BENIGN CYSTS OF THE BRAIN SIMULATING BRAIN TUMOR

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In general the term “benign cyst of the brain” would be regarded as indicating a porencephalic or arachnoidal cyst. Although there is no complete agreement as to the definition of these terms or as to the etiology of these conditions, it is generally believed that the term “porencephalic cyst” refers to a congenital condition in which a cystic cavity is created by a defect in the brain. Such cysts usually communicate either with the ventricular system or with the intracranial subarachnoid spaces.\textsuperscript{5,6,11,12,15–17} Occasionally a cystic cavity is seen that is separated from the ventricular system or the subarachnoid space by a thin membrane. Such cavities may be distinguished by the terms: incomplete porencephaly, pseudoporencephaly or closed porencephaly.\textsuperscript{16} In the main, however, any symptoms of cerebral involvement presented by patients with porencephalic cysts arise as the result of the cerebral defect and not because of the cyst, which is purely passive and secondary development. The symptoms most commonly seen in such cases are hemiparesis and convulsions. Arachnoidal cysts may arise secondary to an infectious or inflammatory process in the leptomeninx, or they may be congenital or occur following severe cerebral trauma.\textsuperscript{2,3,8,9,14,19,20} If symptoms of cerebral involvement are present with such cysts, convulsions are the most common.

Here we are concerned with a group of cysts that do not fall into either of the above categories, neither are they neoplastic in origin. These cysts lay within the brain substance and did not communicate either with the ventricular system or the subarachnoid space. They gave rise to symptoms and signs of increased intracranial pressure and were associated with other manifestations which were similar to those commonly associated with intracranial neoplasms. Two of these cysts lay in the cerebral hemisphere and one was in the cerebellum.

CASE REPORTS

Case 1. I.B., a 25-year-old married female, was first seen at the University of Chicago Clinics in 1931, complaining of convulsive seizures, tremor of the right hand and headaches of 1 month’s duration. She had had several convulsions in childhood, and they recurred in 1923 following the birth of her child. They had then again subsided to recur in 1930 and to continue at intervals of every 2 or 3 months.

Examination. On May 27, 1931, positive neurological findings were a right hemi-

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paresthesia, a right hemihypesthesia, and a right lower quadrant homonymous hemianopsia. Lumbar puncture revealed nothing abnormal. The initial pressure was 160 mm. of water. The fluid was clear and colorless. It contained 3 white blood cells. There was a trace of globulin on Pandy's test. The Wassermann reaction was negative and the colloidal gold curve was 000000000.

She was placed on anticonvulsant medication but when she returned in February 1932, her convulsions were more frequent and her headaches more severe. The findings on examination were unchanged.

Operation. On Feb. 24, 1932, ventriculography was done. Through burr holes in the occipital region blunt cannulae were inserted into the region of both lateral ventricles. In all, 75 cc. of clear colorless fluid were removed and replaced with air. The ventriculogram revealed an enormous cyst in the left temporoparietal region. It measured 10×6.5 cm. in the lateral roentgenograms and 7×10 cm. in the anteroposterior films. The cyst was separated from the left lateral ventricle by a thin membrane (Fig. 1) and the ventricular system was displaced toward the right. In the lateral view (Fig. 2) the lateral ventricle was lifted upward and forward and again was separated from the cystic cavity by a layer of cerebral tissue. Until these films were seen it was not realized that the cannula on the left side had entered a cystic cavity. Fortunately the fluid from the two needles had been collected separately. That from the right needle (right lateral ventricle) contained 21.8 mg. of protein per 100 cc. That from the left needle (cystic cavity) contained 95.5 mg. per cent.

On Feb. 29, 1932, additional roentgenograms of the skull were made. The cystic
cavity was still well outlined but there was no air in the ventricular system. Thisuggested that the two did not communicate.

Subsequent Course. Following drainage of this cyst the patient’s condition was greatly improved and she considered herself well until September 1936, when convulsions, headaches, a right homonymous hemianopsia, and loss of memory recurred.

On Nov. 12, 1936, a needle was inserted through the old burr hole on the left side and 130 cc. of yellow fluid were removed from the cystic cavity, and 120 cc. of air were injected. Roentgenograms again demonstrated the cystic cavity which had not changed during the more than 4 years that had elapsed (Fig. 3). There was no air in the ventricular system and it seemed very likely that the cystic cavity did not communicate with the ventricular system or the subarachnoid space. The cystic fluid contained 96.2 mg. per cent of protein. However, the patient became worse after this procedure, and aphasia, severe headaches, and stupor developed. The cyst was again evacuated. The advisability of making a craniotomy and establishing a communication between the cyst and the lateral ventricle was considered. However, as she promptly recovered this was not necessary. When seen in January 1937, her speech was somewhat slow and the defect in the visual field was still present, but there was no definite aphasia and she had had no convulsions.

