Supratentorial Brain Cysts

PAUL JAKUBIAK, M.D., REMBRANDT H. DUNSMORE, M.D., AND RONALD S. BECKETT, M.D.

Hartford Hospital, Hartford, Connecticut

We have seen four unusual cases of benign, solitary, supratentorial brain cysts, none of which communicated with the subarachnoid or ventricular systems. Until Starkman and Brown described the clinical entity of supratentorial arachnoidal cysts in 1958, most of these lesions had been included in discussions of porencephaly, pseudoporencephaly, or cystic hygroma.

All of the cysts in our series behaved as intracranial masses, were associated with progressive neurological dysfunction, and were diagnosed preoperatively as brain tumors.

Case Reports

Case 1. This 74-year-old woman entered the hospital in September, 1965, with a 5-year history of progressive right-sided seizures associated with speech loss but not with unconsciousness. The past medical history was negative for trauma, infection, or intracranial hemorrhage. She sustained a coronary artery occlusion 3 years before hospital admission and had been treated with anticoagulants since that time. She also had a congenital left sixth nerve palsy.

Examination. Neurological examination was unremarkable save for the left 6th nerve paresis. Blood studies, urinalysis, blood sugar, urea, and skull x-rays were normal. Echoencephalography showed an 8 mm left-to-right shift. A left carotid arteriogram revealed marked stretching of the posterior parietal and angular arteries as they left the Sylvian fissure. There was no evidence of tumor stain or shunting. The Sylvian point was depressed. A brain scan was negative. The preoperative diagnosis was that of an avascular mass lesion in the left parietal lobe.

Operation. On September 13, 1965, operation through a left frontoparietal bone flap revealed dura under tension, beneath which in the parasagittal area was a cystic structure measuring 4 1/2 cm in diameter (Fig. 1). The cyst produced a distinct 3 cm depression in the underlying parietal parenchyma. The surrounding brain gyri were normal in appearance. The roof of the lesion was opaque to light in places, and there was evidence of a clear fluid in the cavity of the lesion. Approximately 60 cc of fluid were aspirated from the cyst. After the cyst roof had been removed (Fig. 2), it was clear that there was no communication with the lateral ventricle; there was at least 2 cm of brain tissue between the cyst and the atrium of the lateral ventricle. Biopsies were taken of the cyst wall, and the procedure was terminated. The dura was closed in a water-tight fashion and the bone flap replaced.

The patient’s postoperative course was unremarkable. In the past 1 1/2 years, she has experienced only one focal seizure.

Pathological examination. The cyst fluid contained 280 mg/100 ml of protein with 20 mg/100 ml in the lumbar spinal level. The sodium content was 150 meq/liter and the potassium was 2.8 meq/liter. Serum electrolytes were not done. Histological examination of the wall of the cyst revealed ciliated epithelium resembling ependyma and resting on a felt-like neuroglial matrix. There was no evi-
dence of inflammatory infiltrates or hemosiderin deposits. The specimen had two types of tissue. The outer portion resembled cerebral white matter, being a network of glial fibers and glial nuclei recognizable as astrocytes, microglia, and oligodendroglia. Lining the cyst was a second component composed of ependymal epithelium. This layer had the following variations in structure that relate it to early development of the brain:

1. The ependymal cells varied in arrangement from flattened cuboidal, to mesothelial, to stratified columnar.

2. The ependymal cells were applied to the glial tissue in a variety of ways, from flat juncture, to shallow and deep indentations, convolutions, inclusion cysts, and diverticula. The papillary configurations, often having connective tissue cores, justified the descriptive designation of “choroidal cyst of subarachnoid space” (Fig. 3).

3. Cilia were particularly clearly seen (Fig. 4) in the presence of high, stratified, immature ependymal cells like those characteristic of the 7-week embryo. Cilia are ordinarily found in histological preparations of ventricle wall in normal newborn or fetal brains, but not in adults. Cilia do not occur on the choroidal plexus epithelium of adults or fetuses.

