Intracystic pressure in patients with temporal arachnoid cysts: a prospective study of preoperative complaints and postoperative outcome

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Background: Arachnoid cysts (AC) can cause a wide spectrum of clinical symptoms. Only a limited number of studies have investigated intracranial pressure in patients with AC. We wished to investigate the relationship between intracystic pressure, preoperative complaints and postoperative symptom relief in adult patients operated on for a unilateral temporal AC.

Material and methods: This was a prospective, population based study involving 38 adult (>18 years) patients (mean age 43 years; range 18–69) with a previously untreated unilateral temporal AC.

Results: For all cyst types, mean pressure was 131 mm H₂O. The main preoperative complaints were headache and dizziness/nausea. By median split, patients were divided into a low pressure (<130 mm H₂O) and a high pressure (>130 mm H₂O) group. Patients with high intracystic pressure had a significantly higher preoperative visual analogue scale (VAS) score (54.7) than that found in patients with low intracystic pressure (39.4). Both pressure groups had a significant fall in VAS score after surgical decompression of the cyst. Intracystic pressure correlated significantly with arterial pCO₂ and mean arterial pressure.

Conclusion: There was a significant correlation between intracystic pressure and the preoperative level of complaints. Mean intracystic pressure in adult patients with unilateral temporal AC seems to lie within the limits of normal intracranial pressure. We therefore hypothesise that factors other than absolute pressure, such as altered compliance and impedance of the brain, may be involved in the pathophysiology of intracranial AC.

Arachnoid cysts (AC) may cause a wide spectrum of clinical symptoms, and several studies have also shown that AC may cause cognitive and metabolic deficits, or affect cerebral perfusion.^{1–11} Is it possible that these symptoms and findings can be explained simply by an increase in intracranial or intracystic pressure, or are other more complex mechanisms at work?

Only a few studies have investigated intracranial pressure (ICP) in patients with AC.^{12–13} Based on a limited number of paediatric patients, these reports indicate normal ICP in most patients with AC. The role of intracystic pressure in the clinical presentation of AC has not been investigated, to our knowledge.

During our operations for intracranial AC,^{14–15} we have often observed that the cyst wall bulges out through the incision when the dura is opened (fig 1), indicating either a generally increased ICP or an elevated intracystic pressure in these patients.

The aim of the present study was to investigate the magnitude of the intracystic pressure, and to determine if this pressure, measured intraoperatively, could be related to the preoperative level of complaints as well as postoperative symptom relief in adults operated on for a unilateral temporal AC.

MATERIAL AND METHODS

Patients

A total of 38 patients were included prospectively (22 males, 16 females; mean age 42.8 years (median 42.7, range 18–69)). Inclusion criteria were age <70 years at the time of operation, and a unilateral previously untreated temporal AC.

Study design/data extraction

The following data were collected: demographic data, Galassi typing,¹⁶ symptoms, intracystic pressure, arterial pCO₂ and

mean arterial pressure during pressure monitoring of patients operated on under full anaesthesia. The preoperative level of symptoms was graded using a visual analogue scale (VAS) on the day before surgery. When given the VAS scale, both before and after operation, patients were instructed to indicate the level of complaint at that very moment.

All patients underwent cerebral CT or MRI prior to surgery and had a postoperative scan within the first 72 hours after operation. Patients were routinely readmitted 3–6 months after surgery for clinical examination, with recording of the postoperative level of their symptoms on a VAS scale, and a CT or MRI scan was performed and the postoperative change in fluid volume was estimated. Patients with any postoperative radiological abnormalities (eg, subdural haematoma or hygroma) were followed until spontaneous remission or until remission following surgical correction of the abnormality.

Operation technique/method of pressure measurement

All patients underwent craniotomy, the majority (n = 33) under general anaesthesia and five under local anaesthesia. General anaesthesia was given as total intravenous anaesthesia with propofol and remifentanyl. Vecuronium bromide (Norcuron) was used as the neuromuscular blocking agent.

Positioning of the patients was standardised, with the operating table flat and the head positioned with the vertex as the centre of the head. The vertex was used as the “zero point” when pressure was measured.

The study was approved by the regional ethics committee.

Abbreviations: AC, arachnoid cysts; ICP, intracranial pressure; NOG, Neuroimaging Outcome Group; VAS, visual analogue scale

Figure 1 Typical intraoperative finding of an arachnoid cyst wall that bulges out through the dural incision in a hyperventilated patient.

