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 $3\times2\times4$ 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. 1 A). There was mild spastic 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. 1 B). 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.
Case 3. Unit No. 803803. J.C., a 7-year-old boy, 8 months prior to admission had been struck by a car and rendered unconscious for 3 days. On regaining consciousness he had shown a left hemiparesis which cleared over a period of several weeks.

Neurological findings on admission were left-sided hyperreflexia and left Babinski sign. Roentgenograms of the skull made 4 months after injury were interpreted as showing erosion of bone at the site of an old fracture, probably by a post-traumatic leptomeningeal cyst in the right parietal region.

A bone flap turned over this area disclosed several small depressed fractures overlying a dural defect 2×5 cm. in size. The dura mater was adherent to the cortex at the margins of this defect and the center of the region was made up of scarred cortex and numerous small multiloculated cysts. The cysts were opened and the dural defect was repaired with a layer of fibrin film.

The postoperative course was uneventful. The patient is now 13 years old and is normal in every respect except for a mild left hyperreflexia.

Case 4. Unit No. 855062. L.A., a 5-year-old girl, was admitted on May 6, 1947, with the chief complaint of convulsive attacks. At the age of 2½ years the child had fallen three stories, striking her head on a concrete surface. She was not unconscious but became blind for several weeks and some right-sided weakness developed. She recovered her vision and was left with only minimal right-sided clumsiness. She also had occasional unilateral convulsive attacks without loss of consciousness.

On admission she was bright and alert, with only minimal right hyperreflexia. Roentgenograms of the skull were interpreted as showing an area of bone erosion with scalloped margins in the line of an old left parietal fracture (Fig. 2 A). Roentgenograms made after replacement of fluid with oxygen through the skull defect revealed the presence of a multiloculated arachnoidal cyst (Fig. 2 B, C).

At operation these cysts were uncapped, the dural-arachnoidal scar was excised and the wound was closed in a water-tight fashion.
Postoperatively the child did well. She developed normally in every respect and was kept on anticonvulsant therapy for 3 years after operation and then this medication was discontinued. She has been free of convulsions for 18 months without medication.

*Case 5.* Unit No. 946693. J.B., an 8-month-old infant, was admitted to Babies
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Hospital on July 21, 1949 because the parents noticed an enlarging skull defect in the right parietal region.

At the age of 4 months this infant had been dropped from its mother's arms, striking its head on a cement floor. There were no neurological symptoms at the time but a wide linear fracture in the posterior parietal region was found.

Four months later new roentgenograms showed that the fracture site had enlarged to a 7×2 cm. bone defect with sclerotic margins.

At operation a 3×4×3.5 cm. cyst was found under the bone defect. There was no connection between this cyst and the ventricle, and the dura mater was entirely absent over its apex. Several operations were needed in order to achieve a watertight closure of the dura mater; the bone defect was covered with a tantalum plate.

The child has developed normally during the 24-month period of postoperative observation.

Case 6. Unit No. 019241. F.A., an 8-year-old boy, was admitted to Babies Hospital on Oct. 28, 1950, for control of his diabetes of 10 months' duration. The past history revealed that at the age of 15 months he had fallen about 10 feet, striking his head on a stone surface. He had been unconscious for a matter of hours; a severe right hemiparesis was present immediately after the injury and had persisted to a lesser degree up to the time of admission.

Neurological findings were a moderately severe right hemiparesis and a decrease in the circumference of the right limbs.

Plain films of the skull were interpreted as showing an elongated defect, 0.7 by 3.5 cm., and several other smaller defects in the left parietal region (Fig. 3 A). Pneumoencephalography revealed a slightly dilated left lateral ventricle without porencephaly. Five days later, after the gas had been absorbed, oxygen was injected directly through the defect and this revealed a multiloculated leptomeningeal cyst (Fig. 3 B, C, D). No gas was seen to enter the ventricles or the free subarachnoid space at this time.

