Intraventricular septations complicating neonatal meningitis

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Ventriculography in seven infants who had developed hydrocephalus after surviving neonatal meningitis and ventriculitis disclosed intraventricular septations or veils. These post-inflammatory septations compartmentalized the ventricular system, interfering with attempted shunting procedures. Pathogenesis and treatment are discussed.

KEY WORD: meningitis hydrocephalus intraventricular septations neonatal shunt complications ventriculitis ventriculography

HYDROCEPHALUS frequently develops in infants who survive neonatal meningitis and ventriculitis. This may be a communicating hydrocephalus as a result of impaired absorption of cerebrospinal fluid (CSF) from the subarachnoid space, or a noncommunicating hydrocephalus due to obstruction of the foramina of Munro, the aqueduct of Sylvius, or the outlets of the fourth ventricle. We have performed air ventriculography in seven infants who had survived meningitis and then developed hydrocephalus. These studies were done prior to shunting procedures to demonstrate the extent of the hydrocephalus and the site of obstruction to CSF flow. Septations, or ventricular veils, were demonstrated within the lateral ventricles.

Clinical Material
Seven infants were selected from those referred to the Children's Hospital of Philadelphia between 1965 and 1969 with neonatal meningitis because they subsequently had developed hydrocephalus (Table 1). Patients were not included if there was any congenital anomaly that might have predisposed the patient to infection of the central nervous system. All seven infants received parenteral antibiotics. In addition, in Cases 1, 2, 4, and 5, antibiotics were injected into the lateral ventricles. Cases 1 and 2 have been previously reported because of the unusual organism involved.

Shunting procedures were performed in all of the infants, with multiple revisions of the shunt in most of the cases and bilateral shunting in two cases. Five of the seven infants died at 5 to 34 months of age. The two survivors had serious neurological defects when last seen at 2 and 4½ years of age respectively.

Results
The ventricular septations were found to vary in extent and distribution (Table 2).
In Case 1, serial ventriculograms demonstrated a changing pattern of septations...
Intraventricular septations complicating neonatal meningitis

**TABLE 1**

*Clinical summary*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Perinatal Complications</th>
<th>First Symptoms</th>
<th>Age (days) at Diagnosis of Menigitis, Ventriculitis</th>
<th>Organism</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>membranes ruptured early; Caesarean section at 41 weeks for uterine inertia; Cyanosis 8th day</td>
<td>11 34</td>
<td>Flavobacterium</td>
<td>died at 8 mos</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>membranes ruptured early; mother treated with antibiotics; Seizures 10th day</td>
<td>10 29</td>
<td>Flavobacterium</td>
<td>at 4½ yrs deafness, seizures, spastic quadriplegia, and moderate mental retardation</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>None; Poor feeding 13th day</td>
<td>15 16</td>
<td>Streptococcus</td>
<td>died at 34 mos</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Amnionitis; Poor feeding 3rd day</td>
<td>17 43</td>
<td>E. coli</td>
<td>at 2 yrs seizures, spastic quadriplegia, and severe mental retardation</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Birth weight of 4 lb 4 oz at 8 mos; Poor feeding 7th day</td>
<td>9 27</td>
<td>E. coli</td>
<td>died at 1 yr</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>None; Poor feeding 3rd day</td>
<td>33</td>
<td>Staph. aureus</td>
<td>died at 5 mos</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Membranes ruptured early; Cyanosis 7th day</td>
<td>8 28</td>
<td>E. coli</td>
<td>died at 8½ mos</td>
<td></td>
</tr>
</tbody>
</table>

