Large leptomeningeal cyst of the brain

Case report

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An unusually large leptomeningeal cyst of the brain is described in a 9-month-old girl who sustained a head injury at the age of 7 weeks. Although the impression was that the cyst was of the post-traumatic variety, it seemed possible that a congenital malformation of the brain antedated the head injury.

KEY WORDS leptomeningeal cyst growing skull fracture hydrocephalus encephalocele

LEPTOMENINGEAL cysts of the brain may be congenital, inflammatory, or traumatic in origin. Recently we encountered a case which was remarkable because of its enormous size and the difficulties it presented with regard to its etiology.

Case Report

A 9-month-old Nigerian girl was admitted to the University College Hospital, Ibadan, Nigeria, in August, 1969, with a large swelling on the back of her head. The child had been delivered at home on November 5, 1968, after a full-term normal pregnancy. She was described as a normal child. On December 24, 1968, when she was about 7 weeks old, a coconut fell on her head. She lost consciousness for about 30 minutes, and was admitted into another hospital in Nigeria where she was kept for a week for observations and investigations and later discharged home. Three weeks after the accident, a small swelling was noticed on the left side of the back of the child's head; the swelling progressively increased in size. According to her mother, there had been no swelling before the head injury, and no other significant symptoms afterward.

EXAMINATION. The child was active, well-nourished, and cheerful, but she could not sit up. She had a large soft cystic nontender swelling in the left parietooccipital region (Fig. 1). It was not pulsatile and measured 36 cm at its base, 26 cm in the sagittal, and 19 cm along the coronal plane. The bone edges were well-defined, and there was no bruit. No neurological deficit was elicited. All other symptoms were normal. Plain skull
radiographs showed a huge parietooccipital skull defect, the edges of which were raised and sclerotic, indicating that the large mass seen overlying the defect had come from within the intracranial cavity. The lateral projection showed a large bone fragment lying to one side of the mass, lifted up and opened out widely, like the page of a book (Fig. 2). Air ventriculography showed gross ventricular dilatation, which was more pronounced in the left ventricle beneath the defect in the skull (Fig. 3). In the brow-down position, it was shown that there was a direct communication between the ventricle and the parietooccipital cystic mass.

**Operation.** Surgical exposure on August 28 revealed a huge thin-walled multiloculated leptomeningeal cyst beneath an intact pericranium. The cyst contained clear colorless CSF which was later found to be normal on microscopic examination. The edges of the dura had receded and disappeared beneath the edges of the defect in the skull. The brain was virtually atrophied and only a thin sliver of brain tissue remained of the whole of the left occipital lobe. In the anterior part of the space taken up by the cyst could be seen the cavities of the posterior and inferior horns of the left lateral ventricle which communicated freely with the cyst. It was obvious that surgical treatment would present difficulties in an advanced case like this.

**Fig. 2.** Plain skull radiograph, lateral projection, showing the eroded, sclerotic bone margin and the fracture fragment widely displaced by the large cyst.

**Fig. 3.** Air ventriculography. *Left:* Frontal projection shows dilated lateral ventricles, especially the left which is ipsilateral to the cyst. *Right:* Brow-down lateral projection shows air at the top of the cyst.
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The posterior part of the cyst was removed. It was not feasible to obliterate the ventricular connections with sutures. The normal dura encountered beyond the edge of the fracture was separated with difficulty from attenuated brain. Part of the redundant pericranium was used to close the defect in dura. Since a watertight closure could not be satisfactorily accomplished because of the size of the defect and the involvement of major dural sinuses, a cranioplasty was not performed.

Three weeks later, a ventriculoperitoneal shunt was performed using a Till-Dahl-Wade valve, for the relief of the hydrocephalus. Blockage at the abdominal end of the shunt necessitated revision of shunt system. We have not seen the child since she was discharged in November, 1969.

Discussion

One of the problems in this patient was that of unraveling the cause of the leptomeningeal cyst. A few examples of this type have been cited by others. In our patient, it seemed more probable that the leptomeningeal cyst was of traumatic origin, although there was the good possibility of an underlying congenital lesion antedating the head injury.

Dyke in 1937 used the term "leptomeningeal cyst" to describe an already recognized syndrome that sometimes complicated head injury in childhood. Later the term "growing skull fracture" was favored by others. The features of this syndrome comprise a parietal skull fracture in infancy or childhood, unsuspected laceration of dura and arachnoid, brain injury beneath the fracture, and subsequent enlargement of the fracture to form a cranial defect. The radiological characteristics of the syndrome include widening of the old fracture, scalloping and sclerosis of the bordering inner table of the skull, localized increase in vascularity of the bone, and enlargement of the ventricle under the skull fracture.

That the large parietooccipital skull defect was the result of the "growth" of a fracture sustained at the age of 7 weeks could have been more convincingly demonstrated had one seen the skull radiographs obtained at the other hospital where the child was admitted immediately after her head injury. Nevertheless, the widely displaced bone fragment shown on plain skull radiography 7 months later, the sclerosis of the borders of the cranial defect, and the ventricular dilatation which was more pronounced in the ventricle on the side of the skull defect, all strongly suggested that this was a post-traumatic leptomeningeal cyst. Lende and Erickson in 1961 have commented on this development in the first year of life.

Goldstein, et al., classified post-traumatic leptomeningeal cysts into three varieties. Our case probably exemplifies still another variety. Following the head injury, the damaged brain could herniate through the defect in the meninges and the skull. If the brain hernia persists and the dural defect is not closed, the dilated ventricle beneath the fracture site could migrate to the surface as the brain progressively atrophies, and later form a connection with the arachnoid cyst. Such a sequence of events probably occurred in our case. This type of cyst is unlikely to be encountered in communities where patients can be followed easily and early diagnosis thus made. Where, on the other hand, lesions are seen when they are very advanced, as in this case, its occurrence is quite possible.

The hydrocephalus noted in our case was probably initiated at the time of the head injury by associated subarachnoid hemorrhage which later interfered with the circulation of cerebrospinal fluid. The enormous size attained by the cyst is understandable as the CSF had easier ingress into the cyst than egress from it. It is reminiscent of the "meningocele spuria" described by Billroth in 1862 which grew gradually until it became as large as the head of the child, and at autopsy there was a direct communication between the cyst and the lateral ventricle.

The other possibility is such that in this child a congenital lesion antedated her head injury. One such lesion could be a preexisting parietooccipital encephalocele which was activated to grow by the traumatically-provoked hydrocephalus. Encephaloceles of the parietooccipital region are not unknown in Nigeria. That the mother denied the existence of a swelling in the head before the injury is not absolutely reliable evidence. In advanced lesions like this, the guilt for delaying hospital treatment often leads relatives to compromise the factual details.
Granted that the history was correct, this child could have had a congenital arachnoid cyst antedating her head injury. Congenital arachnoid cysts of the brain, though rare, have been described in children in the first year of life. Some of these cysts have manifested clinically on their own,\(^1\) while others have been brought to light after a head injury,\(^7\) suggesting a mixed etiological origin. The virtual absence of the left occipital lobe noted at operation in our patient could have resulted from agenesis and replacement by the cyst\(^8\) or from the effect of traumatic disintegration following damage to its blood supply during the head injury.\(^11\)

References

Received for publication November 30, 1970.
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