At operation the bone was found to be eroded in several places and adherent to the underlying dural-arachnoid scar. There was also revealed a 3×4 cm. multiloculated leptomeningeal cyst extending as deep as 2 cm. into the underlying scarred brain substance. This cyst was unapped, the dural defect was covered with a layer of gelfoam, and the remainder of the wound was closed in the usual manner.

In the 12 months since operation there appears to have been some improvement in the hemiparesis. This is difficult to interpret, however, as the patient has been receiving physiotherapy.

Case 7. Unit No. 068950. P.C., a 4½-year-old girl, was admitted on Nov. 10, 1951, because of severe left hemiparesis and bulging in the right parietal region which had become progressively more prominent. At the age of 2 she had fallen 15 feet, striking her head on a stone walk. She had been semicomatose for about 6 weeks.

Neurological findings on admission were a spastic left hemiparesis with hypoplasia of the left extremities, and a 6×8 cm., slightly bulging skull defect in the right parietal region. Roentgenograms made at the time of the injury were reviewed as were several subsequent films. These series of films demonstrated several fractures of the skull which had united spontaneously while one in the right parietal region had undergone progressive enlargement until it formed a huge defect (Fig. 4, A, B, C). Pneumoencephalography revealed porencephaly under the skull defect.
At operation shredded-appearing dura mater was reflected to reveal a large area of multiloculated cysts. These were uncapped and the entire area was connected surgically to an underlying dilated ventricle through a layer of scarred white matter 2 to 2.5 cm. thick. The dura mater was repaired with Cargile membrane and the bone defect was covered with a tantalum plate.

The postoperative course was complicated by a collection of cerebrospinal fluid under the skin flap necessitating a reclosure of the dural defect. The patient has not been followed long enough to evaluate her course.

ANALYSIS OF CASES

Time of Injury and Development of Bone Erosion. The trauma occurred in childhood. The oldest patient of this series at the time of injury was 6 years and the average age was 2 years and 7 months. The time elapsed
between the injury and the discovery of bone erosion by x-ray examination varied from 4 months to 14 years, but in most cases it was less than 3 years.

**Type of Injury.** All patients had received a sharp blow to the head having the mechanical characteristics necessary to produce a skull fracture. Five of the children had been dropped or had fallen on their heads on cement pavements for distances varying from 4 to 30 feet. The other 2 children had been struck by rapidly moving vehicles.

**Severity of Brain Damage and Leptomeningeal Cyst Formation.** No relationship could be established between the development of the cyst and the severity of the brain damage. Two of the patients had rather severe neurological deficit (spastic hemiplegia and hemiatrophy) and 3 had had preoperative seizures. In 1 patient there was gross mental retardation.

**Radiologic Findings.** The roentgenograms in this condition are so characteristic that a correct diagnosis usually has been made on the basis of plain films. The changes were originally described by Dyke¹ and by Schwartz².³

The films disclose an irregular defect in the bone, usually with scalloped margins and some degree of sclerosis of the adjacent bone; both inner and outer tables are involved, the inner table to a greater extent. In some instances the old fracture line may be clearly seen at the ends of the skull defect. In all of the 7 cases the lesion is situated in the parietal region and in 6 of them in the posterior half of the parietal bone close to the superior portion (Fig. 2 A, 3 A).

A preoperative diagnosis can be established by injecting oxygen or air through the skull defect after the withdrawal of fluid from the cyst. In the cases in which this was done a multiloculated irregular cystic space was shown and the injected gas did not enter the free subarachnoid space or the ventricles (Fig. 2 B, C and 3 D).

Pneumoencephalography was performed in 3 cases: In 2 (Cases 2 and 6) the lateral ventricle was moderately dilated on the side of the skull defect without evidence of porencephaly (Fig. 3 B and C). In addition to the cyst the other patient (Case 7) had a dilated ventricle with porencephaly. The question may be raised as to the possible role of the porencephalic dilatation in the production of the bone defect, but porencephaly alone rarely causes pressure erosion in the skull.