Introduction of a mechanoelectric transducer through the very thin and fragile cyst membrane would inevitably have caused leakage and thus deflation of the cyst. We therefore decided to measure the intracystic pressure by direct puncture of the cyst. A burr hole was made immediately posterior to the sphenoid wing in order to gain access to the anterior and most basal aspects of the middle cranial fossa. The dura and underlying cyst was punctured through this very small hole with a 23 G, 25 mm long syringe connected to an Optidynamic spinal fluid manometer (Medioplast AB, Sweden). After pressure equilibration for 3–5 minutes, the pressure, in relation to the centre of the skull, was recorded in mm H₂O. At the same time, an arterial blood-gas sample was collected for simultaneous determination of pCO₂ (kPa). After this procedure, a standard craniotomy with a microsurgical resection and fenestration of the cyst membranes was performed.

Scoring of neuroimaging results

The neuroimaging results of the decompression, measured as the change in cyst volume, were categorised into one of four possible Neuroimaging Outcome Groups (NOG):

- (1) The cyst had disappeared and was no longer visible (NOG 1).
- (2) A fluid volume was still visible where the cyst had been; it was smaller than 50% of the preoperative cyst volume (NOG 2).
- (3) As above, but the residual volume was larger than 50% of the preoperative cyst volume (NOG 3).
- (4) No change in cyst volume (NOG 4).

In a few cases, exact radiological categorisation was difficult to decide. The postoperative radiological result was then always assigned to the less favourable group.

Statistical analysis

All analyses were performed with SPSS 13.0 for Windows. An α -level of 0.05 was regarded as significant in all calculations. Group comparisons were performed using the Student's *t* test or Mann–Whitney *U* test. Contingency tables were analysed with Fisher's exact test or χ^2 statistics. Correlation analyses were performed with linear regression and calculation of Pearson correlation coefficient, or logistic regression and calculation of Spearman rank correlation coefficient.

Table 1 Intracystic pressure in the temporal arachnoid cysts for different Galassi cyst types¹⁰

	Galassi type I (n = 22)	Galassi type II (n = 10)	Galassi type III (n = 6)
Pressure (mm H ₂ O)			
Mean	134	132	119
Low	60	40	80
High	300	180	180

RESULTS

The main preoperative complaints in the 38 patients were headache (35 patients, 92.1%) and dizziness/nausea (14 patients, 36.8%). Fifteen patients (39.5%) presented with more than one complaint that could be related to the cyst. The cyst was located on the left side in the majority of patients (30 patients, 78.9%). Twenty-two patients (57.9%) had a Galassi type I cyst, 10 (26.3%) a type II cyst and 6 (15.8%) a type III cyst.

Intracystic pressure

The pressure measurements for different Galassi types are presented in table 1 and fig 2. For all cyst types, mean pressure was 131 mm H₂O (range 40–300; median 130). Mean pCO₂ was 4.63 kPa (range 3.90–5.40). Patients were divided into a low pressure or a high pressure group by median split. Both groups were matched for sidedness, sex and age. Mean intracystic pressure was 171 and 90.5 mm H₂O in the high and low pressure groups, respectively. Mean arterial pCO₂ and mean arterial pressure were not significantly different between the high pressure and the low pressure groups (table 2).

Correlation between arterial pCO₂ and intracystic pressure

Linear regression analysis with curve estimation (fig 3) of the relationship between arterial pCO₂ and intracystic pressure demonstrated a significant linear correlation (Pearson $R = 0.444$, $p = 0.014$).

Correlation between mean arterial pressure and intracystic pressure

Linear regression analysis with curve estimation (fig 4) of the relationship between mean arterial pressure and intracystic

Figure 2 Box plot of intracystic pressure (mean and range) for Galassi type I–III arachnoid cysts. Open circles with numbers represent cases lying outside the 95% confidence interval.

Table 2 Mean arterial blood pressure and pCO₂ for high and low pressure groups

	Low pressure group	High pressure group
pCO ₂ (kPa)*	4.50	4.77
MAP (mmHg)*	70.9	74.6

*Not significant.

pressure at the time of pressure registration demonstrated a significant linear correlation (Pearson R = 0.336, $p = 0.045$).

Preoperative level of complaint

The 35 patients presenting with headache had a mean VAS score of 50 (range 0–100; SEM 4.1). The 14 patients presenting with dizziness/nausea had a mean VAS score of 32 (range 5–75; SEM 9.0). The difference in VAS score between patients with these presenting complaints was not significant ($p = 0.09$).

Preoperative VAS score and intracystic pressure

In general, patients in the high pressure group had a significantly higher preoperative VAS score (54.7) for the main presenting complaints, assessed by VAS (headache or dizziness) than patients in the low pressure group (39.4) ($p = 0.046$) (fig 5).

