Possible origin of suprasellar arachnoid cysts: neuroimaging and neurosurgical observations in nine cases

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Object. In this study the authors identify and investigate two new classifications of suprasellar arachnoid cysts.

Methods. The authors used computerized tomography cisternography, magnetic resonance (MR) imaging, and neuroendoscopy to investigate nine cases of suprasellar arachnoid cysts. A communicating cyst with early filling and early clearance of a radioopaque tracer was found in seven of nine cases; a communicating cyst with delayed filling and delayed clearance of the tracer was observed in one case; and a noncommunicating cyst was observed in the other. The MR findings indicated a variation in the position of the basilar artery (BA) bifurcation in relation to the ventral surface of the midbrain. A distance existed between the BA bifurcation and the ventral surface of the midbrain in a communicating cyst with early filling, whereas the BA bifurcation was posteriorly displaced in a communicating cyst with delayed filling and also in a noncommunicating cyst, leaving little space between the bifurcation and the ventral surface of the midbrain. Endoscopic observation revealed, in the case of communicating cysts with early filling and early clearance of tracer, that the BA bifurcation is located inside the cyst with no overlying membrane, whereas in a noncommunicating cyst, the BA and its branches can be observed through the transparent membrane of the lesion.

Conclusions. The authors postulate two different types of suprasellar arachnoid cysts: a noncommunicating intraarachnoid cyst of the diencephalic membrane of Liliequist and a communicating cyst that is a cystic dilation of the interpeduncular cistern.

Key Words • suprasellar arachnoid cyst • magnetic resonance imaging • endoscopy • membrane of Liliequist • interpeduncular cistern

Arachnoid cysts account for approximately 1% of intracranial mass lesions. In a European cooperative study of arachnoid cysts in children published in 1992 it was reported that 11.3% of these lesions are located in the suprasellar region. Despite the rarity of these basal midline masses, they are being recognized with increasing frequency since the introduction of MR imaging, and they represent a surgically treatable cause of neurological symptoms.

Suprasellar arachnoid cysts are classified as communicating or noncommunicating cysts on pneumoencephalography and CT cisternography. The majority of suprasellar arachnoid cysts are congenital, secondary to an imperforate membrane of Liliequist. An imperforate membrane of Liliequist results in partial or complete obstruction of CSF flow at the level of the suprasellar cistern. Pulsations in CSF and elevated pressure below the obstructing membrane result in upward expansion of the membrane to form a cystic diverticulum that communicates with the preopticine cistern (communicating cysts). It has been suggested that noncommunicating cysts arise when the thin elongated neck of the diverticulum is pinched off, forming a true cyst. However, a uniform explanation of the pathogenesis certainly is not realistic.

We have treated nine cases of suprasellar arachnoid cysts and investigated them by using MR imaging and neuroendoscopy. According to our findings we propose two different types of suprasellar arachnoid cysts, one being an intraarachnoid cyst of the diencephalic membrane of Liliequist and the other a cystic dilation of the interpeduncular cistern.

Clinical Material and Methods

Nine children (four boys and five girls) were treated in our department. Three were younger than 1 year old and one was older than 10 years of age. We used various diagnostic methods to investigate the nine cases, including: 1) MR imaging with 3D-FSE and with 3D-FASE sequencing; 2) metrizamide and/or isovist CT cisternography; and 3) endoscopic observation of anatomical structures within and outside the arachnoid cyst.

The patients’ clinical characteristics are summarized in Table 1. The patient in Case 3 had an indisputable history of head trauma in infancy, and this child developed an abnormal increase in head circumference after the head injury. In Case 2, prenatal diagnosis by MR imaging indicated that the complaint was congenital in origin (Fig. 1). Of
the nine patients, who underwent various forms of therapy, four (Cases 2, 3, 5, and 7) were treated by either endoscopic ventriculocystostomy or ventriculocystocisternostomy.4,13

Results

On analysis of CT cisternograms obtained using metrizamide or isovist as tracers in nine cases of suprasellar cysts, we found three different types of lesions in terms of inflow and outflow of the tracer. A communicating cyst with early filling and early clearance of the tracer was found in seven of nine cases (Cases 3–9, Fig. 2). A communicating cyst with delayed filling and delayed clearance of the tracer was observed in one patient, in whom the tracer first filled the subarachnoid space surrounding the cyst and then gradually entered the lesion (Case 1, Fig. 3). A noncommunicating cyst was found in one patient (Case 2), in whom we observed that the subarachnoid space surrounding the lesion was filled with the tracer (Fig. 4). There is no previous mention of such an occurrence in this patient’s medical history.