Her condition remained satisfactory until Mar. 17, 1944, when convulsions, visual hallucinations, and headaches appeared. Then she was admitted to The Chicago Memorial Hospital on Mar. 24, 1944.

Neurological examinations again revealed a right hemiparesis and a defect in the right homonymous visual fields.
On Mar. 25, 1944, a needle was inserted through the burr hole in the left occipital region. The cystic fluid was under a pressure of 150 mm. of water. About 25 cc. of fluid were removed and all of her complaints disappeared in a few days, although the abnormal neurological findings persisted.

The patient returned in October 1949 with complaints of numbness of the right hand, generalized convulsions, and squinting of the right eye, and again in October 1952 because of headaches. Examination on both occasions disclosed a minimal right hemiparesis, a hemihypesthesia, and a right homonymous hemianopsia. Roentgenograms of the skull revealed nothing new. The cyst was not tapped. She was reassured and soon resumed her full activities. The patient was considered well when last seen on Nov. 26, 1954, except for the same minimal right hemiparesis and hemihypesthesia and the defect in the visual fields.

Comment. It seems likely that the lesion in this case has existed since her childhood. The etiology of the cyst is unknown. Neither is it clear why simple tapping of this large cyst which does not communicate with either the ventricle or subarachnoid space has sufficed to keep this woman comfortable and nearly symptom-free for over 24 years. We have always been ready to establish a communication with the ventricular system but that has never been necessary.
Except for the location of the cystic cavity in the frontal region the following case is remarkably similar. Here, however, simple evacuation of the cyst did not suffice but establishing a communication with the ventricular system has provided relief which is apparently permanent.

Case 2. C.H., a 26-year-old housewife, was admitted to the neurosurgical service at The Chicago Memorial Hospital on Apr. 5, 1942, because of generalized clonic convulsions.

About 1 year previously, during her sixth month of pregnancy, there suddenly developed several major and minor seizures without any evidence of toxemia, followed by the premature birth of the child, who died after 1 day. Subsequently the patient complained of diplopia, headaches, and transient spells of blindness. Two weeks before admission, the headaches grew more severe and the patient had nausea and vomiting. Examination in another institution revealed papilloedema and an enlarged sella turcica, and the patient was started on roentgen-ray treatment. She grew worse, however, and on Apr. 5, 1942 she suffered from three generalized convulsions. She was then transferred to The Chicago Memorial Hospital.

Examination. On admission, she was semicomatose. Papilloedema was present bilaterally. There were retinal hemorrhages and some secondary optic atrophy. There was a left lower facial weakness. Roentgenograms of the skull revealed decalcification of the dorsum sellae and thinning of the floor of the sella turcica.

Operation. On Apr. 6, 1942, ventriculography was performed. In spite of repeated attempts, the right lateral ventricle was not entered. The left lateral ventricle was entered with considerable difficulty and approximately 10 cc. of fluid were removed. No more could be obtained. Ventriculograms revealed air only in the left lateral ventricle. Its anterior horn was displaced far to the left. A very large infiltrating glioma of the right frontal lobe was suspected.

The patient was returned to the operating room but because of her poor condition it was thought wise to be content with a decompression, and not to attempt to expose the tumor.

Through a linear incision a large bony defect was made in the right temporal region. The dura mater was very tense. A blunt cannula was inserted into the cerebral substance. When the needle was passed forward a large cystic cavity was entered. About 110 cc. of pinkish fluid under great pressure escaped. Microscopic examination of the fluid revealed a moderate number of very crenated red blood cells. When the fluid was centrifuged, the supernatant fluid was faintly yellow. However, it had the consistency of cerebrospinal fluid. The cystic fluid contained 38 mg. of protein per 100 cc. in contrast with the ventricular fluid which contained 26 mg. per cent. After the fluid had been removed, 20 cc. of air were injected into the cystic cavity and additional roentgenograms were made. The cortex of the temporal lobe appeared normal. The roentgenograms revealed a large cystic cavity in the posterior part of the right frontal lobe (Figs. 4 and 5). There was no evidence that the cyst communicated with the ventricular system, and it had every appearance of lying within the cerebral substance.

Because of our experience with I.B. (Case 1) it was hoped that mere evacuation of the cyst would suffice. Following this operation, the patient became more alert and had no headaches. But about once every 3 days it was necessary to aspirate 20 to 40 cc. of bloody fluid from the cyst, and it soon became obvious that something further would have to be done.
2nd Operation. On Apr. 15, 1942, an osteoplastic flap was reflected in the right frontal region. In the upper part of the exposed cortex the wall of the cyst was thin and transparent. An opening was made through this thin cortex into the cavity. The cyst was cone-shaped and extended into the white matter of the frontal lobe. There was no evidence of any tumor or vascular abnormality anywhere in the cyst or its wall. A blunt cannula was inserted through the floor of the cyst into the anterior horn of the lateral ventricle. Following down this needle-track tissue was resected, making an opening 1.5 cm. in diameter connecting the cystic cavity and the lateral ventricle.

