

## **SKULL FRACTURES OF CHILDHOOD**

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## Growing Skull Fractures Of Childhood

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### SUMMARY :

In this paper, a rare complication of head injury in children GROWING SKULL FRACTURES are reviewed. A tear in the dura at a fracture site results in the formation of an arachnoid cyst. The cyst erodes the overlying bone at the fracture margins to produce a defect through which the cyst and its contents may herniate. Most cases occur in children, usually under 3 years of age and present with hemiplegia, epilepsy, or a fluctuant scalp swelling. Radiology, especially CAT scanning is necessary to establish the diagnosis. Treatment initially involves shunt insertion to relieve raised intracranial pressure to stop the fracture growing. If the case came to the Neurosurgeon with a huge defect, then treatment would also involve excision of the cyst and its contents, followed by cranioplasty.

**Key Words :** Skull fracture, growing fracture, leptomeningeal cyst.

### INTRODUCTION :

The persistence or growth of a skull fracture has been described under a variety of terms including leptomeningeal cyst, growing skull fracture, cephalhyd-rocele, posttraumatic porencephally, and other terms<sup>9</sup>.

Enlargement or growth of a skull fracture is a sequella of trauma seen primarily in children, usually under 3 years of age (approximately 90%) and present with neurological deficit, that is, hemiplegia, epilepsy or a fluctuant scalp swelling<sup>3, 4, 5, 7</sup>. Neurological deficit may be avoided by early diagnosis, surgical intervention, dural closure and cranioplasty<sup>3</sup>.

Being first described by HOWSHIP in 1816, this condition in the English literature was first used by LENDE and ERICKSON<sup>3, 4, 8</sup>. Growing skull fractures usually involve the parietal bone but can affect the frontal or occipital bones and sutures<sup>4, 8</sup>. The coincidence of the age of injury with the period of most active growth of the skull is obvious.<sup>4</sup> However, in recent literature, It is reported that Growth has been detected as early as 2 weeks and as late as 20 years after fracture<sup>8</sup>. A fracture line that is destined to grow may be accompanied

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at the time of the initial injury by a cystic subgaleal swelling that tenses when the child cries. This swelling (a "cephalohydrocele") consists of either cerebrospinal fluid (CSF) or a mixture of CSF and blood resulting from an underlying dural-arachnoidal tear<sup>3</sup>. The scalp overlying the fracture is almost never broken at the time of injury<sup>4</sup>. Transillumination may or may not be present, depending upon the blood content of the mixture, but when present it is diagnostic of a dural-arachnoidal tear<sup>5</sup>. Beneath the lesions of skull and dura mater there is a local brain injury which is a constant feature of this syndrome, obviating itself clinically with hemiparesis, hemiatrophy and focal seizures<sup>6</sup>.

Early roentgenograms of the skull show widening of the fracture line, and subsequent roentgenograms also demonstrate scalloped margins and erosion of the inner table. Pneumoencephalography often shows dilatation of the lateral ventricle with or without porencephaly, midline shift to the same side and absence of subarachnoid air in the region of the cyst<sup>8</sup>. On the other hand, To see the extension of cerebral vessels through the bony defect, cerebral angiography should be carried out; suggested by STEIN and TENNER (in reference 8). LYKE and associates studied the CAT scanning role in evaluating this pathology and they reported that CAT scanning can demonstrate both CSF and soft tissue within the cystic swelling. The ventricular detail obtained from the CAT scanning enables the hydrocephalus or a porencephalic cyst to be excluded without resorting to pneumoencephalography. Large vascular structures such as venous sinuses can be enhanced by intravenous administration of contrast medium thus making cerebral angiography no longer necessary<sup>9</sup>.

### **PATIIOGENESIS AND DISCUSSION :**

The incidence of growing fractures of the skull varies with different series from less than % 1 to % 16, but a review of the literature suggests it is rare. Although it was described as early as 1816 by HOWSHIP, insight into the etiology of this lesion awaited the reviews of TAVERAS and RANSOHOFF, and LENDE and ERICKSON<sup>4, 7, 9, 10</sup>. The dura in children is very adherent to the bone and skull fractures tend to produce extensive dural tears which are larger than the bone defects<sup>5</sup>. The authors concluded that the dural tear is the single most important factor in the pathogenesis of these lesions and that without it, the fracture would heal spontaneously as expected<sup>2, 4, 5, 10</sup>. Experimentally it was shown that additional pial, brain or ventricular damage did not increase the incidence of growing fracture. Therefore, these lesions appear to be of secondary importance in the production of growing fracture<sup>2</sup>.

The dural defect allows the arachnoid membrane to project out into the fracture site, resulting in the formation of isolated arachnoidal cysts. STEIN and TENNER have also demonstrated the presence of herniated brain, in the fracture site and porencephaly within the adjacent brain. The latter is thought to be due either to compressed cortical vessels within the fracture site leading to cerebral infarction or associated cerebral contusion or hematoma at the time of the fracture<sup>7, 8</sup>. On the other hand, it is well known that the age of the patient and the width of the fracture facilitate the development of a growing fracture<sup>5</sup>. Leptomeningeal cysts (arachnoidal cysts) rarely occur in patients more than 3 years of age and it is reported that all diastatic skull fractures with an initial width of more than 4 mm. should be explored surgically, if there is also contralateral neurological deficit, to prevent growing skull fracture developing<sup>3, 8</sup>.

RAMAMURTHI and KALYANARAMAN advocate conservative management of the cases which come to the Neurosurgery very late<sup>9</sup>. Most authors agree that early surgery would be beneficial if carried out soon after injury<sup>1, 8, 10</sup>. Part of the reason why the fracture grows is because there is raised intracranial pressure and a shunt, therefore relieves it and stops the fracture growing. Shunt insertion will not, however, reduce the size of any fracture<sup>1</sup>. If we, Neurosurgeons, can get the fracture early, a shunt (ventriculo-peritoneal, ventriculo-atrial) insertion would be the first line form of management<sup>1</sup>. If the patient late or growing fracture is very large, surgical intervention would include fairly extensive removal of bone and tissue in order to get a clear dural plane. Closure of the dura is then essential with a patch and the hole can be filled with acrylic. After surgery, the patient might have raised intracranial pressure symptoms for which a shunt insertion should be considered as well.

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