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CASE REPORT

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Growing skull fracture – Case report & technical aspects of repair

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ABSTRACT

Keywords: Growing skull fracture, Childhood skull fracture, Craniocerebral erosion, leptomeningeal cyst, Infant, Case series

Abbreviations: GFS-Growing fracture of skull, CSF-Cerebrospinal fluid, DFS-Diastatic fracture of skull, MRI-Magnetic resonance imaging, SPECT-Single photon emission computed tomography, CT Scan-Computed Tomography Scanning

1. INTRODUCTION

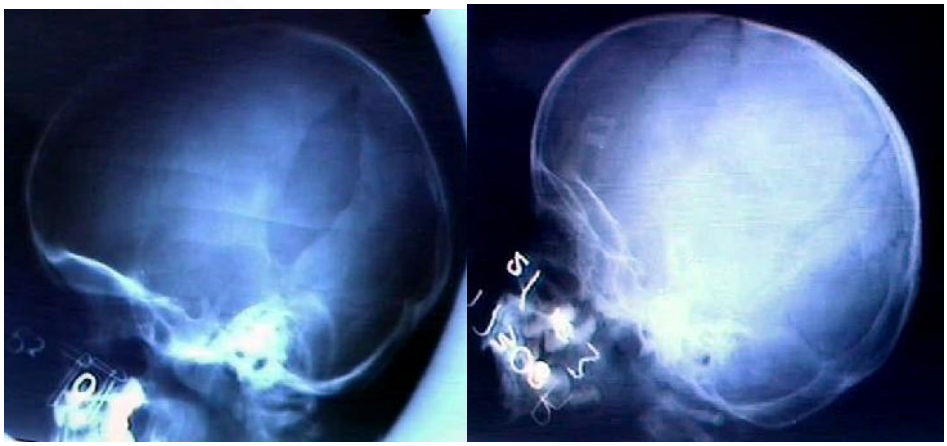
Growing fracture of the skull (GFS) is a rare complication of head injury in infancy and early childhood which is characterized by skull fracture that enlarges with time.Growing skull fracture is recently termed as CraniocerebralErosion. They are estimated to occur in 1% of linear skull fractures sustained under 3 years of age—the most vulnerable age group (Vignes, 2007). They can present many years later with headache, seizures, and hemiparesis. It is characterized by progressive diastatic enlargement of the fracture line. This late complication is also known as a Leptomeningeal cyst because of its frequent association with a cystic mass filled with CSF (Khandelwal, 2002).The exact etiopathological process of growing skull fracture is unclear. The single most important factor in the pathogenesis of growing skull fracture is dural tear (Taveras, 1953). In 1961, Lende and Erickson reviewed the literature on this subject and emphasised on four essential features: (1) skull fracture in infancy or early childhood; (2) dural tear at the time of fracture; (3) brain injury underlying the fracture; and (4) subsequent enlargement of the fracture resulting in a cranial defect (Dyke CG, 1938).

Growing Skull fracture:
A growing skull fracture (GSF) also known as a craniocerebral erosion or leptomeningeal cyst due to the usual development of a cystic mass filled with cerebrospinal fluid is a rare complication of head injury usually associated with linear skull fractures of the parietal bone in children under 3.

Diastatic skull fracture:
Diastatic fractures occur when the fracture line transverses one or more sutures of the skull causing a widening of the suture.

2. CASE REPORT

3 year old male patient presented with gradually progressive right parietal swelling and generalized seizures of 8 months duration. There was history of fall from height 8 months back following which he developed progressive, soft to firm, swelling of right parietal region and episodic generalized seizures. The patient was initially managed with antiepilepticsfor seizures and further underwent radiological intervention. Following investigation were used, Plain X-ray skull (photograph 1), Computerized tomography (CT scan), M.R.I. studies, clearly demonstrated the extent of bony defect and its configuration (photograph 2). He underwent MRI study which demonstrated the extent of brain and arachnoid damage (photograph 3).



Photograph 1 x-ray skull showing fracture site

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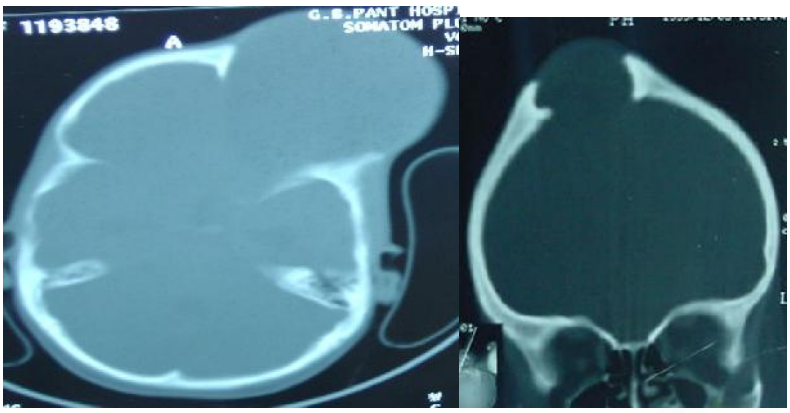
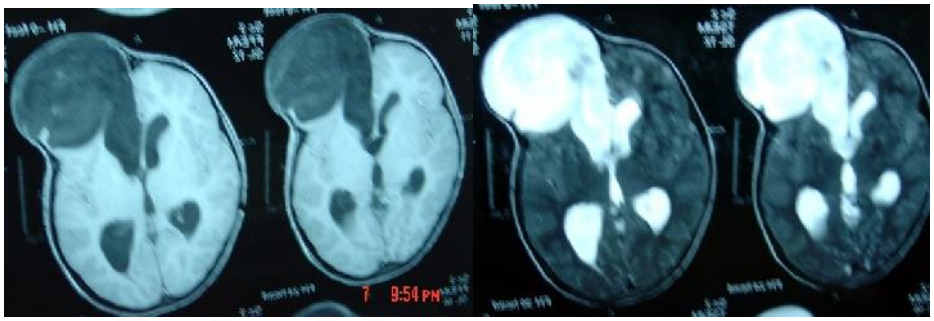


