The cisternal ventricle.

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The Cisternal Ventricle

John R. Jinkins

Hydrocephalus communicating with the basilar subarachnoid cisterns may manifest itself as enlargement of these spaces. Confirmation of this finding is based on resolution of dilatation after ventricular shunting. A series of eight patients points out the CT characteristics, the differential diagnosis, and the importance of the cisternal ventricle in the transmission of CSF.

Various extensions of the cerebral ventricular system have been alluded to in the literature, including the “fifth” and “sixth” ventricles [1–7]. Of great importance is the CSF space interposed between the spinal CSF tract, the subarachnoid spaces over the outer cerebral surfaces, and the CSF domain of the cerebral ventricles. This bridging compartment includes the vallecula, the cisterna magna, and the peribulbar cisterns; they can be considered together as a unit called the cisternal ventricle.

Subjects and Methods

Eight patients ages newborn to 3 years old with communicating hydrocephalus had CT on initial presentation (Table 1). Five-mm axial sections were obtained through the posterior fossa and 10-mm sections were obtained in the supratentorial region. A GE-9800 CT scanner was used throughout. Clinical signs included restlessness, nausea, and vomiting. Rapid circumferential head enlargement was a consistent observation in the very young. The etiology of the hydrocephalus included congenital-idiopathic causes, neonatal germinal matrix hemorrhage, and bacterial meningitis. The patients were rescanned at varying intervals after ventriculoperitoneal shunting. Resolution of the hydrocephalus in this series required 3 to 8 months.

Results

The initial CT examination invariably revealed supratentorial ventricular dilatation and mild to moderate enlargement of the fourth ventricle. In addition, associated with this classic appearance of communicating hydrocephalus was expansion of the vallecula, the cisterna magna, and the peribulbar subarachnoid cisterns (Figs. 1 and 2). After successful ventricular shunting, the cerebral ventricles were decompressed, the patients became asymptomatic, and the widened spaces of the cisternal ventricle largely “filled” with surrounding normal neural structures (Figs. 1 and 2). The structures that expanded to fill the CSF void included the cerebellar hemispheres, the vermis, and the brainstem.

Discussion

It is not surprising that the extracerebral subarachnoid spaces dilate in response to elevated CSF pressures in communicating hydrocephalus. The ventricular, spinal,
TABLE 1: Patients Evaluated with Communicating Hydrocephalus

<table>
<thead>
<tr>
<th>Age</th>
<th>Gender</th>
<th>Etiology of Hydrocephalus</th>
<th>Cisterna Magna</th>
<th>Valeculla</th>
<th>Peribulbar Cistern</th>
<th>Other</th>
<th>Time to Resolution of Hydrocephalus</th>
</tr>
</thead>
<tbody>
<tr>
<td>newborn</td>
<td>M</td>
<td>Congenital</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>6 months</td>
</tr>
<tr>
<td>newborn</td>
<td>M</td>
<td>Meningitis</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>4 months</td>
</tr>
<tr>
<td>2 months</td>
<td>F</td>
<td>Neonatal Hemorrhage</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>3 months</td>
</tr>
<tr>
<td>3 months</td>
<td>M</td>
<td>Neonatal Hemorrhage</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>6 months</td>
</tr>
<tr>
<td>10 months</td>
<td>M</td>
<td>Congenital</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>8 months</td>
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<tr>
<td>3 years</td>
<td>F</td>
<td>Congenital</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>4 months</td>
</tr>
<tr>
<td>3 years</td>
<td>F</td>
<td>Meningitis</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>5 months</td>
</tr>
</tbody>
</table>

Fig. 1.—Case 1. 10-month-old boy presenting with congenital hydrocephalus.
A, Axial CT section through lateral ventricles demonstrates severe hydrocephalus and transependymal CSF flow in frontal regions.
B, CT section through lower posterior fossa illustrates dilatation of temporal horns and of structures comprising cisternal ventricle: the cisterna magna, valeculla, and peribulbar cistern.
C, CT at 1 month after ventricular shunting shows a reduction in temporal-horn dilatation together with partial “filling in” of dilated cisternal ventricle.
D, Final resolution at 6 months after shunting reveals normal CT appearance of posterior fossa.
E, CT section at higher level also at 6 months illustrates complete decompression of supratentorial ventricular system resulting in “slit ventricles.”

optic, and extraventricular cranial CSF compartments all have the ability to act as individual, yet interconnected, units. As a part of this system, the cisternal ventricle behaves in the same manner, dilating and contracting in balance with the other CSF pathways, and it comprises one component of so-called external hydrocephalus. In fact, this "balance" may explain why the fourth ventricle is often not as dilated as are the third and lateral ventricles in patients with communicating hydro-
cerebrospinal fluid (CSF) compartments.

Further, the dilated cisternal ventricle points out a possible pitfall of CT interpretation. Before shunting, the appearance of the posterior fossa structures might be mistaken for mega cisterna magna, irreversible acquired atrophy, neural hypoplasia, or possibly the Dandy-Walker variant [8-15]. Care must be taken, therefore, in interpreting the appearance of the posterior fossa on CT scans of patients presenting with general CT findings compatible with communicating hydrocephalus. Perhaps a definitive evaluation is best delayed until after ventricular shunting and resolution of the hydrocephalus.

It is apparent, then, that the cisternal ventricle lies at the hub of the CSF pathways, whose radii include all the CSF spaces of the cranium and spine. It acts as a remarkably dynamic structure, capable of significant volume fluctuation in response to changes in pressure gradient between any one or combination of the neuraxis CSF compartments.

REFERENCES

2. Dandy WE. Congenital cerebral cysts of the cavum septi pellucidi (fifth ventricle) and cavum vergae (sixth ventricle) diagnosis and treatment. Arch Neurol Psychiatry 1931;25:44-66