LEPTOMENINGEAL CYSTS OF THE BRAIN FOLLOWING TRAUMA WITH EROSION OF THE SKULL

A STUDY OF SEVEN CASES TREATED BY SURGERY

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LEPTOMENINGEAL cysts, which result from trauma, are fluid-filled spaces between the pia mater and the arachnoid membrane. The fluid-containing pockets are not true cysts because they are not completely isolated from the subarachnoid space, although they are confined by pia-arachnoid adhesions from free communication with the remainder of the subarachnoid space. Pulsations of the brain are transmitted through the cysts to the inner table of the skull and erosion of the bone ordinarily occurs in a localized area overlying the cysts.

It is the purpose of this paper to present, in summary manner, the case histories of 7 patients with leptomeningeal cyst following trauma and to evaluate the results of surgical treatment. The theory of formation of the cysts will be discussed together with the mechanism of the associated bone erosion. Cysts resulting from congenital malformation, from inflammation and from all causes other than trauma are excluded from this study.

MATERIAL

At the Neurological Institute of New York leptomeningeal cysts are relatively rare in their occurrence. The lesion has been encountered more frequently in recent years since the clinical features have been made known and patients have been referred for neurosurgical treatment. The development of leptomeningeal cysts has been recognized more often also as a result of repeated radiographic examinations following skull fracture. During the last two decades 7 patients with verified leptomeningeal cysts have been studied at this institution.

CASE REPORTS

Case 1. Unit No. N.I. 9400. M.K., an 18-year-old girl, was admitted on July 2, 1929, complaining of right-sided convulsions and a left parietal skull defect which had been first noticed in childhood and had enlarged progressively. At the age of 16 months she had been struck by a train. The details of the accident and the period of unconsciousness are not known.

Neurological findings on admission were a left parieto-occipital skull defect and slight awkwardness of the right hand. Roentgenograms of the skull revealed an irregular skull defect, 8 cm. in length, in the left posterior parietal region.
At operation the dura mater was found to be thinned out about the periphery of the defect and absent in its center. The area was filled with a $3 \times 2 \times 4$ cm. cyst which was not in communication with the ventricles. The dural defect was closed with a Cargile membrane.

The convulsions persisted for the 1st postoperative year and then disappeared. The patient has been symptom-free without medication for the intervening 19 years.

Case 2. Unit No. 18791. I.M., a 9-year-old boy, was admitted on Jan. 30, 1934, with the chief complaint of left-sided convulsive attacks since 1933. In 1925, when he was 3 months of age, he had fallen from his carriage and had sustained a skull fracture followed immediately by paralysis of the left arm and leg. The hemiplegia cleared up slowly, but not completely.

He was a well-nourished and well-developed child. Irregular bulging was noted in the right posterior parietal region (Fig. 1A). There was mild spastic left hemiparesis with slight left hemiatrophy. He was grossly retarded mentally. Roentgenograms of the skull revealed two large defects in the posterior portion of the right parietal bone with scalloping of the margins of the defect consistent with post-traumatic leptomeningeal cyst (Fig. 1B). Pneumoencephalography showed moderate dilatation of the posterior portion of the body of the right lateral ventricle.

At operation a multicystic space was found. The dura mater was absent over these cysts, which were partially formed by degenerated brain tissue. The dural defect could not be closed at this time because of uncontrollable bleeding which delayed the procedure, but it was tightly closed at a second-stage operation.

Following surgical intervention the convulsions became localized to the left leg and later disappeared completely; the hemiparesis improved. The mental retardation remained unchanged over an 8-year follow-up period.