Acta Neurochirurgica 69, 43—51 (1983)

ACTA
NEUROCHIRURGICA

Service de Neurochirurgie B, Centre Hospitalier Régional, Lille, France

Arachnoid Cysts and Head Injury

By

F. Lesoin, P. Dhellemmes, M. Rousseaux, and **M. Jomin**

With 2 Figures

Summary

A number of hypotheses and much discussion surround the subject of intracranial arachnoid cysts (ICACs). Based on the observation of 7 cases of ICACs which were asymptomatic until aggravated by head injuries, the aetiopathological relationship between head injury and ICAC is discussed. Trauma is not the only origin of ICACs, but even a slight head injury is capable of provoking the rapid decompensation of a previously dormant ICAC.

Keywords: Arachnoid cysts; intracranial; post-traumatic; aetiopathology.

Introduction

Intracranial arachnoid cysts have been reported by various authors since they were first described by Cohen [9] in 1935. Nevertheless, they are only rarely seen and their aetiology is still unclear.

Two forms of cysts have been reported in the literature: intracerebral cysts and corticomeningeal cysts. This discussion will be limited to corticomeningeal cysts of the Sylvian fissures and their particular clinical and neuroradiological manifestations, as well as their treatment and evolution.

Based on both the study of 7 cases seen between 1962 and 1982 and a vue of the literature, the aetiopathological relationship between these cysts and head injury is discussed.

Case Reports

Case 1. A 18-month-old child, was referred for neurosurgery on June 9, 1976 by his family physician, who suspected the presence of a frontal lobe mass. There had been no complications during birth; growth and development had thus far proceeded normally. At the age of 11 months, he suffered a number of

falls, and sustained several minor injuries. When hospitalized, he was found to be macrocephalic, with a head circumference of 52.5 cm, or 3.6 standard deviations above the normal. He showed signs of static cerebellar dysfunction, as well as paralysis of the right abducens (VI) nerve. Fundoscopic examination and EEG activity were normal. Standard roentgenograms showed no abnormal intracranial calcifications, and confirmed the macrocephalia, with an emaciation of the vertex of the skull. Air encephalography showed a marked hydrocephalus. The medial

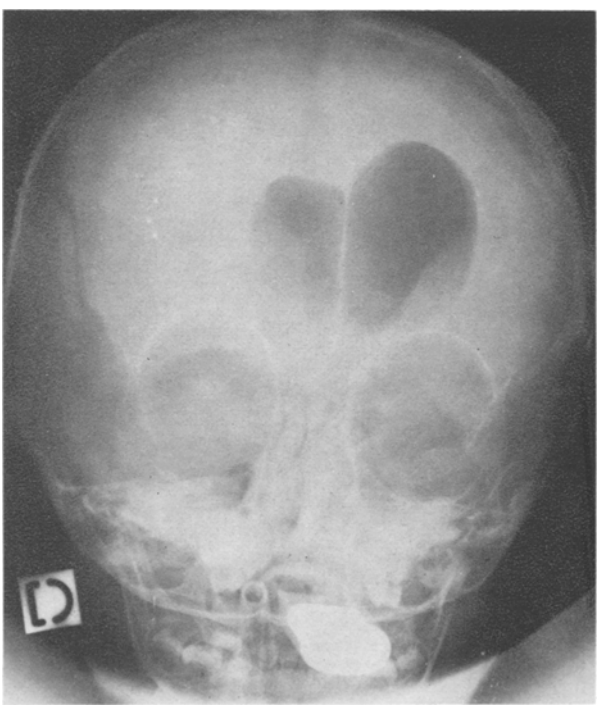

Fig. 1. The encephalography shows the medial structures displaced to the left

structures were displaced to the left, with flattening of the right lateral ventricle (Fig. 1). Right-sided carotid angiography confirmed a 3 cm displacement towards the left side, accompanied by a verticalization of the middle cerebral artery, evoking the presence of an expansive mass in the Sylvian fissure (Fig. 2). The decision was made to operate; a large ICAC was found in the right Sylvian fissure. The optic chiasma was covered by a translucent arachnoid membrane, beneath which could be seen the optic nerves, the chiasma, the internal carotid artery, the middle cerebral artery, the anterior cerebral artery, and the third cranial nerve pair. Microscopic study of this membrane showed it to be made up of innumerable villi whose basal membrane was supported by loose conjunctive tissue which was infiltrated by a number of lymphocytes. Post-operative recuperation was excellent for the first 15 days; on the 15th day, the patient began having difficulty walking, and on June 30, 1976, a Hakim valve was placed to drain the cyst into the peri-

toneal cavity. After this second operation, recovery and recuperation were uneventful and total.

