Varieties of growing skull fractures in childhood

FRANK P. GOLDSTEIN, M.D., SHELDON A. E. ROSENTHAL, M.D., JOHN C. GARANCIS, M.D., SANFORD J. LARSON, M.D., AND CHARLES E. BRACKETT, JR., M.D.

Departments of Neurosurgery and Pathology, Marquette School of Medicine, Milwaukee, Wisconsin, and Department of Surgery (Section of Neurosurgery), Kansas University Medical Center, Kansas Center, Kansas

Clinical examples of three varieties of leptomeningeal cysts secondary to a traumatic dural tear have been discussed. The cysts developed beneath fracture lines and led to erosion of bone and herniation or depression of brain.

Growing fractures of childhood have been described in humans and simulated in an experimental model with puppies. A thorough review of the world’s literature and discussion of clinical features of the syndrome were presented in 1961 by Lende and Erickson. Our three case reports illustrate three varieties of growing fractures that may be encountered clinically.

Case Reports

Case 1

A 1-year-old child sustained a parietal fracture and 1 year later developed a pulsating cranial defect in the left parietooccipital region. X-ray films of the skull revealed a scalloped 7 × 2 cm defect in the left parietal occipital region (Fig. 1 upper). At operation it was noted that the pericranium covered multiple cystic structures; the dural edge had been recessed behind the bone edge of the entire bone defect and was widely separated from the pericranium. Underlying brain tissue was depressed by the cysts (Fig. 1 lower). A rib cranioplasty and dural closure were performed. The postoperative course was unremarkable.

Case 2

A 5-month-old child sustained a frontal fracture and was unconscious for 30 minutes. A large left fronto-temporal-parietal cephalo-hematoma was noted. Five months later, a pulsating defect was noted in the left frontal region. The physical and neurological examinations were normal. Skull films revealed a 3 × 4 cm defect in the left frontal region (Fig. 2 upper). At operation it was noted that there was direct continuity of pericranium and dural edge with the opposing dural edge recessed beneath the bone defect. Brain tissue was depressed by the cysts (Fig. 2 lower). Dural closure and cranioplasty (pericranial graft) were performed. The postoperative course was unremarkable.

Case 3

A 3-month-old child sustained a parietal fracture, but did not lose consciousness. Two months later, examination revealed a pulsating left parietal cranial defect measuring 3.5 × 2.0 cm. The neurological examination was normal. Skull films revealed a defect measuring 3.5 × 2.0 cm (Fig. 3 upper). A ventriculogram demonstrated dilation of the left lateral ventricle beneath the cranial defect; air did not communicate with any cranial cysts.
Goldstein, Rosenthal, Garancis, Larson and Brackett

noted that there was direct continuity of the pericranium and dural edge with the opposing dural edge so far beneath the bone defect that the bone edge on this side had to be rongeured away 1 cm to find the dura. Brain surrounded by cysts herniated into the pericranium (Fig. 3 lower). Specimens were taken of the scarred tissue, pericranium, dura, bone, and brain tissue beneath the cystic mass. Dural closure and cranioplasty (pericranial graft) were performed. The postoperative course was unremarkable.

Discussion
The clinical cases described have the common features associated with the syndrome of growing skull fractures of childhood: 1) a parietal skull fracture in infancy or childhood; 2) unsuspected dural and arachnoid tear at the time of the fracture; and 3) subsequent enlargement of the fracture to form a cranial defect.\textsuperscript{1-3,6,7,10} Other associated features included ventricular enlargement beneath the fracture and herniation of underlying brain.

Pathogenesis
The pathogenic features to be considered are as follows. In 1941, Penfield and Erick-
Growing skull fractures

Sonigan felt that the cranial erosion was similar to that of a Pacchaonian granulation burrowing into the skull. They suggested that brain rather than a leptomeningeal cyst between the edges of the skull defects produced the cranial cerebral erosion.