Figs. 4 and 5. Case 2. (Left) Large cystic cavity lying within the cerebral substance. (Right) Lateral view showing the large cystic cavity in the posterior part of the right frontal lobe. There is no communication with the ventricular system or subarachnoid space.

Postoperative Course. The patient made a good recovery. At the time of discharge (19 days postoperative), she had a slight left hemiparesis, slight diminution of vision, and minimal papilloedema. These soon subsided completely.

The patient was seen at intervals and considered well until over 11 years later. On Dec. 31, 1953, she had a generalized convulsive seizure. Neurological findings were normal. Roentgenograms of the skull revealed only the old bone flap and a parietotemporal decompression. An electroencephalogram showed a spike seizure focus in the right parietal area spreading to the right temporal and left parietal areas as well as minimal slowing in the right temporal area. She was then placed on anticonvulsant medication and has remained well ever since except for an occasional minor seizure.

Case 3.* M.H., a 34-year-old housewife, was admitted to the University of Chicago Clinics on Oct. 16, 1936, complaining of severe headaches of intermittent nature and of blurring of vision.

The patient was perfectly well until August 1935, when occipital headaches developed. These headaches were transitory and not too severe. About the middle of June 1936, 4 months prior to admission, the headaches suddenly became much

* This case was briefly reported by Bucy previously.
worse and almost continuous. In September 1936, she first noticed blurring of vision. In an effort to alleviate the condition, the patient had her teeth removed, her nasal septum extirpated, and had bought glasses, all to no avail.

The family history revealed that the patient's sister had had severe headaches, which began at the age of 20 years and occurred every 3 to 4 weeks, with occasional nausea, vomiting, and blurring of vision. At the age of 35 years, following a "stroke" and paralysis of the right side, she died.

Examination. On admission, positive neurological findings were bilateral papilledema of 3D., bilaterally positive Babinski sign, nystagmus on looking to the left, and adiadochokinesis in both upper extremities. The spinal fluid was under a pressure of 420 mm. of water.

Ventriculography on Oct. 20, 1936 disclosed symmetrically dilated lateral and third ventricles. A cerebellar tumor was suspected.

Operation. A suboccipital craniotomy was made on Oct. 22, 1936, and a cyst was found occupying the position of the cerebellar vermis (Fig. 6). The cyst con-

![Fig. 6. Case 3. Illustration of the appearance and location of the cerebellar cyst seen at operation. It was entirely separate from the fourth ventricle and the cisterna magna.](image)
tained 45 to 60 cc. of clear, colorless fluid similar to cerebrospinal fluid. The cyst did not communicate with the fourth ventricle or the subarachnoid space. The cyst was evacuated and its posterior wall was removed.

Microscopic Examination. The wall of the cyst was composed of a fibrous membrane considerably thicker than the normal arachnoid membrane and at one point there was a small tuft of choroid plexus with papillae smaller than those seen in a normal choroid plexus (Fig. 7). There seemed little doubt but that we were dealing here with a congenital anomaly and that the cyst had been filled by the secretion of this small tuft of choroid plexus.

Postoperative Course. All symptoms were completely relieved and she made a complete recovery and remained well. When last seen on Dec. 20, 1955, she had no neurological symptoms and was leading a normal life.
Comment. Kahn, et al.\(^8\) reported an almost identical case in a 62-year-old man. Trowbridge and French\(^9\) reported a similar case except that no tuft of choroid plexus was found. They also referred to similar cases reported by Craig,\(^5\) Kaplan,\(^9\) and Thompson.\(^19\) They too concluded that these resulted from congenital anomalies, and were inclined to discount inflammation (Craig) and trauma (Thompson) as etiological factors. Our case raises one further point for speculation. In view of the history of our patient’s sister is it not also possible that such cysts may be familial or hereditary?

DISCUSSION

These 3 patients presented symptoms simulating those of tumors of the brain. It is unlikely that such cases will ever be correctly recognized except on ventriculography (when the cystic cavity is entered directly), at operation, or at autopsy. The results of treatment in these cases have been most gratifying and it seems likely that such will usually prove to be the case. Surgical intervention certainly will usually be required in such cases. It is not to be expected that simple evacuation will often prove to be adequate treatment as it was in our Case 1. In most instances it will probably be necessary either to create a communication between the cystic cavity and the ventricular system, as we did in Case 2, or between the cystic cavity and the subarachnoid space (cisterna magna) as we did in Case 3. Others have advocated the complete removal of the cyst and its wall by making a lobectomy.\(^6\) Such may be necessary occasionally, particularly when the cyst is
associated with extensive scarring of the neighboring brain and convulsions are the principal symptom. However, in most instances such treatment will not only be unnecessary but definitely undesirable. In addition to Case 2 reported here, Drew and Grant\(^6\) and Miller\(^3\) have reported other cases in which good results have followed the making of a communication between the cystic cavity and the ventricular system. Their patients were all followed for about 8 years.