Photograph 2 CT Scan



Photograph 3 MRI

3. SURGICAL INTERVENTION

Patient was taken up for exploration and repair under general anaesthesia. During exploration, the scalp was stripped off the swelling. The bone was drilled all around the swelling to identify the dural edge. The duramater was separated from underlying brain. Necrotic brain tissue was excised. Duramatermargins were exposed and were repaired using fascia lata graft or artificial duramater. Skull defect was closed by cranioplasty using split thickness bone graft.

Cranioplasty:
Surgical repair of a defect or deformity of a skull.

4. RESULTS

Post-operative course of the patient was uneventful. Scalp wound was healthy and sutures were removed on 11th postoperative day. The patient was kept on oral antiepileptic for 1 year postoperatively. He was weaned of antiepileptic thereafter and did not report any new episode of seizure or any neurological deficit during his follow up.

5. DISCUSSION

Growing skull fracture, also known as "posttraumatic leptomeningeal cyst" or "craniocerebral erosion," was first described by Howship in 1816. It is a rare complication following head injury and its incidence is less than 0.05% to 1.6% of cases (Howship J,

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1916). It is common below the age of one year (50%), and nearly 90% of the patients are below the age of 3 years, after which the condition is rare (Iplikcioğlu AC, 1990). Common etiologies include fall, vehicular accident, and child abuse; cases following difficult vacuum extraction and corrective surgery for craniosynostosis have also been described. Growing skull fracture commonly involves the calvarial bones. Rarely, it can occur at basiocciput and orbital roof (Parmar RC, 2000).

Most important factor in GSF pathogenesis is skull fracture, with its dural tear and entrapment of arachnoid membrane and brain tissue within the fracture margin.There are 2 main hypotheses to elucidate why the incidence of GSF is higher in infancy and early childhood than in adulthood. One hypothesis is that during the first 2 years of life, rapid growth of the brain and skull occurs; the duramater adheres more tightly to the bone and thus is more easily torn when the skull is fractured (Gugliantini P, 1980). The otherhypothesis proposes that the skull is thinner, less stiff, and more deformable, and in deforming can more readily tear the duramater.

In the early stage of GSF, the main damages to the brain and bone are caused by the injury itself. However, the damages as well as the neurological deficits will increase during the progression of GSF, especially in the late stage. The neurological deficits cause the main disruption in the quality of life for most patients with GSF. Xiu et al divided the progression of GSF into 3 stages during treatment of the disease. Stage 1 is the prephase of GSF. The time frame for this stage is from the time of injury to the time just before enlargement of the fracture. Patient with GSF at this stage must have the following conditions: 1) skull fracture with dural tear, and 2) herniation of brain tissue or arachnoid membrane through the fracture. Traditionally, this stage would not be included in the diagnosis of GSF because there is no enlargement of the fracture. However, Xiu et al argue that the patients who have suffered skull fractures and meet these 2 conditions have the highest risk of GSF, especially when the patient is younger than 3 years of age.

Stage 2 is the early phase of GSF. The time frame extends from the initial fracture enlargement to 2 months after the beginning of enlargement. Based on our observations and according to other studies, gross enlargement of the fracture occurs about 2 months following the initial fracture enlargement. Therefore, in this stage, the bone defect is small, and the deformity of the skull and the neurological deficit are mild. Growing skull fracture diagnosed and treated during this stage will have a better prognosis. Stage 3 is the late phase of GSF. It begins at 2 months after initial enlargement. During this stage, the bone defect becomes larger, and skull deformity and neurological disorder become severe if left untreated (Liu XS, 2012).

Every infant/child who has sustained the trauma should undergo a plain X-ray to rule out any fracture. If a fracture is found, CT scan should be done to rule out injury to the brain. Based on the CT appearance, growing skull fractures are subdivided into three types: Type I refers to growing skull fracture with a leptomeningeal cyst, which may be seen herniating through the skull defect into the subgaleal space. Associated brain damage or gliosis is seen in type II, while type III is associated with porencephalic cyst. (Naim-Ur-Rahman, 1994)

The surgical techniques for treating GSF are well described. The procedure includes resection of the leptomeningeal cyst and degenerated brain tissue, repair of the dural defect, and cranioplasty. If hydrocephalus or seizures have occurred, VP shunt placement or resection of the scar tissue inducing the seizures, respectively, should be performed. The most important step of surgery is water-tight closure of the dural defect (Ramamurthi B, 1970)

During surgery, the surgeon should avoid excising any functional brain tissue exposed under the skull defect, especially during the early stages of GSF. If a patienthas suffered seizures before the operation, one should perform electrophysiological monitoring during resection of the lesions inducing the seizures. Ventriculoperitoneal Shunt placement may be necessary for patients who develop hydrocephalus due to GSF.

6. CONCLUSION

Diastatic Skull fracture with pseudomeningocle is a complex disorder involving chronic white matter loss, disturbances of cerebrospinal fluid and chronic ischemic changes. Surgical repair of duramater and bony defect prevents further neuronal damage. The natural course of an untreated case is progressive in nature.There is progressive cranial and cerebral damage and thus there is a need for early surgical intervention. However the ischemic effects of initial trauma continue to persist for long time requiring long-term surveillance.

DISCLOSURE

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