In 1953, Taveras and Ransohoff proposed the following mechanism:

"Trauma produces a skull fracture and underlying dural tear; at the same time there is probably sufficient subarachnoid hemorrhage to hinder the local circulation of the cerebrospinal fluid. The arachnoid membrane projects out through the dural tear into the fracture site. This trapped arachnoidal hernia aided by the normal pulsations of the brain gradually erodes the edges of the bone and at the same time compresses the underlying cortex. There must be some degree of ball valve mechanism at work also, with the cerebrospinal fluid having easier ingress into, than egress from, the cyst. Arachnoidal adhesions above the margin of the lesion probably also play a part in trapping the fluid locally."

The authors felt that a dural tear was the single most important factor in the pathogenesis of the lesions and that without it the fracture would heal as expected.

In 1967, Goldstein, et al., demonstrated experimentally that both the dura and arachnoid must be opened for an "enlarging skull fracture" to occur, and the additional involvement of pia, brain, or ventricular communication did not affect the incidence of an "enlarging skull fracture." Cisternal injection of India ink into the cerebrospinal fluid space failed to demonstrate a communication between the leptomeningeal cyst fluid and cerebrospinal fluid. Traumatic growing skull fractures could also be produced in mature experimental animals, indicating that an enlarging cranial vault was not a prerequisite. There was an increased incidence of growing fractures when a pericranial pouch was artificially produced. The direct connection of pericranium to dura without pouch formation was less likely to entrap fluid. Therefore, it was not the simple pulsation of fluid against bone but rather of fluid pulsation in a cyst or pouch that produced the higher incidence of bone erosion. This experimental evidence gave support to the proposal of Taveras and Ransohoff.

Our Case 2 resembles the experimental type which had a pericranial pouch with the pericranium attached directly to the dura. The infrequency of this lesion clinically may be related to the necessity of forming a trapped cystic structure after the pericranium is abnormally attached to the dura by trauma.

In Case 3, we noted that, in addition to the cyst, viable brain herniated through the bone opening and protruded into the scarred pericranium which was attached to the opposite edge, the other dural edge being recessed 1 cm behind the bone and attached to cicatrix (Fig. 4). This was similar to experimentally-produced growing skull fractures. The nature of the cyst contents could not be determined. In Case 3, light microscopy revealed that the cyst wall was composed of fibrous tissue, and electron microscopy that the surface of the cyst wall was composed of loosely arranged collagen fibers without cell lining. Deeper areas consisted of densely packed collagen fibers in bundles, and a few fibroblasts.

With growing fractures, there is a variable latent period of 4 mos to 12 yrs before the linear fracture enlarges. Taveras believes there is first a projection of the arachnoidal membrane through the dural tear and into the fracture site, and that later cerebrospinal fluid pulsations aid in the craniocerebral erosion.

In our cases, however, pericranium was interposed between the bone edges. The pericranium is a natural inhibitor of bone growth and has been used to inhibit bone growth after linear craniectomies in patients with craniosynostosis. This technique is particularly effective in children 3 years or older, inhibiting bone growth for 18 mos or longer. (The major incidence peak for growing fractures is over 3 years.) Later, scar tissue may have produced entrapped loculated cysts, which contributed to the craniocerebral erosion. In other cases, brain tissue herniated through the cyst, possibly associated with a tear in the pia mater (Fig. 4).

Conclusions

On the basis of clinical and experimental evidence we point out three mechanisms that may produce cranial erosion:

Type 1 (Taveras and Ransohoff, Case 1). A dural tear leads to re-
cessation of dural edges, and formation of arachnoidal hernia (Fig. 1 lower).

Type 2 (Goldstein, et al., Case 2). A dural tear leads to recession of a dural edge with pericranium attached, recession of the other dural edge behind the bone edge, the formation of arachnoid cysts, and depression of brain (Fig. 2 lower).

Type 3 (Penfield and Erickson, Case 3). The same process takes place as in Type 2, with the additional formation of surrounding arachnoid cysts and protrusion of brain, possibly secondary to a tear of the pia mater (Fig. 3 lower).

References

Received for publication October 13, 1969.
Supported in part by U.S.P.H.S. Grant 1 SO1-FRS5434 O5.

Address reprint requests to: Frank P. Goldstein, M.D., Department of Neurosurgery, 8700 West Wisconsin Avenue, Milwaukee, Wisconsin 53226.