



Case Report

Post-Traumatic Leptomeningeal Cyst in Child: A Case Report and Review of Literature

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Abstract

Leptomeningeal cyst or growing skull fracture is a rare complication of cranial fractures that is usually seen in infants and early childhood following trauma. It presents as cystic, nontender swelling over scalp with underlying bony defect due to old history of trauma. We hereby report one such case along with histopathology. Its early diagnosis is essential for clinicians for a targeted approach so as to avoid occurrence of complications.

Keywords: Leptomeningeal cyst, growing skull fracture.

Introduction

Post-traumatic leptomeningeal cyst occurs as a rare complication of cranial fractures in children with incidence 0.05%–0.6% (Saito et al 2009). It usually presents as enlarging cranial fracture though it can also present as headache, seizures, and hemi paresis many years later (Kutlay et al, 1998). The most vulnerable age group is less than 3 years. We hereby report one such case where a 3-year-old child presented with growing skull mass after he suffered from trauma.

Case Report

We represent a case of a 3-year-old male child presented with a nontender growing scalp swelling in the left frontal supraorbital

region. He had a history of fall from first floor 1 year back. There was no neurological deficit. Local examination showed presence of bony defect in the left frontal supraorbital region. There was a linear fracture in left frontal region extending into left orbital roof in initial noncontrast computed tomography (NCCT) head, with an evidence of diastasis of fracture line over time in comparison with current NCCT head (Figures 1(a) and 1(b)).

Intraoperative examination found bony defect with interposed gliotic brain matter with pia-arachnoid covering densely adherent to the defect (Figure 2(a)). The pia-arachnoid layer was meticulously separated from the scalloped fracture edges all around forming the defect (Figure 2(b)). Underlying

gliotic brain showed presence of leptomenigeal cyst. The adherent dura and scanty glial tissue was excised and sent for biopsy. The histopathology showed peripheral thickened collagenous fibers encircling and extending into the glial tissue.

The glial tissue within the fibrous tissue appeared edematous (Figure 2(c)). Special stain with Masson's Trichrome was done which showed enhancement of fibrous tissue (Figure 2(d)).

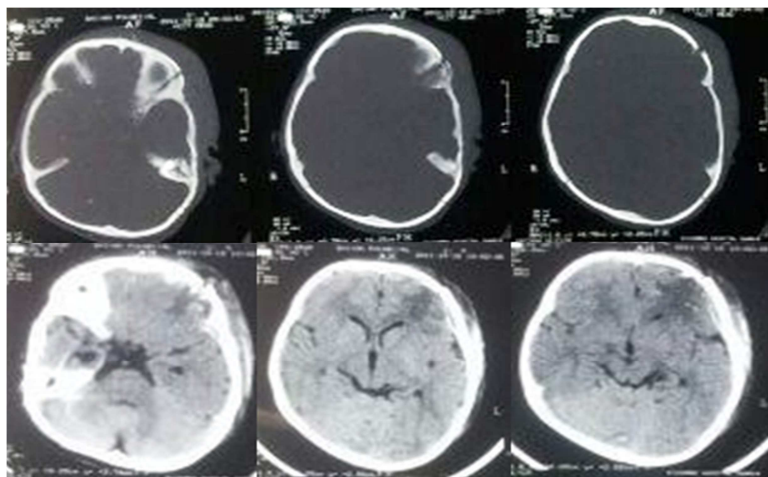


Figure 1(a): Initial Non-Contrast Computed Tomography (NCCT) Head at Time of Injury Showing Linear Fracture in Left Frontal Orbital Region Extending into Left Orbital Roof with Underlying Resolving Contusions in Left Frontal Lobe of Brain

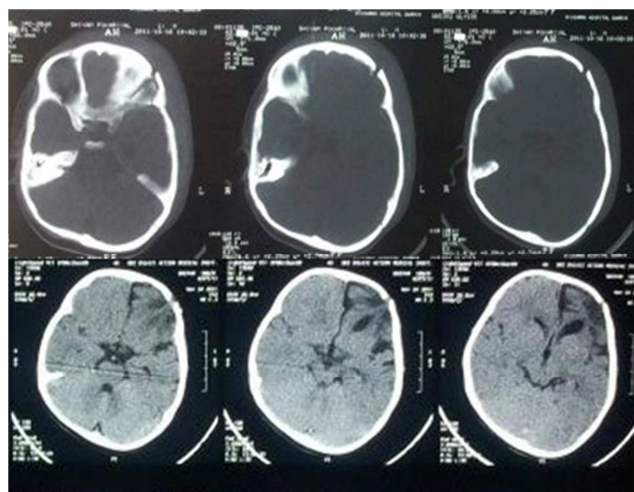


Figure 1(b): Noncontrast Computed Tomography (NCCT) Head 1 Year Following Injury Showing Diastasis of Fracture Line and Underlying Gliotic Brain with Cystic Changes with Dilatation of Ipsilateral Frontal Horn of Lateral Ventricle with Porencephalic Cyst

