Diagnostic method for differentiating external hydrocephalus from simple subdural hygroma

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The term “chronic subdural hygroma” was introduced by Dandy in 1932 to indicate a subdural collection of nonhematic fluid, which can be encapsulated under a thin membrane. The management of subdural fluid collection has been controversial because of the difficulty in determining whether the collection has resulted from an atrophic process, CSF sequestration, or external hydrocephalus. Although the term “external hydrocephalus” has been used to describe enlargement of the subarachnoid or subdural space in the context of increased intracranial pressure in pediatric patients, authors of a recent case report have proposed that the condition could occur in adult patients as well. We believe that the development of a subdural fluid collection after head trauma is the same radiological phenomenon but may result from various diseases such as subdural hygroma, brain atrophy, and external hydrocephalus.

A wide variety of radiological diagnostic criteria have been suggested in establishing a diagnosis of hydrocephalus. Among them, the FHI and Evan index are the most popular measuring methods; however, we think that neither of these indices adequately reflects ventricular enlargement in the context of subdural fluid collection. Furthermore, the reliability of CT scanning in determining the underlying causes of posttraumatic subdural collections—namely, simple hygroma, brain atrophy, and/or external hydrocephalus—has been inconsistent. As a result, the response to CSF shunt procedures in patients with posttraumatic external hydrocephalus has been difficult to predict, and generally the results of CSF diversion in these patients have not been encouraging.

In this prospective study, we present an analysis of our clinical experience with subdural fluid collection following mild head injury and propose a method for establishing a differential diagnosis.

Clinical Material and Methods

Patient Population

The patient sample consisted of 20 patients who had un-

Abbreviations used in this paper: CSF = cerebrospinal fluid; CT = computed tomography; FHI = frontal horn index; mFHI = modified FHI; SDH = subdural hematoma.
Computed Tomography Evaluation

Standard management involved obtaining CT studies as soon as possible at the time of presentation. Regular follow-up CT scanning was undertaken every week or at the onset of symptom progression. Ventricle size was measured by calculating an mFHI, that is, the greatest width of the frontal horns divided by the bicortical distance in the same plane, rather than by the inner table distance (Fig. 1).

Indications for Surgery and Subdural Pressure Monitoring

Bur hole trephination was performed on the appearance of a subdural fluid collection thicker than 15 mm, either persistent (that is, existing longer than 4 weeks) or increasing fluid collection on follow-up CT scanning and concomitant neurological symptoms (mental confusion, memory impairment, and headache), and no other known intracranial lesion throughout the follow-up period. During the procedure, subdural pressure was measured using a manometer before opening the dura mater. To prevent subdural fluid leakage through the puncture site, the intact dura was punctured using a 16-gauge angioneedle (Fig. 2). Subdural drainage was maintained for 3 to 5 days after the operation.

A ventriculoperitoneal shunt was inserted on the appearance of hydrocephalus symptoms after bur hole trephination and an enlarged ventricle (mFHI > 33%) on CT.

Statistical Analysis

Data are expressed as the means ± standard errors of the means. For statistical comparison, chi-square tests and unpaired t-tests were used when appropriate. Numerical differences having probability values less than 0.05 were considered to be statistically significant.

Results

Classification According to CT Findings

The mFHI was varied, ranging from 28 to 46%. Five (50%) of 10 patients with a preoperative mFHI greater than or equal to 33% suffered hydrocephalus after bur hole trephination and underwent shunt placement (p < 0.05). In contrast, among patients with an mFHI less than 33% (10 patients), none had hydrocephalus (Table 1 and Figs. 3 and 4).

Classification According to Subdural Pressure

Subdural pressure was varied, ranging from 3 to 27.5 cm H$_2$O. Hydrocephalus developed after bur hole trephination in four (80%) of five patients with subdural pressures greater than 15 cm H$_2$O and one pediatric patient (2 years old) with a pressure of 12 cm H$_2$O; these patients underwent shunt placement (p < 0.05). Note that none of the other patients whose subdural pressure was less than 15 cm H$_2$O had hydrocephalus or needed a shunt.

Neurological Outcome

No surgical complication occurred, and any neurological symptoms arising from the subdural fluid collections were improved by the operation (85%). Specifically, in the five patients with external hydrocephalus who had undergone shunt insertion, neurological symptoms completely resolved. Typical hydrocephalus symptoms and ventricular enlargement became apparent from 1 week to 2 months after bur hole trephination. Initial neurological symptoms in patients with external hydrocephalus consisted of mild confusion or memory disturbance instead of the typical hydrocephalus symptoms of gait disturbance, memory impairment, and urinary incontinence. After bur hole trephination, typical hydrocephalus did develop in these patients, but the symptoms completely resolved after shunt placement.

Discussion

“Subdural fluid collection” is a radiological term used to describe excess fluid in the subdural space, although the terms “subdural fluid collection,” “hygroma,” “effusion,” and “hematoma” have been variably utilized in many other reports in the literature about the same topic. Different causes of these fluid collections have been identified, including previous surgical procedures, head injuries, infections, congenital malformations, and craniofacial disproportions.

