Treatment of Acute Hydrocephalus After Subarachnoid Hemorrhage With Serial Lumbar Puncture

Djo Hasan, MD; Kenneth W. Lindsay, PhD, FRCS; and Marinus Vermeulen, MD

Computed tomography demonstrated acute hydrocephalus ≤72 hours after subarachnoid hemorrhage in 24 (23%) of 104 patients. Of these 24 patients, six (25%) had no impairment of consciousness. In nine (11%) of the remaining 80 patients, acute hydrocephalus developed within 1 week after subarachnoid hemorrhage. With the exception of three patients, all 104 patients received antifibrinolytic treatment. Delayed clinical deterioration from acute hydrocephalus occurred in seven (29%) of the 24 patients with acute hydrocephalus on admission and in six (8%) of the remaining 80 patients. Serial lumbar puncture was performed in 17 patients. Twelve (71%) of the 17 patients treated with serial lumbar puncture, including 10 (77%) of the 13 patients with delayed deterioration from acute hydrocephalus after admission, achieved improvement in the level of consciousness. Four of these 17 patients (4% of all 104 patients) required an internal shunt. No patient deteriorated from coning following serial lumbar puncture. The rebleeding rate within 12 days after subarachnoid hemorrhage in hydrocephalic patients with serial lumbar puncture was not higher than the rate in those without hydrocephalus (two [12%] of 17 versus nine [13%] of 71). Neither meningitis nor ventriculitis was observed. We conclude that if neither a hematoma with a mass effect nor an obstructive element exists, cerebrospinal fluid drainage with serial lumbar puncture is a good alternative to ventricular drainage in patients with acute hydrocephalus after subarachnoid hemorrhage. (Stroke 1991;22:190–194)

A acute hydrocephalus is a frequent complication following subarachnoid hemorrhage. In a series of 473 patients with subarachnoid hemorrhage admitted ≤72 hours after the initial hemorrhage, hydrocephalus, defined as a bicaudate index on a computed tomogram (CT scan) exceeding the 95th percentile for age, occurred in 20%. Management of these patients presents problems. Not all patients with acute hydrocephalus and a decreased level of consciousness on admission require treatment, and approximately 40% improve spontaneously within 24 hours. However, in some patients a delay in reducing the high intracranial pressure could produce deleterious effects, resulting in irreversible brain damage or death from cerebral ischemia. On the other hand, external ventricular drainage carries a risk of ventriculitis and rebleeding although in a recent editorial Heros recommended “immediate ventricular drainage” in patients with significant hydrocephalus and a decreased level of consciousness. Serial lumbar puncture provides an alternative and simpler approach. We investigated the effectiveness of serial lumbar puncture in restoring the level of consciousness in patients with acute hydrocephalus and as- sessed the complications of this treatment.

Subjects and Methods

We entered 104 consecutive patients with subarachnoid hemorrhage admitted ≤72 hours after the initial hemorrhage into this study. Twenty-six patients were admitted to the Department of Neurosurgery of the Royal Free Hospital, London and the other 78 to the Department of Neurology of the University Hospital Dijkzigt, Rotterdam. All patients had clinical signs of subarachnoid hemorrhage and abnormalities suggesting a ruptured aneurysm on a CT scan. Patients with a perimesencephalic hemorrhage or a negative angiogram and patients who were moribund on admission were excluded.

