Emerging Symptoms of Growing Skull Fracture after Secondary Trauma in an Adult
—Case Report—

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Abstract

A 20-year-old female presented with dysphasia and slight hemiparesis following a head trauma, who had a non-treated growing skull fracture which had remained asymptomatic for about 18 years, despite an encephalocele in the left parietal region. Neuroimaging demonstrated secondary brain damage and herniated brain resulting in gliosis. Electroencephalography revealed epileptic discharge in the affected region. Dural repair and cranioplasty resolved her neurological deficits. Early corrective surgery should be performed for growing skull fracture to prevent secondary brain damage.

Key words: growing skull fracture, head trauma, adult

Introduction

Growing skull fracture is a rare complication of head trauma with an incidence of only 0.05% to 0.1%, but is a significant clinical entity in infancy because progressive enlargement of the fracture may form a permanent cranial deficit and cause secondary brain damage if not treated. The natural course is uncertain, since early corrective surgery is usually performed.

We describe a 20-year-old female with an untreated growing skull fracture that remained asymptomatic despite encephalocele for more than 18 years until a second trauma caused emergence of neurological deficits.

Case Report

A 20-year-old female was involved in a motor vehicle accident and struck her left parietal region on the ground on June 12, 1990. She was transferred to our hospital.

On admission, her Glasgow Coma Scale score was 14. She was dysphasic with slight right hemiparesis. Plain skull x-ray films demonstrated a skull defect in the left parietal parasagittal region and linear fractures extending from the defect to the ipsilateral temporal region and the contralateral parietal region. There was slight separation of the sagittal suture located just medial to the bone defect (Fig. 1). Computed tomography (CT) showed a thin epidural hematoma in the left temporal region and a small contusional hematoma in the left parietal region, which were unchanged in size on a second CT scan. A coronal CT scan and magnetic resonance (MR) imaging clearly demonstrated encephalocele in the left parietal parasagittal area (Fig. 2). Electroencephalography showed epileptic discharges in the left parietal region.

She had suffered head trauma when she was 2 years old and had since had a bulge in her scalp at the left parietal region. No details taken at the time of injury were available. She had been admitted to a hospital for 2 months and treated conservatively. The bulge had gradually increased in size until she was 14 years old.

Surgery was performed on July 3, 1990. At the skull defect, the dura was absent and granular tis-
Sue was tightly adhered between the periosteum and the brain. The cortical surface at the encephalocele was yellowish and atrophic suggesting gliosis and organic damage of the herniated brain (Fig. 3). The arachnoid membrane was thickened but no cerebrospinal fluid (CSF) retention was observed. The bone was rongeured to expose the dural edge and both dural plasty and cranioplasty were performed. The dural defect extended to near the superior sagittal sinus.

The postoperative course was excellent. Follow-up MR imaging demonstrated absence of encephalocele. No CSF retention was observed on follow-up CT scans for 2 years after the operation. Both lateral ventricles were unchanged in size. She had no neurological deficits except for slight dysphasia.

Discussion

Growing skull fractures indicate underlying lesions which are important etiological factors in the clinical presentation and development, and may manifest as seizure (40.9%), focal neurological deficit (43.2%), or loss of consciousness (37.9%). Most patients demonstrate a localized bulge of the scalp.

The etiopathogenesis of growing skull fracture originates with trauma which produces a skull fracture and an underlying dural tear. Leptomeningeal cyst or damaged swollen brain then herniates into the fracture line, transmitting the intracranial pressure pulsation to the site. This causes bone malnutrition, destroys the bone edge, and finally enlarges the fracture line. The dural defect is also enlarged in most cases. A few cases without the dural tear have been reported, but these are controversial. The expanding dural defect is caused by extension of the edge of the lacerated dura in the direction parallel to the edge of the defect, and there is probably some sliding or migration of the dura.

Nine adult cases of growing skull defect have been reported including ours. Seven patients had suffered from trauma in childhood, one at 19 years old, and another not reported. The symptoms due to the non-treated lesion, except for the scalp bulge, first appeared more than 10 years after trauma in six cases. One patient had been asymptomatic for more than 60 years. Diagnosis in older adults must exclude metastasis, multiple myelomas, and epidermoid cyst. Our case of neurological deficits caused by second trauma is
the only one reported.

CT and MR imaging clearly demonstrated the herniated brain caused by the skull defect. CT scan evaluate the extent and content of the brain and cyst, while MR imaging defines the lesion in multiple planes and is the best modality for evaluating the relationship between the brain and cyst.

Ramamurthi and Kalyanaraman\textsuperscript{11} suggested that surgery is unnecessary if there is no bulge and no large cranial deficit because fits can be controlled medically and neurological deficits cannot be improved by dural repair and cranioplasty. Taveras and Ransohoff\textsuperscript{10} advocated early operation in order to minimize the underlying brain damage and reduce the severity of post-traumatic epileptic attacks. Arseni and Ciurea\textsuperscript{13} described the therapeutic results in a series of 21 cases, and found that delayed surgery cannot obtain the same positive effects, especially for the mental development of the child. There are no established operative indications, but we think surgery is better when diastatic fracture and overlying scalp bulge are present.

References


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