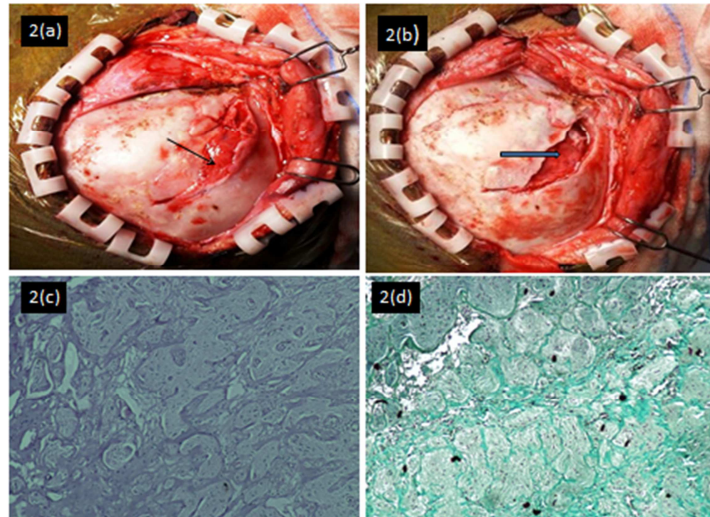


Figure 2: Figure 2(a): Bone Defect with Interposed Brain Matter with Pia-Arachnoid Covering Densely Adherent to Bony Defect (Thin Arrow). Figure 2(b): Scalloped Margins of Bony Defect Seen after Separating the Pia-Arachnoid Covering (Thick Arrow). Figure 2(c): Haematoxylin & Eosin Stained (10x) Tissue Showing Thickened Collagenous Fibers Encircling and Extending into the Glial Tissue. Figure 2(d): Masson's Trichrome Stained Tissue (10x) Showing Enhancement of Fibrous Tissue (Green).

Since clinically and histopathologically the herniated tissue was the cerebrum in our case, it was classified as Type II growing skull fracture (Naim Ur Rahman et al, 1994).

Morphologically, the predominant factor responsible for fracture growth may lie in the subarachnoid space (a leptomeningeal cyst), the cerebrum (herniated brain), or the ventricle (dilated underlying ventricle with porencephalic cyst). These events constitute the morphological basis for the fracture types I, II, and III, respectively (Naim Ur Rahman et al, 1994).

Discussion

Growing skull fracture has been described in the literature as an entity synonymous to leptomeningeal cyst due to collection of CSF underneath. The other names of it include enlarging skull fracture, expanding skull fracture, post-traumatic bone absorption, post-traumatic porencephaly, traumatic

ventricular cyst, craniocerebral erosion, and cephalohydrocele (Suri A, Mahapatra AK, 2002). It was first recognized and reported by John Howship in 1876 (Gupta S K 1997). They mostly arise in cranial convexity, but cases occurring in orbital roof (Mohindra S 2006) and posterior fossa (Gupta S K 1997) have also been reported.

Although the majority of patients present with complaints 2 to 12 months after the traumatic event, delay in presentation for 8 to 20 years has also been reported. Suri et al have reported that orbital floor fractures occur primarily in older children with mean age 12.0 ± 4.2 years due to increased vulnerability of the face due to growth and pneumatization of the maxillary sinus (Suri A, Mohapatra AK, 2002). Meier et al. have reported leptomeningeal cyst of orbital roof in a 47-year-old female who had a remote history of skull fracture at the age of 3 years (Meier JD, Dublin AB, Strong EB, 2009). Halliday AL, Chapman PH, Heros RC, (1990)

and Naim-Ur-Rahman (1994) reported a case of a growing skull fracture following injury in adult life. The most common causes of head injury in children are as follows: falls, child abuse, motor vehicle accidents, sport accidents, assaults, and instrumental delivery (Ciurea AV, 2011). Predisposing factors include linear skull fracture, disruption of the underlying duramater, and raised intracranial pressure due to cerebral oedema, contusion, or in occasions even a normally growing brain (Mohindra S, 2006). Malleability of the infant skull and rapid growth of brain and skull leading to tighter adherence of dura to bone in children may account for the common occurrence of growing fractures in children (Rahimizadeh A, 1986). This complication is caused by a tear in the duramater, through which pulsation of the cerebrospinal fluid (CSF) forces the arachnoid layer with or without brain parenchyma to herniate. This causes bone malnutrition, destroying the bone edge, and finally enlarges the fracture line. Associated lesions like hydrocephalus, encephalomalacia, cerebral infarct, leptomeningeal cyst, brain tissue herniation, subdural fluid collection, and ipsilateral ventricular dilatation were previously reported preoperatively (Ali Akhaddar A, 2011). Post-traumatic aneurysms and subdural hematomas have also been reported to accompany growing skull fractures (Buckingham MJ, 1989 and Locatekku D, 1989) although damage to underlying brain is not a prerequisite for the development of growing skull fractures (Iyer S G, 2003). They can also mimic a skull tumour (Kurosu A, 2004) especially if seen in adults presenting as bone cyst, where histopathology is essential to rule out the tumour.

Prompt diagnosis is essential as early corrective surgery should be performed for growing skull fractures so as to prevent secondary brain damage and neurological deficits which especially occur in late stages and lead to progressive disruption in the quality of life of patients (Liu X, 2012). A clinical, radiological, and histopathological correlation helps in diagnosing types of growing skull fracture and thereby to assess

its prognosis. Surgical treatment involving excision of meningocele and repair of bone and dural defects is associated with good outcome. Dural defect needs to be fully repaired. Although, in infants and children, linear skull fractures should be monitored until definite skull bone consolidation so as to prevent progression, they should not be left neglected.

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