POST-TRAUMATIC ARACHNOIDAL CYST

REPORT OF AN UNUSUAL CASE

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Two varieties of arachnoidal cysts have been described: those developing within the layers of the arachnoid membrane itself, and those arising in the subarachnoidal space, walled off by pia on the inner side and arachnoid on the outer side. The latter are more appropriately called subarachnoidal cysts. Both types are considered to be relatively rare. In 1934 Horrax collected from the literature cases of arachnoidal cysts located in the cisterna magna, de Martel and Guillaume described 7 cases of arachnoidal cysts in the posterior cranial fossa in 1930. Additional cases have been collected by other authors. Contrary to the general belief that arachnoidal cysts are rare, Klein and Brogly estimated that they account for 15 per cent of all infratentorial space-occupying lesions. They referred to cases of their own. Several authors considered a head injury responsible for the development of arachnoidal cysts.

Recently we came across a patient with an arachnoidal cyst in the occipitoparietal region who gave a history of head injury 50 years prior to admission to the hospital. The cyst in this case communicated freely with the left lateral ventricle. Such a condition, as far as we know, has never been reported before in the literature.

CASE REPORT

A 54-year-old male usher was admitted to Medical Department C of the Rambam Government Hospital on Dec. 25, 1959 with chief complaint of headache, fever and stiffness of the neck of 5 weeks’ duration.

Examination. The patient was in good general condition. His temperature was 39°C. He had marked rigidity of the neck and positive Kernig’s and Brudzinski’s signs. Lumbar puncture yielded xanthochromic fluid containing 200 mg. per cent protein, 40 mg. per cent sugar and 116 mEq./liter chlorides. There was marked pleocytosis with 200 polymorphonuclear cells, 50 lymphocytes and 50 red cells per c.mm. Cultures of cerebrospinal fluid and blood remained sterile.

Course. Penicillin and streptomycin were administered and the temperature gradually came down to normal. Since neurological abnormalities persisted a neurological consultation was requested. Further investigation of the patient’s history revealed that at the age of 4 he had fallen on the back part of his head. As far as he knows he remained unconscious for 2 months and since that time he has not been able to see on his right side. At the age of 10 he began to suffer from epileptic fits, which subsided spontaneously some 10 years later. At the age of 28 he was struck upon his face and his nose was broken. In 1942 he sustained a back injury and has been suffering from attacks of low-back pain ever since. Two years prior to admission he noticed unsteadiness of gait and a tendency to fall, especially at night.

Neurological examination revealed slow incoordinate speech, signs of meningal irritation, right homonymous hemianopia, marked ataxia of gait and a positive Romberg’s sign. The left plantar response was extensor. Nystagmus was demonstrated on looking to the sides and upwards. A hard uneven mass covered by normal skin was palpated over the occipital bone. It was not tender. Roentgenograms of the skull showed separation of the two tables of the occipital and the greater part of the parietal bones (Figs. 1 and 2). The space between the two tables had a multilocular appearance because of bony septa of various sizes and shapes. The lesion was thought to be compatible with either a primary cholesteatoma or a multilocular cyst of unknown origin. Pneumoencephalography showed a normal 4th ventricle, aqueduct and posterior part of the 3rd ventricle. The lateral ventricles were not demonstrated adequately. Electroencephalography was reported as normal.

Operation. On Feb. 3, 1960 suboccipital exploration was performed. A huge multilocular cyst of the occipital bone extending far into the parietal bones and communicating freely with the left lateral ventricle was found. The cyst contained cerebrospinal fluid. Part of the posterior wall of the cyst was removed. The anterior wall, composed of the inner tables of the occipital and parietal bones, displaced both cerebellar hemispheres in an anterior direction. Many bony spurs of various sizes and shapes protruded from the inner table into the cystic cavity, giving it the appearance of a cave full of stalactites and stalagmites. In the anterior bony wall of the cyst there was a round opening of approximately 4X4 cm. leading into the left lateral ventricle, in which the choroidal plexus was seen. The left occipital lobe, as well as the meninges over the opening of the ventricle, were entirely absent. The inside of the bony cyst was covered partly by a thin membrane, histologically composed of fibrous connective tissue. Since it was felt that nothing would be accomplished by further exploration, the wound was closed.

Postoperative course was uneventful and the patient was discharged from the hospital 3 weeks after surgery. He returned to his previous work. His neurological status remained unchanged.
DISCUSSION

Horrax considered arachnoidal cysts to be of inflammatory origin and referred to them as "generalized cisternal arachnoiditis simulating cerebellar tumor." Some of them developed following infection of the middle ear, de Martel and Guillaume in their cases emphasized the sudden onset of headache with stiffness of the neck and suggested meningitis or subarachnoidal hemorrhage as an etiological factor. In one of the cases described by Craig a multilocular cyst with fibrous and adhesive changes was found in the posterior fossa in a 9-year-old boy, subsequent to prolonged, severe pulmonary infection, complicated by signs of meningeal irritation. Brobeil and Maneke in one of their cases found histological evidence of inflammation, thus supporting the theory of inflammatory origin of such cysts.

Bucy suggested a congenital etiology. In the case described by him a small tuft of choroidal plexus was found attached to the wall of the cyst. This ectopic choroidal plexus was supposed to be responsible for the presence of fluid within the cyst. The congenital etiology is also supported by Trowbridge and French in spite of the fact that in their case a history of head injury 5 years previously was elicited. Starkman et al. reported 4 autopsied cases of arachnoidal cysts, 3 of which were located between the layers of the arachnoid. They assumed that these cysts arose as a developmental abnormality. In their fourth case, however, the cyst was actually a dilatation of the subarachnoidal space, walled off by adhesions, following severe meningitis, thus being of inflammatory origin.

In 1946 Thompson published the first report in which a traumatic origin of arachnoidal cysts was considered. According to his view, a leptomeningeal inflammatory reaction developed in the region of the cisterna magna as a result of blood or bacteria entering the subarachnoidal space following injury. This inflammatory reaction produced scarring and thickening of the meninges and resulted in partial obstruction of the cerebrospinal pathways leading to dilatation of the cisterna magna. This resulted in the formation of an arachnoidal cyst. In the case reported by Nichols and Manganiello there was a history of head injury 8 years prior to the detection of the cyst. Taveras and Ransohoff offered an explanation of the development of traumatic arachnoidal cysts in a report of 7 surgically verified cases, in all of which the patients had had a fracture of the skull before the age of 6. The same trauma, in their opinion, caused a tear in the dura mater with subsequent arachnoidal herniation. Some degree of a ball-valve mechanism favors the inflow of cerebrospinal fluid into the cyst. The pulsating arachnoidal sac may erode bone and press on the brain. The dural tear is probably the most important single factor in the pathogenesis of this condition. Without it the fracture would heal as expected. In all cases the dura mater was absent in the center of the bone defect, and was markedly