x 4 cm cranial aperture overlying a dural defect and brain injury through which herniated a fluid filled sac.

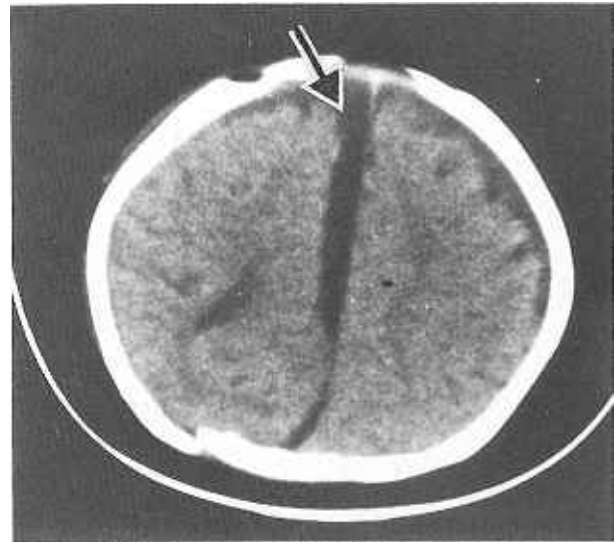
The term "meningocoele spuria" was used by Billroth in 1862 for a cerebrospinal fluid collection under the scalp which grew over 2 years after traumatic forceps delivery to nearly the size of the head itself.

A "traumatic ventricular cyst" was described by Sir Wilfred Trotter in 1923 in which a local cystic dilatation of the lateral ventricle extended out to the cranial defect, also associated with previous trauma. This is similar to the second case we reported.

"Leptomeningeal cysts", as proposed by Dyke in 1937, were formed from loculated fluid-filled spaces. Owing to the pulsations of the brain, the overlying bone is absorbed. This concept of pathogenesis became the major influence on subsequent case reports.

Pancoast et al in 1940 used the term "fibrosing osteitis"

Fig 3 - Case 1. CT head scan. Bilateral frontal and right falcine hygromata.



which was based on pathological study of biopsy material. They considered the cause to be disturbance in blood supply secondary to injury.

"Cranio cerebral erosion", a term used by Penfield and Erickson in 1945, was the alternate pathogenetic mechanism, where pulsating brain tissue rather than leptomeningeal cyst came in between and separated the fracture edges.

Most importantly, these terms depicted individual features of a spectrum of lesions which are now often termed as "growing skull fractures" after Pia and Tonnis, which was preferred for its lack of pathogenetic implications.

The early surgeons needled and aspirated these cysts, injected iodine into them, causing death in some unfortunate patients.

Of prehistoric note is the suggestion of Alajouanine and Thurel, that certain ancient skull openings, presently interpreted as primitive trephining, actually may be defects that followed fractures of infancy.

Review of reported cases

Of the 25 cases reported in the reviewed literature⁽¹⁻⁶⁾, the male:female ratio was 2:1 and the average age of the patient at the time of trauma was 2 years 9 months (ages ranged from 3 weeks to 20 years). The time between injury and treatment averaged 2 years 11 months.

The defect was in the parietal bone in 19 out of the 25 cases, and extended to the frontal, temporal, or occipital in 4 cases. A frontal defect was found in only 2 cases. Occipital defects were rare and considered as a separate entity⁽⁷⁾. The defects were equally distributed to the right and left.

Slightly less than half had hemiplegia and two were mentioned to be mentally retarded. Eight patients had epilepsy of which half were of early and half of late onset.

Of the 25 cases, five were not treated⁽⁶⁾, six cases had duroplasty alone, six had cranioplasty alone and eight cases had both duroplasty and cranioplasty. In three of the six who had only cranioplasty, the authors emphasised that the cerebral scar was not entered, presumably to minimise cerebral damage⁽¹⁾.

Cranioplasty was performed using tantalum in five, autogenous rib in two, acrylic in three and steel in one case. One of the rib cranioplasties resorbed necessitating revision,

Fig 4 - Case 1. Six months post trauma. X-ray skull A-P and lat. The parietal defect had grown to 8 cm by 5 cm.

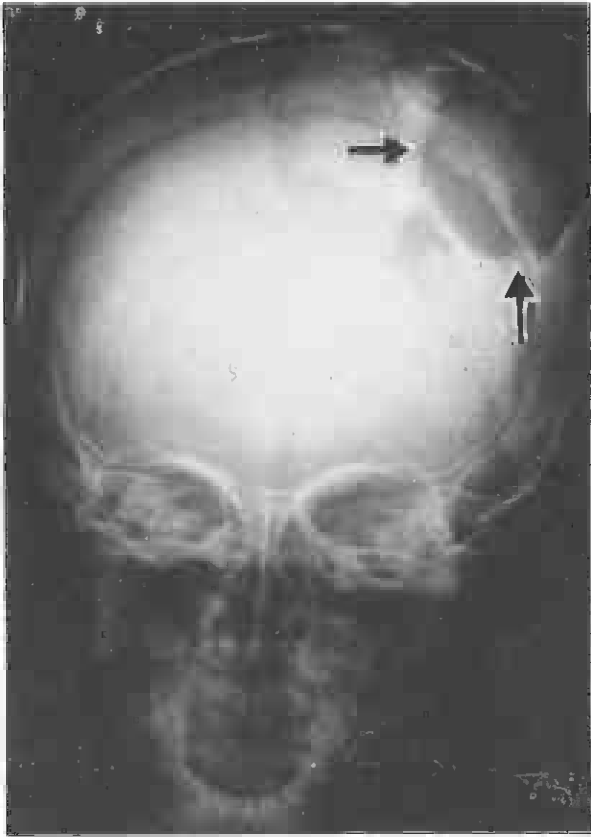
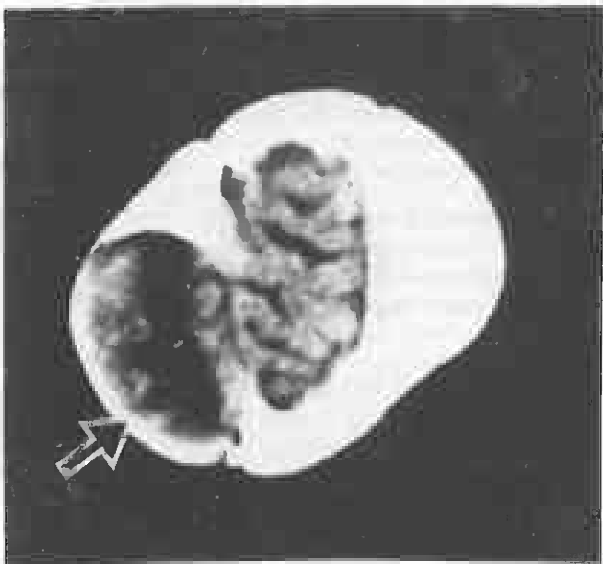


