CEREBROSPINAL FLUID CIRCULATION FOLLOWING SUBARACHNOID HEMORRHAGE

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Alterations in the cerebrospinal fluid (CSF) circulation may follow subarachnoid hemorrhage although the frequency and extent to which this occurs is unknown. Accurate definition of these abnormalities is important because in some instances they result in symptomatic communicating hydrocephalus (1,2). Current methods of assessing the patency of the subarachnoid space and ventricular size include pneumoencephalography and ventriculography. These techniques provide excellent anatomic detail but give little direct information about the functional integrity of the CSF circulation. CSF pressure measurements often are of no help because they may be normal in the presence of marked ventricular dilatation (3).

Radioisotope cisternography has been shown to be useful in the study of CSF circulation in a variety of disease states (4,5). The purpose of this report is to describe the cisternographic patterns of CSF circulation in nine patients 1–6 months after subarachnoid hemorrhage.

METHODS AND MATERIALS

All patients were studied because of persistent neurologic signs and symptoms suggestive of communicating hydrocephalus. In each patient subarachnoid hemorrhage was caused by a ruptured Berry aneurysm which was demonstrated by carotid angiography and treated by surgical clipping. Postoperative angiography showed obliteration of the aneurysm in all cases. The clinical data are summarized in Table 1.

Cisternography was performed using essentially the technique described by Glasauer (6). One hundred microcuries of high specific activity (1–5 mg) 131I-labeled human serum albumin (IHSA) in a volume of 0.1–0.5 ml was given intrathecally by cisterna magna or lumbar injection. The intracranial distribution of IHSA was visualized by scintillation photoscanning at 3, 24, and 48 hr after injection.

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<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Bleeding site</th>
<th>Weeks between SAH and cisternogram</th>
<th>Clinical signs</th>
<th>CSF pressure (mm CSF)</th>
<th>Shunt</th>
<th>Followup</th>
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<tbody>
<tr>
<td>1</td>
<td>33</td>
<td>Anterior</td>
<td>6</td>
<td>Akinetic mute, incontinence</td>
<td>165</td>
<td>Yes</td>
<td>1 yr. Complete recovery</td>
</tr>
<tr>
<td>2</td>
<td>40</td>
<td>Anterior</td>
<td>7</td>
<td>Dementia, incontinence</td>
<td>160</td>
<td>Yes</td>
<td>6 mo. Complete recovery</td>
</tr>
<tr>
<td>3</td>
<td>28</td>
<td>Anterior</td>
<td>26</td>
<td>Akinetic mute</td>
<td>150</td>
<td>Yes</td>
<td>4 mo. Independent in affairs of daily living; some residual</td>
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<tr>
<td>4</td>
<td>51</td>
<td>Anterior</td>
<td>21</td>
<td>Dementia</td>
<td>200</td>
<td>Yes</td>
<td>Infection necessitated shunt removal, awaiting shunt replacement</td>
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<tr>
<td>5</td>
<td>32</td>
<td>Left middle</td>
<td>4 &amp; 12</td>
<td>Headache, aphasia</td>
<td>125</td>
<td>No</td>
<td>6 mo. Essentially complete recovery</td>
</tr>
<tr>
<td>6</td>
<td>59</td>
<td>Left internal</td>
<td>3</td>
<td>Headache</td>
<td>65</td>
<td>No</td>
<td>6 mo. Complete recovery</td>
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<tr>
<td>7</td>
<td>42</td>
<td>Anterior</td>
<td>4</td>
<td>Headache, memory loss</td>
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<td>None</td>
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<tr>
<td>8</td>
<td>49</td>
<td>Right internal</td>
<td>6</td>
<td>Confusion</td>
<td>165</td>
<td>No</td>
<td>3 mo. Partial recovery (residual symptoms not felt to be due to communicating hydrocephalus)</td>
</tr>
<tr>
<td>9</td>
<td>37</td>
<td>Anterior</td>
<td>6 &amp; 19</td>
<td>Incontinence, confusion</td>
<td>75</td>
<td>No</td>
<td>3 mo. Complete recovery</td>
</tr>
</tbody>
</table>

TABLE 1. CLINICAL INFORMATION
FIG. 1. Anterior and left lateral photoscans of Patient 5 at 3, 24, and 48 hr after lumbar injection of IHSA. There is prompt accumulation and persistence of radioactivity in ventricles and absence of radioactivity over convexities indicating basilar block. Note accumulation of activity at operative site (left frontal) on 48-hr scan.

Scans were performed using a rectilinear scanner with a 5-in. NaI(Tl) crystal and a 19-hole focusing collimator with a 3-in. focal length. One anterior and one lateral view were routinely obtained. Stable iodine was given to block thyroid uptake of free $^{131}$I.