**TABLE 2**

*Summary of abnormalities seen in ventriculograms*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (mos)</th>
<th>Abnormality</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>Aqueductal occlusion</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>Obstruction of the foramina of Munro</td>
</tr>
<tr>
<td>5</td>
<td>5</td>
<td>Obstruction of the foramina of Munro</td>
</tr>
<tr>
<td>6</td>
<td>6</td>
<td>Aqueduct and foramina of Munro patent; Obstruction of the outlets of the 4th ventricle; Webs in the rt. lateral ventricle</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>Obstruction of the outlets of the 4th ventricle</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>Aqueductal occlusion</td>
</tr>
<tr>
<td>11</td>
<td>11</td>
<td>Aqueductal occlusion, Loculated cyst in left temporal horn</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>Multiple webs in both lateral ventricles</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
<td>Multiple webs in left lateral ventricle</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>Multiple webs in both lateral ventricles</td>
</tr>
<tr>
<td>6</td>
<td>6</td>
<td>Multiple webs in both lateral ventricles, Obstruction of the foramina of Munro</td>
</tr>
<tr>
<td>5</td>
<td>3</td>
<td>Communicating hydrocephalus</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>Multiple webs in both lateral ventricles</td>
</tr>
<tr>
<td>6</td>
<td>1</td>
<td>Aqueductal occlusion</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>Multiple webs in both lateral ventricles</td>
</tr>
<tr>
<td>7</td>
<td>1</td>
<td>Multiple webs in both lateral ventricles</td>
</tr>
</tbody>
</table>

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Paul Schultz and Norman E. Leeds

Fig. 1. Case 1. Changing pattern of septations. Upper Left: First ventriculogram at 2 months showing hydrocephalus and aqueductal stenosis (arrow). Upper Right: Brow-up view of second ventriculogram at 3 months demonstrating air confined to the dilated left lateral ventricle because of obstruction of the foramina of Munro. Lower Left: Horizontal beam decubitus projection at 5 months demonstrating a septated ventricle with air in several compartments. Lower Right: Fourth ventriculogram at 6 months showing resolution of the obstruction at the foramina of Munro, but with formation of outlet obstruction of the fourth ventricle (arrows).

(Fig. 1). This patient received intensive intraventricular treatment including multiple instillations of cephalothin through an indwelling right lateral ventriculostomy during the 2 weeks prior to the first ventriculogram, multiple instillations of erythromycin through the ventriculostomy during the week prior to the second ventriculogram, and erythromycin in the interval between the third and fourth ventriculograms.

In Case 2, obstruction of the outlets of the fourth ventricle was originally demonstrated at 1 month of age, and 1 month later, aqueductal occlusion was seen. At 7 months, however, a solitary septum was demonstrated in the left temporal horn, enclosing a fluid-filled cavity that resembled a mass. A veil within the left temporal horn forming a loculated cyst was fenestrated, leading to good control of the hydrocephalus. This patient had been given two intrathecal administrations of polymyxin B prior to the first ventriculogram and multiple intraventricular instillations of vancomycin.
Intraventricular septations complicating neonatal meningitis

through an indwelling left lateral ventriculostomy between the first and second ventriculograms. She received no intrathecal or intraventricular medications between the second and third ventriculograms.

In Cases 3, 4, and 7, there were multiple webs in both lateral ventricles in ventriculograms done prior to the administration of any intraventricular medication (Fig. 2). In Case 4, an attempt was made to prevent the proliferation of these webs by instilling intraventricular streptokinase-streptodornase with the polymyxin B being given to eradicate the infection. Follow-up ventriculograms showed persistence of the webs, with no apparent benefit from the enzyme instillations.

In Case 5, ventriculography at 3 months of age showed only communicating hydrocephalus, with no septations and no obstruction to CSF flow. This initial study followed intrathecal, subdural, and intraventricular polymyxin B instillations. Repeat ventriculography 1 month later, with no signs of infection and no intraventricular instillations in the interval, showed multiple webs in both lateral ventricles.

In Case 6, ventriculography at 1 month of age showed only aqueductal occlusion. Three months later, there were multiple webs in both lateral ventricles, there had been no intraventricular medications in the interval.

Pathology

Gross and histological studies were made on the brains from Cases 3, 6, and 7; these infants died at 34, 5, and 8½ months respectively. Coronal brain sections demonstrated multiple intraventricular septations that corresponded to the webs seen in the ventriculograms (Fig. 3). Grossly, the septations appeared as filmy, translucent membranes. Microscopically, they were

Fig. 2. Case 7. Left: Multiple intraventricular webs on the original ventriculogram. Right: Ventriculogram showing numerous loculi within the lateral ventricles produced by the septations.

