

Supratentorial Arachnoid Cysts: Clinical and Therapeutic Remarks on 46 Cases

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Summary

The authors report 46 cases of primary supratentorial arachnoid cysts. Thirty-two patients presented with symptomatic arachnoid cysts, which act as space-occupying lesions and were surgically treated by fenestration. 14 patients presented with a scarsity of symptoms; for this reason they were kept under control by periodical neuroradiological examination. After a long follow-up period (median 11 years), 97% of the operated patients presented neurological improvement and a decrease in the size of the cyst; none of the patients who were not operated on showed any variation in either neurological status or size of the cyst. The authors discuss their findings in the light of the published papers.

Keywords: Arachnoid cyst; fenestration; surgical treatment; outcome.

Introduction

Intracranial arachnoid cysts are congenital lesions most probably arising from anomalous splitting and duplication during the development of the endomeninx²⁵. The congenital nature of these lesions is suggested by their prevalence in the paediatric age group and by the absence of inflammatory or traumatic antecedents^{2,4,9,13,14}. Although there is some discussion over the therapeutic protocol to be adopted, surgical treatment of these lesions usually achieved good longterm results, especially as far as symptoms and signs are concerned^{1-4, 8-14, 16-32}.

This study describes the clinical picture, radiological findings, and follow-up results in 46 cases of primary supratentorial subarachnoid cysts operated on between 1970–1990 and compares them with those published.

Patients, Methods, and Results

29 patients were males and 17 females (M:F ratio; 1:1.7), with an average age of 24 years (range 5-76 years). The decade of life

most frequently affected was the second (28% of cases). The duration of the clinical history averaged 13 months (range 1–24 months). 32 patients (70%) showed progressive symptoms and signs while in 14 (30%) these were both either absent or mild and stable.

The most frequent symptom was headache in 46 cases (70%) and seizures (60%) (Fig. 1).

In the 18 patients treated prior to 1977, neuroradiological diagnosis was made on the basis of plain skull films, pneumoencephalography and cerebral angiography; in the 28 treated subsequently, CT scan, CT-cisternography and MRI were also employed (Fig. 2a, 2b).

Findings

Plain skull films showed an assymetric cranial vault in 8 cases (17%) and thinning of the bone over the lesion in 9 (19%). Pneumoencephalography revealed indirect signs of a space-occupying lesion moderately shifting the ventricular system. Cerebral angiography (4 cases) always showed an avascular mass displacing the contiguous vessels. CT and CT-cisternography showed a cystic lesion, whose density was similar to that of CSF, and showed contrast enhancement. Of

Symptoms	
Intracranial hypertension	65%
(headache, vomiting, papilloedema, macrocephaly)	
Focal signs	30%
(paresis, sexual and visual disturbances, aphasia)	
Seizures	46%
Absent	9%
Progressive 70%	Stable 30%

Fig. 1. Clinical symptoms of supratentorial arachnoid cysts



Fig. 2. The axial MRI (a) showed a temporal arachnoid cyst; the coronal MRI (b) provides detail of the relationship of the lesion to the adjacent structures

the 28 cases, the cyst compressed the cerebral parenchyma in 22 (78%), altered the cranial vault in 4 (14%) and presented a comunication with the subarachnoid spaces in 6 (21%). MRI, done in 6 cases, documented a cystic lesion of CSF-like density, extraparenchymal with well-defined borders, which compressed the surrounding parenchyma.

The cysts were in the following locations: left hemisphere in 26 cases (57%), the right hempisphere in 16 (35%), median in 3 (6%), bilateral in 1 (2%). Their localization was Sylvian in 37 cases (80%), frontal in 3 (7%), suprasellar in 3 (7%), parietal in 2 (4%) and occipital in 1 (2%).

Management

Thirty-two patients with progressive neurological symptoms and signs and without significant ventricular dilatation were surgically treated. This consisted of fenestration and partial resection of the cyst wall, connecting the cystic cavity with the subarachnoid space. Three of the 32 patients (9%) presented postoperative complications: one presented an intraparenchymal haematoma which required surgical evacuation and another 2 presented and a small subdural blood layer visualized on CT, but did not, however, need surgical treatment.

The 14 patients with absent or modest symptoms and with mild or absent neurological signs were not operated on but checked periodically by neuroradiological investigation.

Outcome

In the 32 surgical cases, average follow-up was 11 years (median 13 years; range 10-18 years). Pre-operative symptomatology regressed in 32 patients, totally in 24 (75%). The patient re-operated on for intraparenchymal haematoma presented mild deficits related to this pathology while there was a regression of those due to the arachnoid cyst. Postoperative CT controls showed re-expansion of the cerebral parenchyma in 20 of 22 cases initially assessed by CT: in the remaining 10 cases operated on in pre-CT era neuroradiological follow-up (CT scan in 6 cases, MRI in 4) did not reveal signs of parenchymal compression. The average follow-up in the 14 non-surgical cases was 9 years (median 10 years; range 7-12); and none of these presented any worsening of the symptoms present at diagnosis. In 3 cases, follow-up CT scan showed a slight increase in the volume of the cyst without compression of the cerebral parenchyma.

Discussion

Arachnoid cysts account for 1-13% of all brain lesions and are a incidental finding in about 5 out of 1000 patients submitted to autopsy (28). In our series, arachnoid cysts were more frequently observed in the paediatric age group (II decade of life), with a male preference (63% of cases) and were often localized in the left hemisphere (57% of cases) as in other series¹, $_{3, 8, 10, 13}$.

