Chronic subdural hematoma

Surgery or mannitol treatment

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A controlled clinical trial was planned to compare the effect of mannitol treatment with surgical intervention in chronic subdural hematoma. It was discontinued after the first seven patients showed no response to mannitol therapy. We recommend that operative intervention be considered the treatment of choice.

KEY WORDS: chronic subdural hematoma, mannitol treatment, osmotherapy

The mode of development of chronic subdural hematoma has been the subject of discussion for many years. There seems to be general agreement that it is often preceded by trivial head injury, and is most commonly due to tearing of one of the bridging veins. Proliferation of the inner surface of the dura leads to encapsulation of the collection of blood. It is a common observation that the increase in the volume of a chronic subdural hematoma is associated with increasing osmolarity of the contents, with subsequent drawing in of water from or through the membrane surrounding the hematoma. However, to date it has not been conclusively demonstrated whether osmosis is in itself sufficient to explain the growth of the hematoma, because it has been shown, particularly in children, that there is a raised albumin content and a rapid albumin turnover within these hematomas. There have nonetheless been reports not only of the treatment of chronic subdural hematoma with osmotically active compounds, but also of the spontaneous resorption of extracerebral collections of blood that had been demonstrated arteriographically. Suzuki and Takaku reported 23 consecutive patients with arteriographically demonstrated subdural collections of blood who were treated with daily doses of 1000 ml 20% mannitol intravenously for periods ranging from about 30 to 106 days; they recorded a cure in 22 of these patients. The theoretical basis for osmotherapy is that mannitol and hypertonic glucose facilitate the resorption of the hematoma through the increase in the osmotic pressure of the blood. We considered Suzuki and Takaku's investigation as a therapeutic trial with arteriographic evidence but without either control or placebo groups. We therefore planned a controlled clinical investigation with comparison of two therapeutic methods, namely, surgery and mannitol.

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Clinical Material and Method

Clinical Material

The patients included in this study had arteriographic evidence of a subdural collection of blood which had not developed acutely. The hematoma could have developed spontaneously or could have occurred more than 3 weeks after a recognized injury. The following groups of patients were excluded: children under the age of 15 years, patients in coma or with severe reduction in the level of consciousness, those with evidence of rapidly increasing intracranial pressure, and those in whom an infectious disorder (subdural empyema) was suspected. In addition we excluded all patients in whom it was considered possible that the subdural hematoma might be associated with a pathological hemorrhagic tendency. Patients accepted for the trial were given consecutive numbers and were treated by operation or with mannitol according to the random number tables to be found in the Documenta Geigy Scientific Tables. There was no further randomization.

We had planned to include about 30 patients in each treatment group in order to obtain data suitable for statistical analysis. However, as a conclusive result was obtained after the first nine patients had been treated, we terminated the trial at this stage.

Methods of Treatment

Surgical treatment consisted of craniotomy, removal of the hematoma and parts of the membrane, and closure of the dura.

Osmotic treatment was as follows: daily intravenous infusions of 1000 ml 20% mannitol solution were given over the course of 4 to 6 hours. Treatment was to be terminated if there was: 1) deterioration in the symptoms, in particular, the appearance of convulsions or signs of increasing intracranial pressure; 2) increase in the size of the subdural hematoma demonstrated by control arteriography performed on the 10th day of treatment; or 3) no arteriographic evidence of reduction in hematoma size on the 20th day. Control arteriography was thus carried out after 10 and 20 days of treatment.

Results

A total of nine patients were studied; according to the randomization, two were treated surgically while seven received mannitol therapy. Clinical details are summarized in Table 1. Three were women and six men, ranging in age from 33 to 80 years (five were over 65). As measured on the primary arteriograms, all hematomas were between 15 and 30 mm in diameter. Two patients had bilateral hematomas.