4. In summary, this specimen had the properties of partly formed choroid plexus, immature non-choroid plexus, ependyma, and brain tissue without neurons. We reconstruct these observations as the outcome of a displacement of primitive ependyma and mantle primordia outward into the subarachnoid space, at a time near the 2-month stage of embryo development.

**Case 2.** This 49-year-old man entered the Hartford Hospital on May 13, 1965, with complaints of pounding occipital headache exacerbated when he moved from horizontal to upright position, plus difficulty with balance and memory complicated by 1 week of nausea and vomiting. Throughout his life he experienced a resting tremor involving both hands.

**Examination.** The patient was somewhat evasive in his answers and had a short attention span. A talipes cavus deformity was noted in the left foot. Neurological examination was normal except for a fine low-amplitude tremor of both arms. Skull films were negative for erosion, thinning, or other evidence of intracranial pressure. Electroencephalography showed a dysrhythmic slow activity over the left hemisphere, most prominent in the left temporal area. Brain scan was negative. A pneumoencephalogram showed evidence of a mass lesion involving the left frontal lobe, displacing the left frontal horn beneath the falc. The opening cerebrospinal fluid pressure at the time of this test was 240 mm of water.

**Operation.** On May 16, 1965, through a left frontal osteoplastic bone flap, reflection of the dura showed a large cystic lesion occupying the most anterior and medial portion of the left frontal lobe. The surrounding cortical gyri appeared normal; the medial cyst wall abutted on the anterior falc. After the cyst was unroofed, 100 cc of clear, colorless fluid gushed forth. The interior walls were smooth, glistening, and appeared to be composed of compressed white matter. There was no evidence of a mural nodule or hemosiderin staining. The most medial portion of the lesion showed a bluish ependyma-like lining which probably corresponded to the lateral wall of the third ventricle. At no point did this cyst appear to communicate with the ventricular system or cerebral subarachnoid space.

**Postoperative course.** The patient’s postoperative course was benign. He was dis-
FIG. 3. Case 1. Complex structure of cyst wall showing ependymal epithelium with areas of papillary choroid plexus-like configuration, indentations, and isolated cysts. Subependymal glia cells and brain feltwork are also shown. There are no neurons. H. & E., ×400.
charged from the hospital greatly improved and free of occipital headache. At follow-up examination 1½ years later he was free of his former complaints and working daily. The resting tremor had disappeared except in the right hand.

**Histological examination.** Limited nodules and filaments of brain matrix and glial cells were found partly covered by mature ependymal cells, occasionally showing foci of nuclear variation in size, and stratification of the cells. This arrangement tapered off to places where flattened mesothelial or acellular membrane formed the cyst surface (Fig. 5). Sections across the filaments showed strands of glial tissue, blood vessels, and ependymal epithelium lining the inner wall and mesothelial cells on the outer wall facing the meninges (Fig. 6).

It seems to us that this cyst was similar in nature and origin to that in Case 1, but that it had probably undergone further maturation and lost the immature appearance of nuclei and cells, the choroidal configuration, and the ciliation of the ependymal epithelium.

**Case 3.** This 32-year-old man had had monthly grand mal seizures for 1 year. He had experienced an isolated seizure at age 19. The past medical history was otherwise unremarkable.

**Examination.** Neurological examination was completely normal. Hemogram, urinalysis, blood sugar, and urea were all normal. Skull films showed a 4 mm shift of the pineal gland toward the left. Electroencephalography showed evidence of right parasagittal, paroxysmal rhythm, more compatible with a seizure disorder than a space-occupying lesion. Pneumoencephalography showed a right parasagittal mass lesion displacing the anterior horns of both right and left lateral ventricles to the left (Fig. 7 and 8). The preoperative diagnosis was a parasagittal mass lesion.