Primary arachnoid cysts are initially asymptomatic due to their small size and/or comunication with the subarachnoid spaces^{5–7, 25, 31}. Subsequently they may became symptomatic for the following reasons:

1) an increase in the osmotic gradient of the liquid contents of the cyst, probably due to intracystic haemorrhage^{30, 31};

2) creation of a value mechanism between the arachnoid cyst and subarachnoid space that causes an increase in its size and the consequent onset of symptoms^{3, 15};

3) the cyst wall secrets liquid^{11, 13} which augments its size, especially when there is a co-existing valve mechanism or when the cyst does not present communication with the subarachnoid space.

The onset of symptoms and signs is due to three principle factors: cortical irritation, compression of the cerebral parenchyma and obstruction of CSF circulation. In our series, 32 cysts were symptomactic and 14 paucisymptomatic; this was probably due to the fact that the cyst still communicated with the subarachnoid spaces.

The most frequent symptoms and signs were those of endocranial hypertension (headache, vomiting, papilloedema and cranial vault modifications); signs of compression and parenchymal irritation (focal deficits, epilepsy) were slightly less frequent. In suprasellar cysts (3 cases) there were also sexual dysfunctions.

The incidence of symptoms in our serires was similar to the other large series^{1-6, 16-31} and varied according to the patient's age, with the exception of headache which was the most frequent symptom and was not influenced by age. Epilepsy mainly affected adults (80% vs 20%) and occurred most frequently in the III decade (75% of cases). Focal signs (paresis, visual field disturbances, sexual dysfunction) affects adults and children with the same frequency. Macrocephaly was a characteristic in children (8 cases).

Neuroradiological investigation in our series always revealed direct/indirect signs typical of arachnoid cyst. As reported by other authors^{10, 32}, we found MRI, although performed in only 6 cases, to be better than CT for distinguishing the arachnoid cyst from the surrounding parenchyma in T 1-weighted images and for assessing parenchymal compression in T 2. MRI provides a useful adjunct to cT in the differential diagnosis with other cystic lesions such as gliomas, epidermoids. CT-cisternography and, up to 1977, pneumoencephalography, proved useful for studying the 14 cases in whom symptoms and neurological signs were not progressive. CT-cisternography (and pneumocephalography to a lesser degree) revealed a communication between the arachnoid cysts and the subarachnoid space: this aspect, typical of arachnoid cysts termed "static" by Clavel⁶, persuaded us no to operate in these 14 paucisymptomatic patients.

Treatment of arachnoid cysts varies according to their size, the severity of neurological deficits and whether there are any associated anomalies. Some authors believe^{7, 12, 14, 18, 29} that the cyst requires treatment when it exerts a mass effect and/or causes an endocranial hypertension syndrome (with or without hydrocephalus).

There are 2 principal methods of treatment, aimed at draining the cyst and preventing fluid accumulation: 1) shunt; 2) fenestration of the cyst wall so that the cystic cavity communicates with the subarachnoid space, or, rarely, total removal of the cyst itself.

Usually, shunting is performed in non-communicating cysts, associated with hydrocephalus^{2, 5, 12, 17, 29}. Not infrequently, it may present complications such as obstruction (10–40%), inability to treat multiloculated cysts, shunt-dependency (40–60%), inability to treat any associated lesions and infection $(10\%)^{3, 16, 22}$. The main advantage of fenestration is that it is not shuntdependent, but it may be complicated by intraparenchymal or subdural haemorrhage (5–25%), recurrence (10–30%), aseptic meningitis (5–10%), inability to treat hydrocephalus (30–100%) and, in rare cases, postoperative mortality (1–10%)^{8, 29}. In our surgically treated cases, the cyst was always fenestrated.

In symptomatic cases, fenestration was always preferred to avoid shunt-dependency and, the septic and obstructive complications associated with it^{2, 4, 12, 14, 19, ²⁴. Moreover, out of the total of 52 cases in those series where shunting was considered the main form of treatment^{12, 17, 29} there were 6 shunt revisions, 2 cases of infection (one of which was fatal) and one case of shunt malfunctioning. As pointed out by Raffel²³, it should be noted that in the three above-mentioned series, the follow-up period was brief. We agree with Sekino²⁷ Hovind¹⁵ and Raffel²³ that fenestration should be preferred in all cases and use of the shunt limited to those cases where it gives unsatisfactory results. In fact, as stated by Raffel²³, successful fenestrations saves the patient the risks of further operation.}

In our seriers, postoperative complications were rare (3 cases:9%). Almost all cases (97%) presented an

chymal haematoma presented neuroloigcal deficit due to the haematoma itself. It is worth noting that in our series postoperative complications were less frequent than in other recent series^{8, 13, 23}. After a long follw-up period (median 10 years), none of the non-surgical cases presented significant variations in either neurological status or the size of the cyst.

On the basis of our experinece, we can conclude that surgical treatment of arachnoid cysts should always be preceded by careful clinical and radiological assessment, taking into consideration the severity of any deficits, their evolution and whether the cyst communcates with the subarachnoid space. The present study indicates that fenestration may, in the first instance, be considered the main option for supratentorial arachnoid cysts, limiting shunt insertion to patients who do not benefit from this type of treatment, to those with hydrocephalus and to elderly patients.

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