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Growing Skull Fracture Simulating a Rounded Lytic Calvarial Lesion

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Growing skull fracture is a rare complication of head injury in which 90% of the cases occur among children less than 3 years old. Only two cases have been reported in the adult population [1, 2]. We report a rounded lytic skull defect in a 75-year-old man that was surgically and pathologically determined to be a growing skull fracture despite a deceptive radiologic appearance simulating a lytic calvarial lesion. The typical features of a growing skull fracture are discussed.

Case Report

A 75-year-old man presented with acute onset of mild right hemiplegia and dysarthria, which lasted several days and resolved completely. A reversible ischemic neurologic deficit was suspected. The patient had no history of head injury; however, he stated that a painless "hard bump" had been palpable on his skull for many years—he could not recall precisely when it first appeared.

An initial CT scan revealed no evidence of infarction; however, a well-defined 3.0-cm lytic lesion was identified in the left parietal calvarium (Figs. 1A and 1B). The underlying cerebral cortex bulged slightly into the defect, although this was only appreciated in retrospect. No associated fracture line was seen on CT or on skull radiographs (Fig. 1C). Only a faint rim of increased uptake was noted at the margins of the lesion on a radionuclide bone scan (not shown). A skeletal survey demonstrated no other abnormalities.

A localized left parietal craniectomy and duraplasty were performed. Upon turning the scalp flap, the surgeon noted a well-defined punched-out calvarial lesion. The underlying cerebral cortex bulged out through an associated dural defect. The abnormal cortical tissue and the calvarial lesion were excised and the dura was closed.

Pathologic examination of the bone revealed a circular, well-defined lytic lesion (Fig. 1D). Microscopically, there was evidence of remodeling at the bony margins surrounding the defect. Excised brain was gliotic with no evidence of tumor. The pathologic findings indicated an acquired (probably posttraumatic) encephalocele; that is, a growing skull fracture.

Discussion

The diagnostic possibilities usually considered in the face of a solitary lytic rounded calvarial defect in an older adult include metastasis, multiple myeloma, epidermoid cyst, and surgical defect [3, 4]. Because malignancy was suspected in this case, it was managed surgically. During the operation, a growing skull fracture was identified and repaired appropriately.

Growing skull fracture was first described by Howship in 1816 [5]. Since that time, numerous terms have been used to describe this condition, including leptomeningeal cyst [6], craniocerebral erosion [7], and various others [8-12]. Although leptomeningeal cyst is a commonly used term, no sound pathologic proof exists for cyst formation in the great majority of cases. Tandon et al. [13] found no evidence of true leptomeningeal cyst formation within the herniated arachnoid in their review of 60 cases. They demonstrated that the apparent leptomeningeal cysts were in fact either "cystic encephalomalacia surrounded by gliotic brain" or fluid collections within abnormal leptomeninges. Tandon et al. therefore recommended that this term be discarded. Since there is progressive enlargement of the skull fracture, generally with involvement of the underlying brain in the defect with resultant neurologic symptoms, Penfield and Jasper [7] suggested that the term craniocerebral erosion be used. However, the most commonly accepted term in current literature is still "growing skull fracture."

The lesion is thought to arise from a skull fracture with an associated dural tear, almost always occurring during childhood. As a result, repeated pulsations of brain and CSF are thought to cause erosion of adjacent calvarial margins [14]. Although the exact reason for the nearly exclusive occurrence in the young pediatric age group is not known, active skull growth as well as firm attachment of dura to the skull are probably responsible.

The defect is typically irregular in contour and elliptical in shape. According to Taveras and Ransohoff [14], these lesions generally exhibit scalloped margins and some degree of sclerosis of the adjacent bone; both inner and outer tables are involved, the inner table to a greater extent. In some instances, the old fracture line may be clearly seen at either end of the elliptical defect. We have found no previous case reports describing a circular lesion in this condition.

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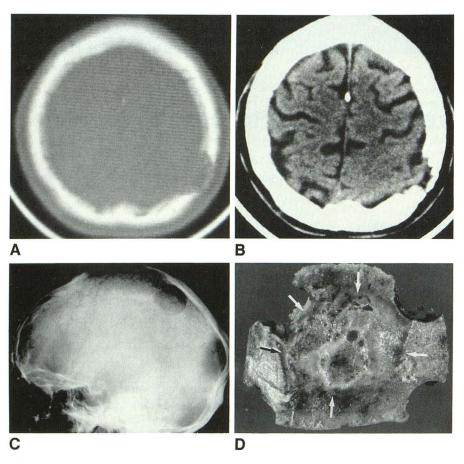


Fig. 1.—75-year-old man with growing skull fracture.

A and B, Cranial CT scans, bone window setting (A) and soft-tissue window setting (B) show

A and B, Cranial CT scans, bone window setting (A) and soft-tissue window setting (B), show rounded calvarial defect in left parietal region with scalloped margins. Underlying brain bulges into defect.

- C, Skull radiograph, lateral view, also shows rounded calvarial defect.
- D, Pathologic craniectomy specimen shows circular, lytic skull lesion (arrows). Gliotic brain had herniated into the defect (see text).

CT scanning usually clearly delineates the calvarial defect. Herniation of the underlying brain and meninges into the defect is frequently seen and there may be enhancement of the pia mater or highly vascularized "pseudodura" [15].

Clinically, the small growing fracture may present as a lump over the skull. As the lesion increases in size it may progress to form a large pseudomeningocele [13]. This is often associated with development of focal neurologic signs and symptoms such as seizure or neurologic deficit. These signs are thought to be the result of direct compression of the underlying cerebral cortex by the "cyst." It is for this reason that skull fractures in the pediatric population should be followed closely. If a growing skull fracture develops, surgery, with cranioplasty or duroplasty, is the generally accepted form of management [13]. However, Ramamurthi and Kalyanaraman [16] reported four patients who did well without surgery, and they argue against operating, since this condition is not always progressive. Our patient, asymptomatic at the age of 75, supports this contention.

In summary, our report of a growing skull fracture in an adult is unusual in that instead of the usual elliptical or oval lesion, the skull defect was perfectly round, simulating a lytic calvarial lesion; moreover, the patient remained asymptomatic for many years without surgical intervention.

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