In the 2 cases in which we have been able to obtain the films made at the time of the initial injury, the original fracture is wide, measuring as much as 4 mm. in width. On the basis of these 2 observations no definite conclusions can be reached, but they suggest that perhaps in every case in which a fracture appears wide when first seen, the patient should be followed for several years, or until the fracture heals, since it would appear that the wider the original fracture the greater the possibility of dural tear and arachnoid herniation with subsequent formation of leptomeningeal cysts. This applies more specifically to children.

Roentgenologically the differential diagnosis of the condition is relatively
easy: With a clearcut history of skull injury several months or years before, the presence of an elongated skull defect with scalloped margins and irregular sclerosis of the bone involving both tables of the skull and usually situated in the posterior parietal region is almost pathognomonic. The skull defects sometimes seen during healing of a cephalohematoma should not cause confusion. With a leptomeningeal cyst there is no evidence of a shell of subperiosteal new bone as is seen in cephalohematoma.

Operative Findings. In all cases at operation the dura mater was found to be absent in the center of the bone defect and markedly thinned and adherent to the underlying arachnoid about the margins of the lesion. The fluid within the leptomeningeal cyst was a clear colorless liquid grossly resembling cerebrospinal fluid. In one case in which the cyst fluid was analyzed chemically it was found to be identical with cerebrospinal fluid. An active search was made for a communication of the cyst with the underlying ventricle, but none was found in any instance at operation.

DISCUSSION

It is our opinion, based on the factors mentioned above, that the mechanism involved in the production of this syndrome is as follows (Fig. 5):

![Diagram](image)

Fig. 5. Diagram showing our conception of the mechanism of production and the progressive changes leading to the formation of a leptomeningeal cyst and erosion of the skull. (A) The original fracture with tear of the dura mater and herniation of the arachnoid membrane. (B) Widening of the dural aperture and beginning erosion of the skull. (C) The final stage when there is marked erosion of bone from within, with further widening of the dural aperture, arachnoidal adhesions around the margins, and depression of the brain.
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Trauma produces a skull fracture and an underlying dural tear. At the same time there is probably sufficient subarachnoid hemorrhage to hinder the local circulation of cerebrospinal fluid. The arachnoid membrane projects out through the dural tear into the fracture site. This trapped arachnoidal hernia, aided by the normal pulsations of the brain, gradually erodes the edges of the bone and at the same time compresses the underlying cortex. There must be some degree of a ball-valve mechanism at work also, with the cerebrospinal fluid having easier ingress into than egress from the cyst. Arachnoidal adhesions about the margin of the lesion probably also play a part in trapping the fluid locally. It seems probable that the dural tear is the single most important factor in the pathogenesis of these lesions and that without it the fractures would heal as expected (and as indeed other skull fractures healed in these same patients).

Treatment of these lesions consists of excision of the leptomeningeal cyst, repair of the dural aperture and plating the resulting bone defect if necessary. Since this condition is progressive, early diagnosis is important if one is to avoid extensive erosion of bone and dura. The possibility of minimizing the underlying brain damage and lessening the severity of post-traumatic epileptic attacks also constitutes good reason for early therapy. Two of the 3 patients with preoperative convulsions became seizure-free following surgical repair of the lesion. Because diagnosis is the first step in therapy, we feel that roentgen examination of the skull at 3 and 6 months following injury is indicated in children who have sustained skull fracture, particularly if the fracture is in the parietal region.

**SUMMARY**

Seven cases of post-traumatic cerebral leptomeningeal cysts are reported. The bone along the fracture was eroded and the fracture defect was irregularly widened.

Analysis of the factors common to all these cases indicates that rupture of the dura mater and herniation of the arachnoid into the fracture is probably the mechanism by which the lesion is formed.

Because of the importance of early diagnosis, it is advisable to have repeated x-ray examinations of the skull following fracture of the skull in children at intervals of 3 months until the fracture heals.

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**REFERENCES**