Intracystic pressure and postoperative VAS

Both pressure groups had a significant fall in VAS score after surgical decompression of the cyst, a VAS reduction of 38.3 in the high pressure group (from 54.7 to 16.4) ($p < 0.001$) and 23.5 in the low pressure group (from 39.4 to 16.0) ($p = 0.001$). Although there was a trend towards a more pronounced reduction in VAS score in patients with a high intracystic pressure than in patients with a low pressure, this difference failed to reach statistical significance ($p = 0.093$). Postoperatively, there was no difference in VAS score between the groups.

Postoperative volume reduction

The cyst was no longer visible on the postoperative images (NOG 1) in nine patients (23.7%), or reduced to less than 50% of the preoperative volume (NOG 2) in 13 (34.2%). Another 12 patients (31.6%) also had a postoperative volume reduction but with a residual volume larger than 50% of the preoperative volume (NOG 3). Thus in four patients (10.5%) no change in cyst size could be observed after operation (NOG 4).

There was no difference between patients with high and low intracystic pressure in postoperative volume reduction, as evaluated by the NOG scale. Furthermore, postoperative volume did not influence the postoperative level of the preoperative complaint (table 3) as the improvement in patients with a significant reduction in volume (NOG 1 and 2) did not differ from that of patients with a moderate (or none) postoperative volume reduction (NOG 3 and 4).

DISCUSSION

Most AC display some radiological signs of expansion, such as midline shift, compression of the lateral ventricles or an increased volume of neighbouring intracranial compartments. It thus appears reasonable to assume that these phenomena, as well as the symptomatology, are due to a pressure gradient caused by the cyst. To our knowledge, the present study is the first to investigate the magnitude of the intracystic pressure and its possible role in the arachnoid cyst symptomatology.

We found that the mean intracystic pressure for all cyst types was within the limits of what is considered normal ICP,^{17–19}

Figure 3 Correlation with linear regression line between intracystic pressure and arterial pCO₂ at the time of pressure measurement.

although normality for ICP is poorly defined. Our findings contrast to a certain extent with the results of Di Rocco *et al* who, in a limited number of paediatric patients, found an increased ICP only in patients with large cysts, and a normal ICP in children with smaller type I cysts.^{12,13}

Even if the intracystic pressure appeared to be within normal limits, the association we observed between the magnitude of the intracystic pressure and symptom severity indicates that intracystic pressure may be instrumental, at least in part, in causing these symptoms. One possible explanation may be that the pressure inside the cyst is higher than the pressure outside the cyst, and that the symptoms are caused by this pressure gradient. However, we cannot rule out or verify this possibility as we did not simultaneously measure ICP in our patients. We are currently investigating this possibility.

Other more complex mechanisms may also be at work. A normal ICP does not exclude pathology in the ICP dynamics. Factors other than the absolute pressure and pressure gradients may also be involved in the pathophysiology of AC. Several recent studies on normal pressure hydrocephalus have shown

Figure 4 Correlation with linear regression line between intracystic pressure and mean arterial pressure at the time of pressure registration.

Figure 5 Comparison of preoperative level of complaint (measured using a visual analogue scale (VAS)) for the high and low pressure groups. Open circles with numbers represent cases lying outside the 95% confidence interval.

Table 3 Postoperative change in preoperative complaint (measured by VAS) for different levels of postoperative volume reduction

Postoperative cyst volume	Postoperative change in VAS
<50%†	32.8*
>50%‡	28.5*

NOG, Neuroimaging Outcome Group; VAS, visual analogue scale.

†NOG 1 and 2.

‡NOG 3 and 4.

*Not significant.

that it may not be the pressure itself that mediates the symptoms but an additional altered compliance and impedance of the brain, leading to pathological CSF pressure wave amplitudes and reduced vascular pulsatility.^{20–25} A similar mechanism may alter the ICP dynamics in patients with AC. The cyst represents a large closed compartment leading to a reduction in the subarachnoid space and a subsequent possible reduction in intracranial compliance.

At present, there is no model that fully explains the dynamics of AC and the effects these cysts may have on ICP physiology. We are therefore presently investigating whether AC may affect intracranial compliance and other parameters of ICP dynamics.

CONCLUSION

Mean intracystic pressure in adult patients with unilateral temporal AC seems to lie within the limits of a normal ICP. Nevertheless, intracystic pressure appears to be of some significance as it correlated significantly with symptom severity in our patients. We hypothesise that factors other than absolute pressure, such as altered compliance and impedance of the brain, may also be involved in the pathophysiology of intracranial AC.

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