Case 2. A 12-year-old boy was brought to the casulty room and hospitalized on May 16, 1979 following a head injury of the left parieto-occipital region. When he arrived in the Department of Neurosurgery, he was unconscious, with left sided mydriasis, and massive left-sided hemiplegia. He was able to respond

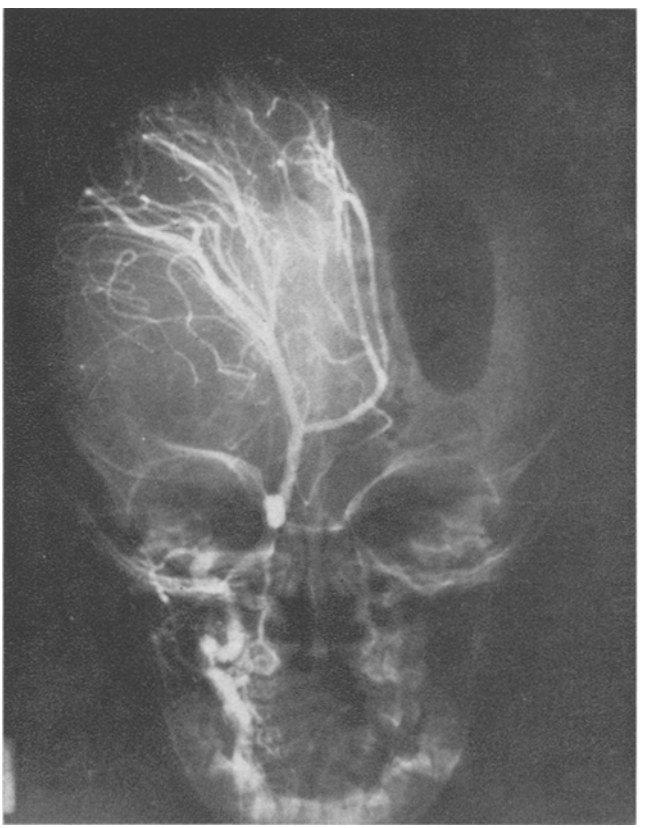

Fig. 2. Verticalization of the middle cerebral artery, evoking the presence of an expansive mass in the Sylvian fissure

to painful stimuli and able to respond to simple commands, but was unable to answer simple questions. Standard roentgenograms showed a left sided parieto-occipital fracture. A left-sided carotid angiogram was done at once, and the middle cerebral artery was seen to be laterally displaced. It was decided to operate immediately. During the operation, a large ICAC was found in the Sylvian fissure. There was extensive damage to the cisterna of the base of the brain, and the middle cerebral artery was exposed. The floor of the cavity was covered with many large veins which drained into the sphenoparietal sinus. Recovery and recuperation were satisfactory during the first 8 post-operative days,

when suddenly the patient's condition worsened. A computerized axial tomographic (CAT) scan was done, and an accumulation of liquid between the brain and the dural sack was found on the left side. The patient was re-operated, and a Hakim valve was placed to drain the excess liquid into the peritoneal cavity. Following the second operation, recovery was uneventful, and recuperation was complete. Seen as an out patient six months later, the neurological examination was normal, as were his EEG and CAT scan. It is interesting to note that there was an exacerbation of his anti-social behaviour which existed before his injury.

Case 3. A 23-year-old man was hospitalized in a state of coma. Three months earlier, he had suffered a head injury and was hospitalized in a state of coma with left-sided mydriase and decerebrate rigidity. He was found to have a subdural haematoma and an ICAC in the Sylvian fissure, and an emergency operation was performed. Recuperation was slow, and he was transferred to a physiotherapy centre where, three months later, he lapsed into a coma. Standard cranial roentgenograms and fundoscopic examination were normal; left-sided carotid angiography showed signs of an expanding mass. The decision to operate was taken, and it was found that the ICAC had reformed, now containing 300 ml of clear liquid. The membrane was excised for the second time, and the internal carotid, the second, third and fourth cranial nerves were dissected free. The circulation of the CSF was still partially blocked, and a hakim valve was put in place. The pathologist's report showed the cyst to be of arachnoid origin, with traces of old bleeding. Recovery and recuperation were uneventful at first until the valve blocked up 5 days after the operation. After re-operating and changing the valve, there were no further problems.