Cases similar to our Cases 1 and 2, in which symptoms simulating those of intracranial tumor were associated with benign non-neoplastic intracerebral cysts, have been reported by Love and Groff,

Drew and Grant,

Pendergrass and Perryman,

Drew and Grant,\(^6\) and Miller.\(^3\) However, no one has been able to elicit any definite evidence that explains the development of increased intracranial pressure in these cases. In our Case 1, the patient suffered from increasing headaches but there were no objective signs of intracranial hypertension. In Case 2, however, the intracranial pressure was seriously increased. In this instance there was evidence of hemorrhage into the cyst. The fluid was pink and contained many crenated erythrocytes. When the fluid was centrifuged it was somewhat yellow. In Case 1 the fluid was quite xanthochromic when it was aspirated some 4 years after the first operation. Both of these developments indicate that hemorrhage can occur into these cysts and that such a development may be responsible for an acute aggravation of symptoms and the appearance of signs of increased intracranial pressure.

Intracerebral cysts of this type must be differentiated from parasitic cysts and neoplastic cysts. The only parasitic cyst with which these benign intracerebral cysts are likely to be confused are the hydatid or Echinococcus cysts. Fortunately such parasitic cysts are so rare in the United States that the differentiation will seldom have to be made. In any instance, however, simple inspection will serve to distinguish between them. The hydatid cyst has a definite membrane of its own,\(^5\) whereas there is no distinct membrane with the cysts described here. In cases of doubt, however, the surgeon should never attempt to make the differentiation by aspirating the contents of the cyst. The hydatid cysts contain numerous hydatids which are apt to be scattered if the cyst is punctured, giving rise to many daughter cysts in other parts of the brain. Differentiation from neoplastic cysts may not be possible until the cyst is actually exposed. However, the fluid within neoplastic cysts is usually distinctly xanthochromic and has a much higher protein content than was found in the cysts reported here.

Two other points here attract one’s interest. In both Cases 1 and 2 the symptoms appear to have been either precipitated or aggravated by pregnancy. Why this is true is not apparent, but similar observations have been made regarding the symptoms of intracranial tumors.\(^5,10\)

In Case 1 convulsions occurred at intervals of many years, although there have been none now since 1949. In Case 2 convulsions also occurred prior to the operation. Following the operation these ceased and did not recur until over 11 years later. These cases indicate the difficulty in assessing the sur-
tical treatment of epilepsy and in concluding that any particular form of surgical intervention has abolished convulsive seizures. The observations are also in keeping with those of Foerster and Penfield who reported that post-traumatic epilepsy may be delayed for as long as 14 years after the injury.

**Intracerebellar Cysts.** There appears to be no group of symptoms typical of a benign cyst of the cerebellum. The symptoms are merely those of increased intracranial pressure with mild evidence of cerebellar dysfunction. It is only natural, therefore, that they will usually be mistaken for an intracereellar neoplasm. In most instances the localizing diagnosis will have to be made by ventriculography. At operation the surgeon must distinguish these cerebellar cysts from instances of atresia of the foramina of Magendie and Luschka. As Taggart and Walker have shown, such malformation of these foramina results in an obstructive hydrocephalus with a tremendous dilatation of the fourth ventricle. The dilated fourth ventricle often almost completely fills the posterior fossa, severely compressing the cerebellar hemispheres into the lateral angles of the fossa. Symptoms of atresia of the foramina of Magendie and Luschka usually appear early in childhood, whereas these benign cerebellar cysts give rise to symptoms in adult life. In the case reported by Kahn and his associates the patient was 62 years of age. Furthermore, the results of surgical therapy have been poor in patients with atresia of the foramina of Magendie and Luschka whereas it is to be expected that the results will be much better with such benign cysts of the cerebellum as we have reported here.

**SUMMARY**

Three cases of benign cysts of the brain are reported. Two were in the cerebral hemispheres and 1 was in the cerebellum. All 3 patients presented themselves with serious signs and symptoms of an intracranial space-occupying lesion with signs of increased intracranial pressure and of focal lesions of the brain. All 3 cysts were treated surgically. One was simply aspirated on several occasions. One of the others was opened into the lateral ventricle, while the posterior wall of the cerebellar cyst was removed so that it communicated with the cisterna magna. All 3 patients recovered promptly and have been well for many years.

**REFERENCES**