Events were prospectively recorded during 28 days after the initial subarachnoid hemorrhage or until death or surgical treatment of the aneurysm. An initial CT examination was carried out on admission,
and CT was repeated after any clinical deterioration. The amount of cisternal blood on the initial CT scan was graded on a scale of 0 to 3 separately for each of the 10 cisterns (maximum score of 30).9 Similarly, the amount of intraventricular blood was graded separately for each of the four ventricles (maximum score of 12).8,9 Because an intraventricular score of 1 reflects sedimentation of blood in the ventricle, we defined intraventricular blood to be present if at least one of the four ventricles had a score of >1. We assessed the level of consciousness with the 14-point Glasgow Coma Scale (GCS).10,11 A decreased level of consciousness was defined as a reduction of at least 1 point in the motor or verbal score on the GCS. We defined events as probable delayed cerebral ischemia: gradual development of focal neurologic signs, with or without deterioration in the level of consciousness, without confirmation by CT or autopsy; definite delayed cerebral ischemia: deterioration in the level of consciousness or development of focal signs, or both, with CT or autopsy confirmation of cerebral infarction; probable rebleeding: sudden deterioration and death, without the possibility of proof by CT or if autopsy was refused; definite rebleeding: sudden deterioration with an increased amount of blood on a repeat CT scan or at autopsy compared with a previous CT scan; and acute hydrocephalus: increase in the bicaudate index beyond the upper limit (95th percentile for age) measured on the initial CT scan or on a CT scan repeated ≤1 week after the initial subarachnoid hemorrhage.1,2 The upper limits were <36 years of age, 0.16; 36–45 years, 0.17; 46–55 years, 0.18; 56–65 years, 0.19; 66–75 years, 0.20; and 76–85 years, 0.21.12,13 Deterioration from hydrocephalus was defined as deterioration in the level of consciousness with no detectable cause other than hydrocephalus, confirmed by repeat CT.

Serial lumbar puncture was performed when acute hydrocephalus appeared to cause impairment of consciousness in the absence of a hematoma with a mass effect and provided that blood did not completely fill the third or fourth ventricle, obstructing the ventricles system.14 If such an obstructing element exists, lumbar puncture is contraindicated and ventricular drainage should be performed. Each time a maximum of approximately 20 ml cerebrospinal fluid (CSF) was removed, we aimed at a closing pressure of 15 cm H₂O.

Of the 104 patients, 101 received tranexamic acid. Fluid intake was maintained at 3 l/day in all patients; fluid restriction and diuretic medication after admission were avoided. Patients receiving antihypertensive therapy on admission continued to receive treatment; otherwise none was given. Cerebral angiography and aneurysm operation were performed according to the patient's clinical condition. Aneurysm operation was usually planned between days 7 and 10 in London and on day 12 in Rotterdam. All survivors were followed up at 3 months and graded according to the five-point Glasgow Outcome Scale.15

The fourfold tables were analyzed with the two-sided Fisher's exact probability test.

Results

Measurement of the initial CT scan indicated acute hydrocephalus in 24 (23%) of the 104 patients. Of these 24 patients, six (25%) had no impairment of consciousness on admission (a total score on the GCS of 14), five (21%) had a GCS score of 13, and the remaining 13 (54%) had a GCS score of <13. In another nine patients (11% of the 80 patients without hydrocephalus on admission), repeat CT ≤1 week after the initial hemorrhage demonstrated delayed ventricular enlargement.

Patients with acute hydrocephalus had a higher incidence of impaired consciousness on admission, a higher score for cisternal blood, and a higher frequency of ventricular blood than those with normalized ventricles (Table 1). Clinical deterioration occurred after admission in seven (29%) of the 24 patients with acute hydrocephalus on the initial CT scan and in six of the nine patients who had delayed ventricular enlargement (i.e., in 8% of the 80 patients without acute hydrocephalus on the initial CT scan).

Serial lumbar puncture was performed in 17 patients, immediately in five of them because they had a decreased level of consciousness on admission. In the remaining 13 of the total 18 hydrocephalic patients with a decreased level of consciousness on admission (GCS score of <14), lumbar puncture was not performed. Six of the 13 improved spontaneously after admission; in four patients there was doubt as to whether the decreased level of consciousness was caused by hydrocephalus, and three patients died <24 hours after admission, one with a massive ventricular clot and two with early rebleeding. One of these latter two patients had a hematoma with a mass effect. In the remaining 12 of the 17 patients who underwent serial lumbar puncture, this treatment was started ≤1 week after admission. In 11 of these 12 patients without a decreased level of consciousness on admission, lumbar puncture was not performed until clinical deterioration from acute hydrocephalus had developed. The twelfth patient had hydrocephalus and a decreased level of consciousness on admission, but lumbar puncture was delayed because of fluctuation in the level of consciousness.