Case 4. A 45-year-old man was hospitalized on March 23, 1981, after injury to the occipital region followed by a temporary loss of consciousness. On arrival at the hospital, his level of consciousness and neurological examination were normal. Standard cranial roentgenograms showed a paramedial occipital fracture, and his EEG showed focal disturbances. For the first 48 hours after his injury, he showed no signs of being unwell, when suddenly he lapsed into a deep coma with left eye mydraisis and massive right-sided hemiplegia. A CT scan was done at once, and a cyst was seen in the region of the Sylvian fissure. It was decided to operate immediately and it was discovered that a cyst containing bloody liquid had ruptured into the subdural space, with contusion and herniation of the temporal lobe. The liquid was drained and the dural sack repaired, but the recovery period was long and stormy. Even after one year, recuperation of the right hemiplegia was only incomplete for his superior limb.

Case 5. A 49-year-old man was the victim of a road accident on January 31, 1979. He sustained a head injury without fracture. Twenty four hours after the accident he was hospitalized in a stupor with a left-sided hemiparesis which especially affected his arm and face. Fundoscopic examination was normal; EEG examination showed right temporal lobe disturbances. Carotid angiography and a CAT scan clearly showed a large ICAC. The patient was operated immediately and a valve was placed to drain the excess liquid into the peritoneal cavity. Recovery was uneventful, and one week after the operation, recuperation was complete. Three years after the operation, the patient is in excellent health.

Case 6. A 27-year-old man was hospitalized on April 12, 1962, showing signs of cranial hypertension. Direct questioning revealed that he had suffered a head injury and scalp wound in early February 1962. Two weeks after the injury, he began suffering from intense bilateral frontal cephalgia, followed a few days later by diplopia. On examination, he was found to have a slight left arm motor

deficit, and bilateral papilloedema was discovered. Diffuse disturbances were seen in his EEG, with the right-side electrogenesis less well organized than the left. Both anterior and lateral views of the carotid angiography showed the left middle cerebral artery to be displaced, in favour of an expanding intra-cranial lesion. The patient was operated on May 16, 1962. A left-side temporo-frontal window was made in the skull, and the tightly stretched dural sack was opened, liberating a large collection of clear liquid. The cyst membrane was thin, translucent and situated between the dura mater and the brain itself without adhering to either structure. The cyst had occupied the anterior half of the temporal fossa, pushing itself into the Sylvian fissure, the space between the brain and internal carotid, and severely compressing the temporal lobe. The carotid bifurcation, the anterior communicating artery, and the optic nerve were visible under the thin membrane, which was removed. The pathologist's report described the membrane as fibrous, richly vascularized, oedematous, with tiny deposits of pigment, attesting to small effusions of blood followed by haemolysis. Recovery was uneventful and recuperation was complete; seen a year after the operation, both his neurological examination and his EEG were normal.

Case 7. A 7-year-old boy was referred to the Department of Neurosurgery on February 12, 1977 by his family physician because of signs of cranial hypertension. His birth, growth and development had thus far been completely normal; his IQ was 120. The history revealed that he had recently fallen down a short flight of stairs, after which be began having headaches and bouts of vomiting. On examination, he was found to have a cerebellar ataxia, and fundoscopic examination revealed bilateral papilloedema. Standard roentgenograms showed separation of his cranial sutures. EEG examination showed overall slowing of the wave patterns, and left-sided carotid angiography showed displacement of the middle cerebral artery. The patient was operated on February 16, 1977, and an ICAC was found in the Sylvian fissure. A valve was placed to drain the excess liquid into the peritoneal cavity. The pathologist described the cyst membrane as richly vascularized endothelial tissue, with a glial cell support. Recovery was uneventful, and recuperation was complete. His post-surgical IQ remained at 120.

Discussion

A. The ICAC

The ICAC can be defined as a benign, intradural, extracerebral lesion filled with CSF, which imposes itself on neighbouring cerebral tissue without changing or destroying it. This working definition merits some discussion:

Although it is histologically a benign lesion which often remains dormant, an episode of intracranial hypertension can awaken it, requiring immediate surgery.

It is always extracerebral, that is, it pushes on brain, but does not infiltrate it.

The cyst is not a completely closed system; it often communicates with the ventricles and the cisterna through very fine canals, which are rarely visible, even during the operation.

The definitive diagnosis of an arachnoid origin of the cysts rests with the pathologist.

ICACs often, but not always, contain CSF. Xanthochromic liquid containing a high protein concentration has been observed, and it has been suggested that this is an indication of poor communication between the cyst and the cisterna.