When CT cisternographic findings were compared with MR imaging findings obtained using either 3D-FSE or 3D-FASE sequences, there was a variation between the positions of the BA bifurcation with respect to the ventral surface of the midbrain. A communicating cyst with early filling and early clearance of the tracer demonstrated distance between the BA bifurcation and ventral surface of the midbrain (Figs. 2 and 5), whereas the BA bifurcations of a communicating cyst with delayed filling and a noncommunicating cyst were posteriorly displaced, leaving little space between the bifurcation and the ventral surface of the midbrain (Figs. 3 and 4).

In the case of a communicating cyst with early filling and early clearance of the tracer, endoscopic observation revealed the BA bifurcation to be located inside the cyst without an overlying membrane. We could see the membrane at the bottom of the cyst and the slit through which the BA ascended (Figs. 2 and 5). In the case of the noncommunicating cyst, the BA and its branches could be observed through the transparent membrane of the cyst (Fig. 4).

Discussion

According to Starkman, et al.,21 Naidich, et al.,11 and other investigators, primary arachnoid cysts can be defined as developmental cavities that 1) are wholly located within the arachnoid membrane; 2) are lined with collagen and arachnoid mater cells; 3) contain clear CSF-like liquid; 4) have inner and outer walls at cyst margins; and 5) are continuous with the surrounding normal and noncystic arachnoid mater, signifying that the lesion is intrarachnoidal.

McLone and Bondareff9 stated that development of pia-arachnoid membranes in mice occurs in four stages. The mesenchyme over the telencephalic surface of a 10-day fetus typically has a large extracellular space. Cerebrospinal fluid begins to seep in and replace the mesenchymal ground substance by the 13th fetal day. The peripheral mesenchymal extracellular compartment is reduced, resulting in a compact pia-arachnoid zone that limits the peripheral extent of the subarachnoid space. This compact layer will itself form the outer layer of arachnoid and dura, and the loose mesenchyme remaining deep will form the pia, the inner arachnoid layer, and the subarachnoid space. By the 21st postnatal day, a subarachnoid space typical of an adult animal has been established.

According to Rengachary and Watanabe16 a minor aberration in flow of the CSF in the primordial stage of the development of the subarachnoid pathway results in sequestration of an enclosed chamber or diverticulum within the developing arachnoid membrane. Although we do not question the presence of congenital intraarachnoid

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**TABLE 1**

**Clinical characteristics of nine children with suprasellar arachnoid cysts***

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, Sex</th>
<th>History</th>
<th>Symptoms</th>
<th>Findings on CT Cisternography</th>
<th>Operation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5 mos, F</td>
<td>prenatal MR image</td>
<td>psychomotor retardation</td>
<td>delayed filling &amp; delayed clearance</td>
<td>SF &amp; CP</td>
</tr>
<tr>
<td>2</td>
<td>6 mos, M</td>
<td></td>
<td>macromenina at 6 mos</td>
<td>no filling</td>
<td>VC</td>
</tr>
<tr>
<td>3</td>
<td>11 mos, F</td>
<td>head trauma at 3 mos</td>
<td>macromenina posttrauma</td>
<td>early filling &amp; early clearance</td>
<td>VCC</td>
</tr>
<tr>
<td>4</td>
<td>2 yrs, F</td>
<td></td>
<td>psychomotor retardation</td>
<td>early filling &amp; early clearance</td>
<td>VP</td>
</tr>
<tr>
<td>5</td>
<td>4 yrs, F</td>
<td></td>
<td>macromenina at birth</td>
<td>early filling &amp; early clearance</td>
<td>SF</td>
</tr>
<tr>
<td>6</td>
<td>4 yrs, F</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>4 yrs, F</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>4 yrs, F</td>
<td></td>
<td>macromenina</td>
<td>early filling &amp; early clearance</td>
<td>VCC</td>
</tr>
<tr>
<td>9</td>
<td>14 yrs, F</td>
<td></td>
<td>visual impairment</td>
<td>early filling &amp; early clearance</td>
<td>SF</td>
</tr>
</tbody>
</table>

* Hydrocephalus was present in all but Case 9. Abbreviations: CP = cystoperitoneal shunt; SF = microsurgical subfrontal fenestration; TVF = microsurgical transventricular fenestration; VCC = endoscopic ventriculocystocisternostomy; VP = ventriculoperitoneal shunt.