Figure 1 summarizes the period during which lumbar punctures were required and the number of punctures performed in each patient.

If analysis is restricted to the 13 patients who showed clinical deterioration from acute hydrocephalus, serial lumbar puncture improved the level of consciousness in 10 (77%). If analysis includes all 17 patients who underwent lumbar puncture, the level of consciousness improved in 12 (71%) (within 1 day in 10 patients and within 6 days in two); six patients recovered fully (GCS score of 14) and the other six did not improve beyond a GCS score of 13 (disoriented). In two of these latter six patients, an internal shunt was inserted, followed by sustained improvement in
TABLE 1. Entry Characteristics of 104 Patients With Aneurysmal Subarachnoid Hemorrhage by Presence of Acute Hydrocephalus and by Treatment With Serial Lumbar Puncture

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No hydrocephalus (n=71)</th>
<th>Hydrocephalus (n=16)</th>
<th>Hydrocephalus (n=17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>GCS on admission</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>≤12</td>
<td>20</td>
<td>28</td>
<td>8</td>
</tr>
<tr>
<td>13</td>
<td>16</td>
<td>23</td>
<td>2</td>
</tr>
<tr>
<td>14</td>
<td>35</td>
<td>49</td>
<td>6</td>
</tr>
<tr>
<td>Cisternal blood</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>0–6</td>
<td>11</td>
<td>15</td>
<td>2</td>
</tr>
<tr>
<td>7–12</td>
<td>10</td>
<td>14</td>
<td>2</td>
</tr>
<tr>
<td>13–18</td>
<td>26</td>
<td>37</td>
<td>7</td>
</tr>
<tr>
<td>19–24</td>
<td>14</td>
<td>20</td>
<td>2</td>
</tr>
<tr>
<td>25–30</td>
<td>6</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>Not scored</td>
<td>4</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Ventricular blood</td>
<td>7</td>
<td>10</td>
<td>7</td>
</tr>
</tbody>
</table>

GCS, total score on 14-point Glasgow Coma Scale.

FIGURE 1. Scatter plot of period during which lumbar punctures (LPs) were required (left) and number of LPs performed (right) in each patient.

the level of consciousness (GCS score of 14), and the remaining four patients died of other complications several days after serial lumbar puncture was started. In five (29%) of the 17 patients, serial lumbar puncture had no effect; two of these patients received an internal shunt followed by sustained improvement (GCS score of 14), and the other three patients died of other complications several days after the commencement of serial lumbar puncture. In summary, four (12%) of the 33 patients with acute hydrocephalus received an internal shunt (4% of all patients). No patient who underwent serial lumbar puncture or who received an internal shunt developed clinical signs of meningitis or ventriculitis. Although five patients failed to improve after lumbar puncture, none deteriorated from transtentorial herniation during the days following serial lumbar puncture.

Analysis of probable and definite rebleeding was restricted to the first 12 days. The incidence of rebleeding in hydrocephalic patients treated with serial lumbar puncture was not higher than that in patients without acute hydrocephalus (two [12%] of 17 versus nine [13%] of 71 patients, Table 2). Rebleeding occurred more frequently in hydrocephalic patients who were not treated with serial lumbar puncture than in those without acute hydrocephalus (Table 2), but this difference was not significant (p=0.999).

The incidence of definite and probable cerebral ischemia in patients with acute hydrocephalus was no higher than that in those without hydrocephalus (9 [27%] of 33 versus 22 [31%] of 71 patients). The frequency of cerebral ischemia in hydrocephalic patients treated with serial lumbar puncture was similar to that of patients without acute hydrocephalus (6 [35%] of 17 versus 22 [31%] of 71 patients, Table 2). Cerebral ischemia in patients with acute hydrocephalus treated with serial lumbar puncture was more often fatal (four [67%] of six patients) than in patients without hydrocephalus (seven [32%] of 22 patients), but the number of patients was small and the difference was not significant (p=0.141).
TABLE 2. Rebleeding and Cerebral Ischemia in Relation to Acute Hydrocephalus and Serial Lumbar Puncture in 104 Patients With Aneurysmal Subarachnoid Hemorrhage

<table>
<thead>
<tr>
<th>Serial lumbar puncture</th>
<th>No hydrocephalus (n=71)</th>
<th>Hydrocephalus (n=16)</th>
<th>Hydrocephalus (n=17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rebleeding by day 12</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Cerebral ischemia</td>
<td>9</td>
<td>13</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>22</td>
<td>31</td>
<td>3</td>
</tr>
</tbody>
</table>

Definite and probable events.