B. ICAC and Head Injury

The characteristic onset of neurological troubles which announce the presence of an ICAC after a head injury seems to indicate that there is a relationship between the two.

a) Onset of Neurological Symptoms

In 135 cases reported by various authors, we found that 50 cases were associated with head injuries. In 19 of these cases, the symptoms began slowly, some time after the injury, which was often slight (Baumann [2], Bull [7], Chikde [8], Deruty [11], Gruss [14], Lambert [17], Losede [18], Petit-Dutaillis [20], Robinson [22], Tiberin and Torma [24], and our case 1).

In 31 observations, the ICAC began showing itself almost after the injury:

acute manifestation: 14 observations reported (Childe [8], Filhasre [3], Hardmann [15], Lambertz [16], Oliver [19], Pichard [21], Robinson [22], Torma [24] and our cases numbers 2–5);

subacute manifestation; the clinical picture suggests the presence of a subdural haematoma; the ICAC is discovered during the course of the operation. Seventeen such cases have been reported (Baumann [2], Bhandari [4], Bret [5], Brotchi [6], Deruty [11], Lambertz [16] and our case numbers 6 and 7).

b) Pathological Discussion

Some authors (Deruty [11], Oliver [19], Pichard [21], and our cases numbers 3, 6, and 7) report the presence of rich vascularization of the cystic membrane, with or without the presence of pigmentation, which suggests trauma-induced bleeding. The presence of old blood clots or deposits of RBCs in the wall probably suggests the same thing, whereas the importance of the presence of a clear liquid is not yet known. The presence of RBC pigments and of glial scar tissue seem to indicate a relationship between injury and the formation of an ICAC.

C. Aetiopathology: The Role of Head Injury

There is still no agreement concerning the pathogenesis of ICACs. Most authors accept the existance of both primary congenital cysts and secondary cysts.

The definition of ICACs is implied in the problem of pathogenesis. According to Brotchi [6] and Tiberin [23] a head injury with fracture can cause a breach in the dura mater, and create a closed pouch which subsequently fills with liquid. This constitutes a leptomeningeal cyst, which we have excluded from the definition of ICACs.

There are four ways to approach the relationship between head injury and ICACs:

1. In certain cases, head injury can provoke the formation of a cyst. A serious injury can provoke the formation of a cyst either by creating a haematoma, or by necrotic erosion of underlying brain tissue.

A rebound counter-blow mechanism explains the formation of the cyst opposite the anatomical point of injury, and it also eliminates the necessity of an associated fracture. Contusion-attrition of the temporal lobe on the splenium is frequently seen. This favours formation of a pocket in which lysis of the blood clots present produces a hyperosmotic protein-rich environment. It will be remembered that in certain cases RBC pigment deposits were found on the cyst wall membrane, and also that these cysts are found in the same anatomical locations as certain intracranial haematomas. This hypothesis has been supported by the work of Bernard [3], who observed that following wide-spread subcortical birth injury, there was liquification of the necrotic tissue, and subsequent formation of large cysts. Of course, the same reaction is observed after injury of a porencephalic subject, but this is usually limited to birth injuries or injuries of very young children. None of the cases reported here fall into this category.

The previously mentioned intracystic hyperosmolarity accounts for the filling of the ICAC, which eventually results in compression of the neighbouring brain tissue. Banna has suggested that certain cysts are either the result of disintegration of an old subdural haematoma, or a subdural accumulation of CSF following a tearing injury of the arachnoid membrane.

2. Injury may modify the anatomical structure of the already existing cyst, thereby favouring its growth and subsequent clinical expression. Often however, the injury is too recent to account for the size of the cyst (Torma [24]). Bret [5] and Deruty [11] noted that injury may cause an intracystic haematoma, due to bleeding from the membrane wall and subsequent fibrosis, giving rise to a xanthochromic liquid, and sudden clinical expression. This same mechanism could give rise to an increase in the intracystic oncotic pressure, which

would allow the cyst to slowly expand and explain late clinical symptoms.

Finally, Weinberg and Flom [25], discuss the traumatic rupture of the external wall of the cyst, which leads to subdural accumulation of cystic liquid.

3. Elsewhere, ICACs have been discovered quite by chance after an injury which was not accompanied by signs of an expanding intracranial mass. Torma [24], Brotchi [16], and Baumann [2], have all reported cases of totally asymptomatic ICACs which they discovered during routine post-cranial trauma examinations. Bret reported a case in which the intracranial hypertension was the combined result of a subdural haematoma and an ICAC.