RESULTS

The pattern of CSF circulation was distinctly and uniformly abnormal in all nine patients. At 3 hr there was prominent accumulation of IHSA in the basal cisterns and in the lateral ventricles which appeared enlarged. The ventricular radioactivity persisted on all of the 24-hr and on some of the 48-hr scans. Circulation of IHSA into the cerebral subarachnoid space and over the convexities was decreased and no radioactivity appeared in the region of the sagittal sinus (Fig. 1). This circulatory pattern is consistent with that previously described in communicating hydrocephalus (6). Concentration of IHSA in the area of the operative site was also routinely seen on the 48-hr scans.

In the four patients with the most severe symptoms, pneumoencephalography confirmed the presence of symmetrical ventriculomegaly and an absence of air over the convexities indicative of basilar subarachnoid block. These four patients subsequently received ventriculo-atrial shunts. Three showed excellent clinical improvement; the fourth patient’s shunt became infected, requiring its removal. Because the remaining five patients showed steady clinical improvement, shunting was not performed. Repeat cisternography in Patients 5 and 9 (not shunted) 2 and 3 months later, after marked clinical improvement, showed essentially no change in the abnormal CSF circulation pattern.

DISCUSSION

The normal pattern of CSF circulation depicted by IHSA is well described (4,6,7): When IHSA is injected into the lumbar subarachnoid space or cisterna magna, it promptly flows into the basal cisterns and cerebral subarachnoid space and migrates over the cerebral hemispheres to the sagittal sinus. Within 24 hr most of the IHSA is absorbed into the general circulation (Fig. 2). Normally IHSA does not enter the ventricles, presumably because CSF produced primarily by the choroid plexus creates a continuous flow or current of CSF from the ventricles into the basal cisterns.

The findings in this group of patients are consistent with a block in the CSF circulatory pathway which prevents normal egress of CSF from the basal cisterns.
cisterns. This results in ventricular dilatation and reversal of the normal flow, permitting reflux of IHSA into the dilated ventricles. Absorption of IHSA into the general circulation apparently then occurs directly across the ventricular walls by way of the ependymal cells (8). The mechanism by which the block is produced is not well understood but is thought to be related to arachnoiditis and fibrosis caused by bleeding into the subarachnoid space (9). Since all of these patients were operated upon, the additional possibility that the abnormal CSF circulation resulted from the operative procedure itself cannot be excluded.

Despite similar circulatory patterns on cisternograms, these patients had variable clinical courses. Four patients developed progressive symptomatic communicating hydrocephalus which was relieved by ventriculo-atrial shunt. The remaining five patients improved spontaneously without shunting, and in two of these (No. 5 and No. 9) the abnormal CSF circulation pattern persisted after almost complete neurologic recovery had occurred. These cases provide further evidence that communicating hydrocephalus can vary from incapacitating neurologic disease to an asymptomatic "compensated" condition.

Patients suffering subarachnoid hemorrhage often present complex management problems. Persistent neurologic deficits or clinical deterioration following successful surgical treatment can result from brain injury at the time of subarachnoid hemorrhage, at operation, or postoperatively from arterial spasm, as well as from subsequent hydrocephalus. Similarly, communicating hydrocephalus can present such diverse clinical signs as dementia, akinetic mutism, ataxia, and spastic paraparesis. In this clinical setting, cisternography appears to be a safe and sensitive method for detecting the abnormal CSF circulation characteristic of communicating hydrocephalus.

Our experience suggests that significant accumulation of IHSA in the lateral ventricles indicates ventriculomegaly, and failure of the IHSA to circulate over the cerebral convexities indicates basilar subarachnoid block compatible with communicating hydrocephalus. As presently performed, however, cisternography does not differentiate compensated from decompensated hydrocephalus. For this reason, caution should be used in recommending shunts on the basis of abnormal cisternograms in the early postsubarachnoid hemorrhage period (up to 12 weeks) since many patients will improve spontaneously despite abnormal CSF circulatory patterns.

Conversely, a normal cisternogram in symptomatic patients following subarachnoid hemorrhage should effectively exclude basilar subarachnoid block and communicating hydrocephalus as a diagnostic possibility. While none of the patients in this series had normal CSF circulatory patterns, they were highly selected, and the frequency with which CSF circulatory abnormalities occur following SAH remains to be established.

**SUMMARY**

Radioisotope cisternograms were performed on nine patients suspected of having communicating hydrocephalus following subarachnoid hemorrhage. All patients showed uniformly abnormal CSF flow, the main features being accumulation and persistence of IHSA in the enlarged ventricles, decreased migration of IHSA over the cerebral hemispheres, and late accumulation of IHSA in the area of the operative site. Despite the similar appearance of the cisternograms, the clinical courses were quite variable; four patients developed progressive neurologic signs and symptoms which were relieved by ventriculo-atrial shunt while the remaining five patients improved spontaneously. Repeat cisternography performed on two of the latter patients showed a persistently abnormal pattern even though they had made an excellent recovery. Cisternography is a sensitive means of delineating abnormal CSF circulation; however, it does not differentiate compensated from decompensated communicating hydrocephalus.

**ACKNOWLEDGMENT**

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**REFERENCES**

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