Mannitol therapy failed in all seven patients treated by this means. Two of them (Cases 1 and 4) showed evidence of increasing intracranial pressure after 2 days of treatment, and surgery was therefore carried out; one (Case 4) also developed progressive hemiparesis, which regressed after operation. Another patient (Case 5) developed generalized convulsions and was operated on after 4 days of treatment. Two (Cases 6 and 7) showed no change in the size of the

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### TABLE 1

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex, Age (yrs)</th>
<th>Hematoma Width (Arteriography) (mm)</th>
<th>Treatment</th>
<th>Reasons for Interruption of Mannitol Treatment</th>
<th>Duration of Treatment (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M 72</td>
<td>30</td>
<td>mannitol</td>
<td>increasing intracranial pressure</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>F 80</td>
<td>30</td>
<td>operation</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>M 33</td>
<td>25</td>
<td>operation</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>F 74</td>
<td>25/20 (rt/lt)</td>
<td>mannitol</td>
<td>progressive hemiparesis</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td>F 60</td>
<td>20</td>
<td>mannitol</td>
<td>grand mal convulsions</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>M 66</td>
<td>30</td>
<td>mannitol</td>
<td>unchanged hematoma after 20 days</td>
<td>20</td>
</tr>
<tr>
<td>7</td>
<td>M 65</td>
<td>15/15 (rt/lt)</td>
<td>mannitol</td>
<td>hiccough</td>
<td>9</td>
</tr>
<tr>
<td>8</td>
<td>M 56</td>
<td>25</td>
<td>mannitol</td>
<td>increase in size of hematoma</td>
<td>11</td>
</tr>
<tr>
<td>9</td>
<td>M 69</td>
<td>28</td>
<td>mannitol</td>
<td></td>
<td>2</td>
</tr>
</tbody>
</table>
Treatment of chronic subdural hematoma

hematoma in the control arteriogram taken after 20 days of treatment, and were therefore treated surgically. On the 9th day of treatment one patient (Case 8) was operated on because of distress due to intractable hiccup which had developed during the mannitol treatment, and which disappeared after operation. The last patient (Case 9) was operated on after 11 days of treatment when control arteriography the previous day had revealed that the hematoma had increased from 28 to 35 mm in diameter.

In all seven of these patients, operation revealed typical chronic subdural hematomas containing between 100 and 200 ml of old blood, within the usual membrane. The same was found in the two patients who were operated on immediately according to the trial plan.

Discussion

At the point at which the controlled clinical trial was concluded, only two patients had been included in the surgical treatment group, while seven entered the mannitol-treated group. These figures are too small to permit statistical comparisons. From general experience and reports in the literature it appears that in 90% to 95% of the cases operative treatment leads to disappearance of the hematoma. Calculation of exact confidence limits reveals that in our material the “true” percentage of failure of mannitol treatment with 95% probability was greater than 59, and with 99% probability was greater than 47. On the basis of these results, we considered it unjustifiable to continue the therapeutic trial.

Our results differ completely from those reported by Suzuki and Takaku, but the two groups of patients are not directly comparable. It is, however, noteworthy that only three of Suzuki’s 23 patients were over 65 years of age, while this was true of five of our seven patients. We are not sure whether there were differences in the mannitol solutions used, but have been informed that they were prepared differently.

Spontaneous or partial resorption of subdural collections of blood has been reported, but it is not clear whether this is also true of verified typical chronic subdural hematomas. Operative removal of a chronic subdural hematoma is well tolerated by patients and, if necessary, can be carried out under local anesthesia. It leads to definitive cure in 90% to 95% of cases. There are no reports of injurious effects of the operation itself, although Suzuki and Takaku seem to assume that these occur. It would appear that early operation would cause far less damage to the brain than long-lasting compression by the hematoma which lasts throughout the duration of the osmotic therapy. The advantages of a short hospital admission are also obvious.

On the basis of these findings we must conclude that mannitol therapy should not be considered as a replacement for surgery in the treatment of chronic subdural hematoma.

Acknowledgment

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References

11. Pudenz RH, Shelden CH: The lucite calvarium—a method for direct observation of the

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brain. II Cranial trauma and brain movement. 


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