**Operation.** After reflection of the dura through a 2-inch right, medial, frontal trephine, a cyst was uncovered which appeared at the cortical pial surface with normal surrounding brain gyri. The cyst was attached at the periphery of the cortical indentation by a thin white line. After the roof had been removed, the arachnoidal membrane could be dissected away from the pial surface of the compressed, but otherwise normal, gyri at the depth of the lesion. The cyst measured 6 cm
Fig. 5. Case 2. Filament of cyst wall with flattened and cuboidal epithelial surface above and to left, flat membranous surface below; calcified venule, and ribbon of glial tissue between the two surfaces. The less cellular surface is that facing the meninges. H.&E., ×100.

Fig. 6. Case 2. Section through cyst at junction of several filaments. Epithelium generally more mature than in Case 1, but apparently several layers thick. No cilia or choroid plexus-like configurations. Glial tissue abuts on cyst wall. H.&E., ×400.
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FIG. 7. Case 3. Pneumoencephalogram showing displacement of frontal horn and body of lateral ventricle beneath falx. The cyst does not fill with air.

FIG. 8. Case 3. Pneumoencephalogram showing indentation of body of lateral ventricle by cystic mass.

FIG. 9. Case 3. Cyst wall contains collagen fibers and flattened cells of mesothelial or meningocyte appearance arranged in sparse and occasionally interrupted patches and fragments. No glial tissue or ependymal cells can be seen. H. & E., X400.

in diameter and displaced the superior and middle frontal gyri laterally, while resting on the anterior falx medially. Approximately 150 cc of clear spinal fluid was contained within it.

Postoperative course. The patient's course, to the time of this writing, reveals that he has fewer seizures and is able to work full time at his profession.

Pathological examination. Sections were much less complex than those of Cases 1 and 2, and contained principally fibroblasts and collagen fibers with rare capillaries (Fig. 9). Occasional clusters of discontinuous flat mesothelial cells were present on both surfaces. Areas like this were seen on the meningeal surface of Case 2 and in some circumscribed zones in Case 1 where the ependyma was absent or markedly flattened. We felt that this lesion was a cerebral arachnoid cyst probably formed within a split arachnoid membrane, as described by Starkman and Brown.17

Case 4. This 50-year-old man had two right-sided seizures on the day of admission followed by an aphasic episode which lasted approximately 12 hours. The past history was noncontributory.

Examination. Except for slight residual receptive aphasia, the neurological examination was entirely normal. A pneumoencephalogram showed 5 mm shift of the midline structures from left to right. X-rays of the skull were normal; the pineal was not calcified. A left carotid arteriogram gave evidence of a mass lesion in the left temporoparietal area. Brain scan was negative.

Operation. A left temporoparietal bone flap was turned, and after reflection of the dura, a large cystic structure was identified which ap-
peared to occupy the Sylvian fissure. It was covered with a filmy membrane with a rather dense, thin, white line at the periphery of the lesion. When the membrane had been removed, essentially normal gyri could be seen in what appeared to be the island of Reil at the depth of the exposure. Approximately 170 cc of crystal clear fluid were within the cystic cavity.

Pathological examination. The microscopic appearance of the cyst wall was that of a double membrane separated by loose strands of collagen, fibroblasts, and occasional capillaries. Each of the surfaces consisted of a patchwork of flattened or cuboidal cells. At irregular intervals they proliferated to form clusters, having the appearance of the meningocytic caps normally present in the arachnoid. There was none of the ependymal features described in Cases 1 and 2, and the cyst was felt to be similar in character, origin, and development to that in Case 3.

Discussion

Several types of brain cysts have been described that can be considered in the differential diagnosis.