4. Certain reports [1, 6, 12] have spoken of functional neurological manifestations related to both the cyst and the head injury. Sequella seem to be proportional to the gravity of the head injury, and to the general state of the patient when he is operated. The simple ICAC which requires only a drainage valve seems to be followed by fewer complications than the ICAC which has bled and requires a longer and more complicated operation.

References

1. Anderson, P. M., Landing, B. H., Cerebral arachnoid cysts in infants. J. Pediat. *69* (1966), 88—99.
2. Baumann, F., Kystes arachnoïdiens intracrâniens chez l'enfant. Thèse. Paris. 1970.
3. Bernard, R., Vigouroux, R., Mariani, R., Pinsard, N., Pierron, H., Arachnoïdite kystique avec trouble de la vascularisation corticale. Pédiatrie *20* (1965), 979—982.
4. Bhandari, Y. S., Non communicating supra-tentorial subarachnoid cysts. J. Neurol. Neurosurg. Psychiat. *35* (1972), 763—768.
5. Bret, P., Deruty, R., Laupras, A., Kystes bénins du cerveau révélé à l'occasion d'un traumatisme crânien. A propos de 10 observations. Lyon Med. *232* (1974), 18, 523—525.
6. Brotchi, J., Petrov, V., Reznik, M., Kystes arachnoïdiens révélés par un traumatisme crânien. Acta Neurol. Belg. *74* (1974), 107—111.
7. Bull, J. W. M., Diagnosis of chronic subdural hematoma in infant and adolescent. Brit. J. Rad. *22* (1949), 68—71.
8. Childe, A. E., Localized thinning and enlargement of the cranium with special reference to middle fossa. Amer. J. Roentgenol. *70* (1953), 1—22
9. Cohen, J., Chronic subdural accumulations of cerebrospinal fluid after cranial trauma. Arch. Neurol. Psychiat. *18* (1927), 709—711.
10. Craig, G. F., Chronic cystic arachnoiditis. Amer. J. Surg. *17* (1932), 384—386.
11. Deruty, R., Les kystes bénins du cerveau (à propos de 25 observations). Thèse. Lyon. 1970.
12. Drew, J. H., Grant, F., Benign cysts of the brain: an analysis with comparison of results of operative and non-operative treatment in thirty cases. J. Neurosurg. *5* (1948), 107—111.

13. Filhastre, H., Kystes liquidiens sus-tentoriels observés chez l'enfant à l'exclusion des kystes tumoraux et parasitaires. Thèse. Marseille. 1975.
14. Gruss, P., Aver-Doinet, G., Zur Genese der temporalen Arachnoïdcysten. Neuropaediatrie *5* (1974), 175—178.
15. Hardmann, J., Asymetry of skull in relation to subdural collection of fluid. Brit. J. Radiol. *12* (1939), 455—458.
16. Lambert, M. P., Contribution à l'étude des kystes arachnoïdiens de la vallée sylvienne. Thèse. Rennes. 1969.
17. Lambertz, J., Rougerie, J., Job, J. C., Rossier, A., Kyste arachnoïdien de la vallée sylvienne. Pédiatrie *25* (1970), 529—532.
18. Loseke, N., Les kyktes arachnoïdiens intracrâniens sus tentoriels. Acta Neuro. Belg. *78* (1978), 148—152.
19. Olivier, L. C., Primary arachnoid cysts: report of two cases. Brit. Med. J. *1* (1958), 1147—1153.
20. Petit-Dutaillis, D., Chavany, J. A., Caron, J. P., Hydromes intracrâniens chroniques. Presse Med. *59* (1951), 708—709.
21. Pichard, J. C., Les kystes arachnoïdiens de la vallée sylvienne. Thèse. Paris. 1968.
22. Robinson, R. G., Intracranial collections of fluid with local bulbing of the skull. J. Neurosurg. *12* (1955), 345—347.
23. Tiberin, P., Gruskiewicz, J., Chronic arachnoidal cysts of middle cranial fossa and their relation to trauma. J. Neurol. Neurosurg. Psychiat. *24* (1961), 86—90.
24. Törmä, T., Heiskanen, O., Chronic subarachnoidal cysts in the middle crania fossa. Acta Neurol. Scand. *38* (1962), 166—169.
25. Weinberg, P. E., Flom, R. A., Intracranial subarachnoid cyst. Radiology *106*, (73), 329—331.

Authors' address: F. Lesoin, M.D., Service de Neurochirurgie B, Lille — C. H. U., F-59037, France.