Discussion

In patients with a hematoma occluding the third, fourth, or lateral ventricles, an obstructive element exists, contraindicating lumbar puncture and making ventricular drainage necessary. In our series of 104 consecutive patients with aneurysmal subarachnoid hemorrhage, serial lumbar puncture performed in 17 with acute hydrocephalus resulted in an improvement in the level of consciousness in 12 (71%). Half of the patients recovered fully and the other half improved, but not beyond a GCS score of 13. Of all 104 patients, only 4% required internal shunting.

The frequency of acute hydrocephalus in this series matches that in other recent studies.1,16 The extent to which acute hydrocephalus on admission affected the level of consciousness closely approximated that of a previous series.2 The proportion of patients with acute hydrocephalus who improved after lumbar puncture is similar to that in previous series who improved after ventricular drainage.2 A direct comparison of treatment with external ventricular drainage versus treatment with lumbar puncture is not feasible. It is likely that serial lumbar puncture is carried out more readily and with less delay than is external ventricular drainage in hydrocephalic patients with an impaired level of consciousness, but it is also likely that some of these patients might have improved spontaneously if treatment had been delayed. However, the beneficial effect of lumbar puncture cannot be attributed to spontaneous improvement in all patients since progressive deterioration in the level of consciousness from acute hydrocephalus halted and improved in 77% of the patients shortly after lumbar puncture.

In the series of patients treated with ventricular drainage, the risk of rebleeding was significantly increased during the first 12 days.2 Several factors may play a role: rapid decreases in CSF pressure and ventricular size may displace the aneurysm clot; insertion of the ventricular catheter may induce intracranial fibrinolytic activity, resulting in lysis of the aneurysm clot; and there may be a difference in neurologic grade between the treated and the untreated groups.3,17,18 On the other hand, in a large series of patients in which the occurrence of rebleeding was studied prospectively with clearly defined criteria, no association between rebleeding and the initial condition could be demonstrated.19 We observed no such difference in the rebleeding rate between patients who underwent lumbar puncture and those who did not. A possible explanation for this is that the drop in CSF pressure after lumbar puncture is more gradual than that after insertion of a ventricular catheter or that lumbar puncture does not induce intracranial fibrinolytic activity. However, this cannot be concluded with certainty since there is an important difference between this study and the previous study.2 In the previous study analysis of patients treated with ventricular drainage was restricted to those not receiving antifibrinolytic treatment, whereas in this study nearly all patients received tranexamic acid.

A high CSF pressure in patients with acute hydrocephalus after subarachnoid hemorrhage may reduce cerebral perfusion and theoretically contribute to ischemic complications. Continuous ventricular drainage should ensure a constant reduction in CSF pressure, even though the small defect in the dura after lumbar puncture may not be large enough to prevent fluctuations in CSF pressure. However, in
this series, the incidence of cerebral ischemia was not different between patients with and without acute hydrocephalus despite a higher rate of risk factors for cerebral ischemia in the former group. On the other hand, when cerebral ischemia did develop, it was more often fatal among patients with acute hydrocephalus.

In conclusion, ventricular drainage produces a significant risk of ventriculitis and may increase the risk of rebleeding. In contrast, none of our patients developed ventriculitis or meningitis and the incidence of rebleeding was not increased after serial lumbar puncture. No patient deteriorated from coning resulting from lumbar puncture. Serial lumbar puncture is therefore a simple, safe, and effective way of treating acute hydrocephalus. This "conservative" treatment readily identifies those patients in whom the hydrocephalus will spontaneously regress and those who will require an internal shunt.

Acknowledgments

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References


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