The porencephalic cyst was defined by Heschl as communicating with either the subarachnoid space or ventricular system. Since none of these cyst lesions communicated with the subarachnoid space or ventricular system, they do not correspond to Heschl’s description of porencephalic cysts. The cyst linings from our cases were carefully examined histologically for evidence of hemosiderin deposits and inflammatory infiltrates but there were none found. One would expect that if previous cerebral infarction or spontaneous hemorrhage were etiological, the lining of the cyst cavities would consist of neuroglial fibers with possibly fibrous strands transversing the cavity, as well as hemosiderin deposits in the cyst wall. Heschl favored a developmental origin for these lesions, but Kundrat favored an arterial vascular accident since many of these porencephalic cysts were in areas of middle cerebral artery distribution. There was no evidence of bleeding, infarction, or communication with a ventricle or subarachnoid space in our cases, so that porencephaly is ruled out.

Yakovlev, et al., have described cleft-like symmetrical hemispherical lesions which became cyst-like with a concomitant hydrocephalus. They termed this condition “schizencephaly” and ascribed it to a developmental defect of the cerebral mantle. However, they emphasized the presence of micropolygyria adjacent to the lesion as an almost constant finding. The cases that they described at postmortem examination were imbeciles during life. This was not so in our patients. Davison described the case of a 70-year-old man with an apparently asymptomatic cyst in the right inferior frontal lobe discovered postmortem. The essential findings consisted of a cystic structure overlying atrophic gyri of the third frontal and temporal convolutions. Histological examination did not confirm the gross atrophic appearance. A detailed description of the cyst lining was not given. The defect was ascribed to a defect in the development of the brain mantle layer and thought to be present at birth.

Lichtenstein has stated that ependymal streaks, ependymal rosettes, and ependymal-lined cavities are frequently observed in the occipital white substance as a result of growth of tissue surrounding the occipital horn of the lateral ventricle. These ependymal cells can also be found in the aqueduct as well as in the lateral angles of the ventricular system. One would wonder if isolated collections of these ependymal cells in the periventricular location could not have given origin to the cysts in our first two cases.

Netsky, in a recent paper on the genesis of neuro epithelial cysts in and around the third ventricle, showed that evagination of the ependymal layer could occur and thereby produce ependymal outpouchings in an extraventricular position and thus resemble a mass lesion. It is difficult to say why cysts of the subarachnoid areas such as ours should slowly enlarge and act as space-occupying lesions except that it is entirely possible to have epithelia-like lining maintaining a secretary function. The hypersomolality of the cyst fluid could also draw water from surrounding tissue and thereby contribute to its enlargement. Cases 1 and 2 could represent an extension of Netsky’s demonstration that an extraventricular choroid plexus epithelium is the origin of colloid or neuro epithelial cysts.

At the second month of brain development, there are three identifiable layers. The ependymal layer which is innermost, the mantle layer which is intermediate, and the marginal
layer. As development takes place, the ependymal layer sends out processes to the marginal layer. Some neuroblasts migrate out to the mantle layer and take up positions in what will become the cortical gray matter. These cells are neuroblasts and have been derived from spongioblasts in a periventricular ependymal position.

Cooper, et al., and Wolbach have described nodules of glial tissue in the subarachnoid space. They report ependyma-lined canals within these nodules which they attribute to developmental changes. We feel that these structures could be precursors to the cysts described in this paper. It is entirely possible that what we have seen in Cases 1 and 2 are developmental defects in which there was a disorder in the formation of the mantle layer followed by displacement of the ependyma into the subarachnoid space. The isolated and pinched-off immature lining cells give origin to these cysts. We regard Cases 3 and 4 as representative of the arachnoidal cyst as described by Starkman and Brown in which they demonstrated splitting of the arachnoidal membrane at the periphery of the defect. The thin white line at the periphery of the defect probably represents the site of splitting of the arachnoidal membrane. The parasagittal location has been mentioned as a potential site; the others being the Sylvian fissure, orbital sulcus, and central fissure.

**Summary**

We have reported the successful surgical treatment and pathological study of four cases in which a supratentorial brain cyst acted as a space-occupying lesion. We have discussed the differential diagnosis and speculated as to the developmental defects that gave origin to the cyst.

**References**

12. Netsky, M. G. *Personal communication.*