Fig. 3. Case 7. Coronal section of the brain showing marked hydrocephalus with filmy intraventricular webs corresponding to the septations seen on ventriculography (arrow).
composed of fibroglial elements, with round cells and some polymorphonuclear cells. The characteristic findings of subacute and chronic ventriculitis were also present, with subependymal gliosis, small areas of denuded ependyma, and glial tufts extending through the denuded ependyma into the ventricular lumen (Fig. 4 left). The fibroglial membranes appeared to be extending from the glial tufts. In addition, the aqueduct of Sylvius was obliterated by a web of organized debris and inflammatory cells (Fig. 4 right).

Discussion

Meningitis is seen more frequently in the first month of life than in any other 30-day period. The incidence of neonatal meningitis is estimated at 0.13 to 0.37 cases per
Intraventricular septations complicating neonatal meningitis

1000 full-term births, and 1.36 to 2.24 cases per 1000 premature births.\(^5_{,}^{11}\) The mortality rate ranges from 60% to 75%,\(^1_{,}^{11}\) 16,19,20,21 with serious neurological sequelae in 32%\(^1_{,}^{19,21}\) to 63%\(^2_{,}^{20}\) of the survivors, even in the antibiotic era. The morbidity and mortality rates are worse in premature infants.\(^5_{,}^{11}\) The variety of clinical presentations of neonatal meningitis, the etiological agents, and the relationship of neonatal infections to obstetrical complications are described in numerous reviews of neonatal meningitis.\(^2_{,}^{4_{,}^{,}5_{,}^{,}11_{,}^{,}19_{,}^{,}20_{,}^{,}21}\) Lorber and Pickering\(^7\) estimated that the incidence of hydrocephalus in survivors of neonatal meningitis was 31%. In a clinical description of their experience with post meningitic hydrocephalus in the newborn, they described “multiple obstructions to the flow of CSF in many infants” and “multiple loculi which precluded any possibility of surgical treatment” in one infant, but offered no radiological demonstrations or pathological descriptions.

Histological evidence of ventriculitis was found in 92% of a series of autopsied cases of neonatal meningitis.\(^2\) The actual incidence of ventriculitis in surviving patients, however, is uncertain, since diagnostic ventricular taps are not done routinely in the management of neonatal meningitis. Usually only infants who respond poorly to antibiotic therapy or have a rapid increase in head circumference have the ventricular taps that provide the opportunity for substantiating the diagnosis of ventriculitis antemortem.

The treatment of ventriculitis with systemic antibiotics is often unsuccessful. In our experience, and in that of others,\(^6_{,}^{10,18}\) intrathecal and intraventricular instillations of antibiotics are frequently necessary to sterilize the CSF.

Shunting procedures are required to control the hydrocephalus that is present in these infants. The compartmentalization of the lateral ventricles caused by the postventricular septations interfere with the drainage of the ventricles, making therapeutic shunting difficult. To cope with this problem, numerous shunt revisions and occasional bilateral shunts are required. Fenestration of the ventricular veil, performed in our Case 2, was successful in controlling the hydrocephalus.

To reduce the inflammatory response in neonatal meningitis and ventriculitis, intraventricular instillations of hydrocortisone has been advocated.\(^8\) We gave intramuscular corticosteroid in Cases 1 and 5 without apparent benefit. An attempt to prevent the formation of septations by intraventricular instillation of streptokinase-streptodornase was unsuccessful in Case 4.

Pathogenesis

Septations within the lateral ventricles have not been described in previous discussions of neonatal meningitis.\(^1_{,}^{,}3,11_{,}^{,}15_{,}^{,}16_{,}^{,}17,19\) We feel these septations probably represent the organization of intraventricular exudate and debris accompanying the ventriculitis. A subependymal gliotic process disrupts the ependyma, producing glial tufts that act as a nidus for the formation of the septations within the lateral ventricles.

The formation of the intraventricular septations was not related to any specific bacterial agent. The instillation of intraventricular antibiotics might be considered a causative factor; however, three of the seven infants had no intraventricular treatment prior to the demonstration of multiple webs in both lateral ventricles. Furthermore, in three other cases, webs proliferated during a period when no intraventricular medications had been given. The elevated ventricular fluid protein concentration in all seven infants may have been a factor related to the formation of the intraventricular septations.

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