Hemorrhage into an intradiploic arachnoid cyst

Case report

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Intracranial arachnoid cysts are relatively rare; it is believed that they account for only 1% of all intracranial space-occupying lesions. When they occur in the intracranial cavity, they usually develop in relation to an arachnoid cistern as a pocket of cerebrospinal fluid within two layers of arachnoid membrane. Five cases of intradiploic arachnoid cysts have been reported, but an arachnoid cyst presenting as an extradural mass has not been described before. The authors present an unusual case of hemorrhage into a massive intracranial extradural arachnoid cyst with no intradural communication.

Key Words • extradural mass • cyst, arachnoid • hemorrhage • skull

Intracranial arachnoid cysts are rare, and account for only 1% of all intracranial space-occupying lesions. These lesions are benign developmental cysts that occur in the cerebrospinal axis and affect the arachnoid membrane. Virtually all intracranial arachnoid cysts are located intradurally, with the possible exception of intrasellar arachnoid cysts, which are external to the diaphragma sellae. In 1831, Bright was the first to accurately describe the disease, and in 1964 Robinson reported a large number of personal cases of middle fossa arachnoid cysts, postulating temporal lobe agenesis as the cause. All of Robinson’s cases of intracranial arachnoid cysts were strictly intradural.

Five cases of intradiploic arachnoid cysts have been reported. We describe the case of a patient presenting with hemorrhage into a massive intracranial extradural arachnoid cyst.

Case Report

This 35-year-old man had the sudden onset of headache, nausea, and vomiting 1 year prior to admission. He was treated with intravenous fluids at a municipal hospital in another city, after which he recovered and was discharged. Thereafter, he complained of episodic morning headaches associated with nausea and visual obscurations. The frequency of these episodes gradually increased. Four months before his present admission he suddenly exhibited proptosis with redness of the right eye. Over the next few days the redness subsided, but the proptosis persisted. He had no diplopia. About 5 years previously, he had been involved in a motor-vehicle accident and was dazed for 10 minutes. At that time, he had experienced serous otorrhea of his right ear which ceased after local instillation of ear drops.

Examination. On admission to our hospital, the patient had early papilledema in the right eye, depressed right corneal and facial sensation, and jaw deviation to the right. His vision was 6/6 in both eyes. The right eye was proptosed and displaced downward; external movement of the right globe was slightly restricted.

Roentgenograms of the skull (Fig. 1) showed a large right frontotemporal cartwheel-like osteolytic lesion with inward displacement and a thinned inner table of the skull. Multiple trabeculae were seen traversing the lesion. The right lesser wing of the sphenoid bone and the ipsilateral orbital roof were displaced upward. The outer table of the skull was preserved. There was a defect in the orbital roof. Computerized tomography (CT) scans of the head showed a huge extradural, uniformly hyperdense, doubtfully enhancing mass in the right frontotemporal region. The mass extended into the right orbit and the middle fossa and medially up to the parasellar area (Fig. 2). It compressed the right frontotemporal portion of the brain and displaced the ipsilateral frontal horn.

Operation. A large right frontotemporal scalp flap was raised. While a low temporal burr hole was being
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Figs. 1, 2. X-ray films of the skull, anteroposterior (left) and oblique (right) views, showing expanded diploic space with a trabeculated appearance. The ipsilateral lesser wing of the sphenoid is elevated and a lesion is clearly visible.

Drilled, blackish thick fluid oozed out. After the bone flap was made, an encapsulated mass filled with thick, sticky, greenish-black material was seen. It was inspissated and had to be scooped out because it could not be suctioned away. The mass extended into the orbit through a hole in the orbital roof. The capsule, which was loosely adherent to the inner table of the skull and firmly attached to the dura, was removed piecemeal. No defect could be found in the dura despite a diligent search. After hemostasis was achieved, the bone flap was replaced and the incision was closed in layers.

Postoperative Course. The postoperative course was uneventful; the patient’s proptosis decreased, but the right corneal and facial hypesthesia persisted. Histopathological examination of the mass was consistent with organized hematoma with inspissated plasma; there were few red blood cells or cholesterol crystals. The capsule resembled arachnoid membrane (Fig. 3).

Discussion

Intradiploic arachnoid cysts have been reported in the literature by Sartawi, et al., D’Almeida and King, and Weinand, et al.; these cysts extended through dural defects into the diploic spaces. D’Almeida and King described two patients who had asymptomatic osteolytic lesions of the skull associated with “cerebrospinal fluid diploic fistulae.” They postulated that each patient had suffered a forgotten head trauma resulting in a small linear fracture of the inner table of the skull with injury to the dura and subsequent development of an arachnoid pouch through the dural defect. The patient described by Sartawi, et al., had a history of trauma, although x-ray films of the skull revealed no fracture. They found a diploic arachnoid cyst originating via a small defect in the dura. In these three cases trauma was postulated as the cause of the arachnoid cysts. Weinand, et al., reported two patients with diploic arachnoid cysts of congenital origin, neither of whom had a history of head trauma. The eroded area of the inner table of the skull had smooth and well-defined margins, in contrast to the appearance in posttraumatic cases.

We believe that all five previously reported cases represent modified pacchionian corpuscles rather than true intradiploic arachnoid cysts for two reasons. First, the skull defect found in all of these cases was either parasagittal or related to the lateral sinuses, where arachnoid granulations or pacchionian bodies abound. The lesions produced impressions on the
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inner table of the skull, but in some instances also involved the diploe and sometimes even the outer table of the skull. The mass may be unicameral or made up of closely packed chambers. Second, all of these patients presented moderately late in life (in their fifth or sixth decades). Pacchionian bodies appear at 18 months of age, then gradually increase in size, number, and extent with advancing years. None of the five patients described above had symptoms or signs suggestive of raised intracranial pressure; they presented only with local pain. They were operated on because skull x-ray films showed osteolytic lesions, which might also have been produced by a tumor.

Our patient differed from the previous five cases. He was a young man with a history of insignificant head trauma 5 years earlier. He had symptoms of raised intracranial pressure (episodic headaches, nausea, and vomiting) and of a space-occupying mass (proptosis and papilledema in the right eye). Roentgenograms of the skull did not show a fracture, but were suggestive of diploic space expansion rather than osteolytic lesions. There was displacement rather than destruction of the orbital roof and the lesser sphenoid wing. During surgery, no defect was found in the dura, although the appearance of the cyst wall was typical of arachnoid tissue.

The exact pathogenesis of the lesion in our case can only be speculated upon. There may have been a congenital defect in the dura and overlying bone with subsequent development of an arachnoid pouch through the dural defect. The continual pulsations of the arachnoid membrane herniating through this defect and the inner table of the skull led to expansion of the diploe. Upward displacement of the lesser sphenoid wing and the orbital roof favor the possibility of the arachnoid cyst originating near the pterion. Hemorrhage within the cyst followed, but organization of the capsule may have obscured the delicate relationship between the hematoma and arachnoid membrane and may have sealed off the connection between the arachnoid cyst and the intradural compartment. Spontaneous disappearance of intradural arachnoid cysts following hemorrhage, presumably due to occlusion of the intradural connection by the organizing hematoma, has been described in the literature.

In another possible etiology, the arachnoid may have been congenitally sequestered into the diploe then gradually enlarged inward within the diploe, destroying a large part of the inner table of the skull, leaving the outer table intact. A similar mechanism is implicated in congenital spinal extradural arachnoid cysts, where an arachnoid pedicle breaks through a developmental defect in the dura.

References


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