Fig 5 - Case 1. Six months post trauma. CT head scan. A bony parietal skull defect. A leptomenigeal cyst herniating through the defect.



and infection occurred in one of the tantalum cranioplasties. Fits occurred in one case after cranioplasty without duroplasty.

The cerebral scar was dealt with in several ways. The cysts were communicated with each other and to the underlying ventricle in five cases⁽⁵⁾. Cysto-atrial shunts were performed for two cases⁽³⁾. Cysts were uncapped in five cases⁽⁵⁾. The cysts were surgically communicated to the underlying ventricle in two cases⁽³⁾.

Fits ceased in five cases after duroplasty and cranioplasty

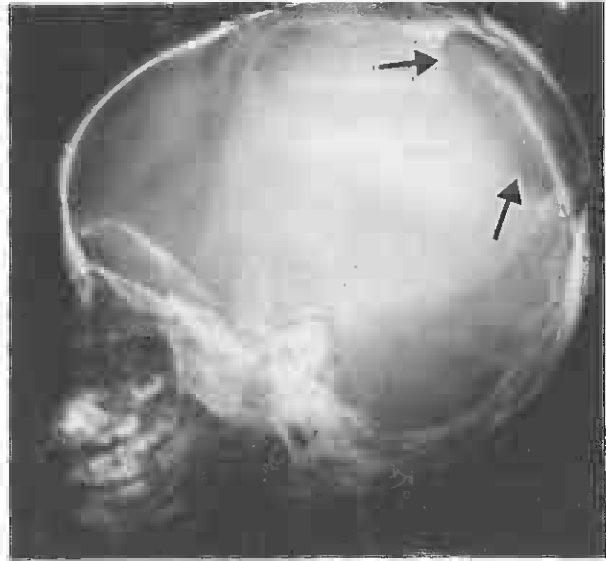


Fig 6 - Case 1. Post operative head CT head scan. Acrylic cranioplasty shown. (Air within the acrylic plate is a normal finding) The leptomenigeal cyst had been obliterated by operative drainage.



and hemiplegia improved in one case⁽⁵⁾. In none of the surgical reports had there been a cure for the hemiparesis or for atrophy of limbs.

Pathology and Pathogenesis

These lesions are characterised by a skull defect, dural defect, cerebral scar consisting of gliotic brain, with or without single or multiple cysts. Concomitant ipsilateral cerebral atrophy and ventricular enlargement are common.

The most likely cause of the absence of bone union is the interposition of pericranium, torn dura between the fracture during infancy when the skull is relatively soft and rapidly growing. Skull erosion, involving more of the inner than the outer table, is probably due to the transmission of brain pulsations either through a leptomeningeal cyst or through a pial tear against the fracture edges.

In an experimental controlled study, interposed dura or pericranium prevented fracture union. Erosion and enlarge-

ment of the fracture required arachnoid tear as well. The presence of cerebral injury and ventricular communication were not found to contribute to enlargement of this lesion⁽⁸⁾.

Diagnosis

A post-traumatic soft, enlarging and pulsatile scalp lump is the commonest presentation. Epilepsy, hemiparesis or atrophy of limbs are manifestations of the initial cerebral damage.

Radiologically, a skull defect, with scalloped edges is seen. CT scan shows a fluid-filled cyst or brain herniating out of this defect. The ipsilateral hemisphere most often shows signs of the initial trauma - atrophy and ventricular dilatation. Air pneumo-encephalograms, arteriograms were done in the pre-CT era to image cerebral tissue within or herniating out through the defect.

Whether or not the leptomeningeal cyst causes epilepsy or imparts mass effect is open to debate^(1,6). However, viable cerebral tissue, encephalomalacia and cerebral cyst herniating through the fracture may well perpetuate or even worsen neurological deficits⁽³⁾.

Treatment

Small skull defects with no neurological deficits or uncontrolled fits can be safely observed. We feel that in cases of doubt, decision to offer surgery should be undertaken based on the CT findings and most importantly after a period of

follow-up. Where the skull defect is enlarging progressively or when there is fear of injury to the brain due to the deficient skull, then cranioplasty is indicated.

The important point in surgery is not to open into the scar if CT shows cerebral tissue within the scar. The dural edge, should in most cases be traced under the bone if it has not retracted too far away and a watertight closure with a dural substitute (autogenous if possible) performed. The skull defect is then repaired, most commonly with acrylic. If a large cyst with mass effect exists this should be decompressed, either by surgical communication with the underlying ventricle, or by a shunting procedure.

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