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**FIG. 1.** Case 2. Prenatal T2-weighted MR images revealing a suprasellar arachnoid cyst.
cysts in various locations, a consistent description of the pathogenesis of these cysts certainly is unrealistic.

The temporal agenesis syndrome proposed by Robinson may result in middle cranial fossa arachnoid cysts, but he later abandoned his theory. Failure in the outward passage of CSF through the fourth ventricle or the imperforate inferior membranous area may result in persistent Blake’s pouch, whereas failure of CSF passage through the imperforate membrane of Liliequist may result in suprasellar arachnoid cysts.

The microanatomy of the basal cistern was reported by Matsuno, et al., who stated that the membrane of Liliequist splits into two as it spreads upward from the dorsum sellae and across the interval between the oculomotor nerves, and gives rise to two separate arachnoid sheets. An upper sheet, called the diencephalic membrane, attaches to the diencephalon at the posterior edge of the mamillary bodies and separates the chiasmatic and interpeduncular cisterns. The lower sheet, called the mesencephalic membrane, extends backward and attaches along the junction of the midbrain and pons to separate the interpeduncular and prepontine cisterns. The lateral edges of the diencephalic and mesencephalic membranes attach to the arachnoidal sheath surrounding the oculomotor nerves. The mesencephalic membrane frequently is incomplete and has an opening through which the BA ascends to reach the interpeduncular fossa. This membrane may form a tight cuff around the BA, but more commonly it has a large opening through which the BA ascends (Fig. 6 left).

Where cystic dilation of the interpeduncular cistern had occurred, the diencephalic membrane would constitute the dome, and the mesencephalic membrane the bottom of the cyst. In such a case, the BA bifurcation would remain inside the cyst (Fig. 6 right). In contrast, if there were progressive occurrence of an intraarachnoid lesion of the diencephalic membrane, the interpeduncular cistern would be compressed, leaving the BA bifurcation behind the posterior wall of the cyst (Fig. 6 center).

**Fig. 2.** Case 7. **Upper:** Series of CT cisternography studies revealing a communicating cyst with early filling and early clearance of tracer. **Lower Left:** Axial 3D-FSE MR image demonstrating the distance between the BA bifurcation and the ventral surface of the midbrain (arrow). **Lower Right:** Endoscopic views showing BA located inside the cyst with no overlying membrane (upper) and, at the bottom of the cyst, membrane and slit through which the BA ascends (lower).
Origin of suprasellar arachnoid cysts

Our proposal for a predictive classification is supported by the endoscopic observations of suprasellar cysts as reported by Santamarta, et al., and other investigators, including Caemaert, et al., and Schroeder and Gaab. Santamarta, et al., stated that the anatomical relationship between a cyst and the BA and its branches could be recognized through the transparent walls of the lesion. The other two groups of investigators reported that the BA and its branches were included inside the cyst.

The slit in the mesencephalic membrane that we observed endoscopically in two patients (Cases 3 and 7) could be either a slit valve or just a normal variation. We speculate that the slit functions as a one-way valve through which CSF flows to expand the cyst. According to this speculation, endoscopic treatment would have to be performed to remove this valve by making an opening in the lower wall of the communicating cyst. Therefore we recommend that ventriculocystocisternostomy should be performed to treat communicating cysts. On the other hand, a wide opening in the upper wall of the lesion would be enough to manage noncommunicating cysts, because this procedure could connect a cyst to basal cisterns and ventricles.

The origin of suprasellar arachnoid cysts does not appear to be consistent. As we observed in the patient in Case 3, a head injury in infancy may contribute to the pathogenesis of these types of cysts. It may be that not only congenital intraarachnoid cysts but also head injuries...
in infancy, as were reported by Choi and Kim,\(^3\) as well as unknown factors, may contribute to the formation of suprasellar arachnoid cysts.

**Conclusions**

In a retrospective study, neuroimaging and neurosurgical observations in nine patients with suprasellar arachnoid cysts indicated that the lesions were divided into the following two causally different types of cysts: 1) intrarachnoid cysts of the diencephalic membrane of Liliequist; and 2) cystic dilation of the interpeduncular cistern.

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**References**

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