The mechanism of excessive subdural fluid accumulation is not yet fully understood. It has been widely held that dura and arachnoid are separated by a virtual space containing a minimal amount of fluid; this traditional concept was challenged by Caldarelli, et al., who suggested that the subdural space is a postmortem artifact. In this view, the cranial meninges in humans have no subdural space but physiologically comprise a tight layer of densely packed cells, the interface layer, composed of the innermost portion of the dura mater and the outermost portion of the arachnoid without any clear dividing line between them. A lack of cohesion between the dura and the arachnoid, due to the absence of collagen tissue or other alterations of intercellular contact with or without the interference of other factors (such as trauma, infections, and so forth), may produce a subdural space within sheets of torn dural border cells and not within a preexistent tissue compartment. In these cases, the tear in the arachnoid acts as a ball-valve device, which prevents the restoration of cohesion within the dura–arachnoid interface layer.

In this study, the initial signs of subdural fluid collection were observed as early as 7 to 8 hours after head injury and became evident by 2 weeks posttrauma; in general, however, posttraumatic subdural fluid collection is most likely to develop in the 1st month after head injury. In the present study, most of the subdural hygromas usually developed within 2 weeks after head trauma, and surgical intervention was required approximately 1 month postinjury.
External hydrocephalus is a well-defined clinical entity in infants, is characterized by macrocephaly, has a benign self-limiting clinical course, and demonstrates hypodense extraaxial fluid collections on CT. Although most cases of pediatric external hydrocephalus are idiopathic, some are associated with macrocephaly or occur as a result of trauma, infection, or venous hypertension. Infants seem to be more susceptible to fluid accumulation in the extraaxial space, most likely because of the patency of the cranial sutures, the plasticity of the immature skull, and the softness of the unmyelinated infantile brain. The factors responsible for the conversion of external into internal hydrocephalus are still unknown. Both seem associated with an underlying disturbance in CSF absorption.\(^1,4,6,11,13,17,20,26,28\)

Although radiographic similarities exist among brain atrophy, external hydrocephalus, and simple subdural hygroma, these diseases appear to be distinct clinical entities that can be clearly differentiated from one another. Yoshi moto, et al.,\(^29\) reported that in most patients with external hydrocephalus, the lateral ventricles are usually increased in size and that simple subdural hygroma occurs almost exclusively in elderly patients, whereas external hydrocephalus develops in relatively young patients. In the patients in the present study, the ventricles were enlarged, too.

Diagnosing hydrocephalus based on CT criteria was introduced by Gudeman and colleagues.\(^7\) These criteria include the appearance of distended anterior horns of the lateral ventricles and the enlargement of the temporal horns and third ventricle and concomitant normal or absent sulci. Periventricular translucency is considered as confirming evidence as well.\(^4,12,13,18\) Note, however, that the diagnosis of subdural fluid collection based on serial CT measurement of ventricle size alone would be unreliable because progressive collections are observed not only in patients with subdural hygroma but also in those with external hydrocephalus.\(^18\) In the present study, we assessed ventricular enlargement by calculating an mFHI. We believe that modifying the frontal horn index by using the bicortical distance in the same plane as the largest width of the frontal horns, instead of using the inner table distance (FHI), would better reflect ventricular enlargement in patients with subdural fluid collection.

Some authors have proposed that hydrocephalus after head trauma results in ventricular enlargement and increased intracranial pressure.\(^7,18,19,25,27\) In the present study, subdural pressure was measured using a manometer before opening the dura. The subdural pressures were varied, but in four of five patients with a pressure greater than 15 cm H\(_2\)O, hydrocephalus developed after bur hole trephination (p <
Hydrocephalus occurred in only one patient with a subdural pressure less than 15 cm H₂O: a pediatric patient (2 years of age, subdural pressure 12 cm H₂O).

The neurological manifestations of external hydrocephalus in the present study were somewhat different from those of internal hydrocephalus. Typical normal-pressure hydrocephalus symptoms resulting from cerebral parenchymal compression by the enlarged ventricles include gait disturbance, urinary incontinence, and memory impairment. In the five patients who had undergone shunt placement, different initial neurological symptoms of external hydrocephalus (not simple subdural hygroma) were observed, such as confusion, headache, and memory disturbance. Other authors have suggested that the symptomatic differences between internal and external hydrocephalus result from anatomical differences of mass lesions.

Cardoso and Schubert have suggested that extraaxial collections after trauma or subarachnoid hemorrhage, commonly referred to as “subdural hygroma,” might represent adult forms of external hydrocephalus and should therefore be monitored on repeated imaging because the form of hydrocephalus could evolve into the internal type and thus require CSF diversion. Furthermore, it is important to differentiate external hydrocephalus from chronic SDHs given that their responses to CSF shunt insertion are quite different. An SDH usually enlarges after ventricular shunt insertion, whereas external hydrocephalus generally resolves.

In the present study, after bur hole trephination, a subdural collection remained in eight patients and may have resulted from compensatory fluid collection caused by brain atrophy.

Lee and colleagues have insisted that a subdural hygroma did not resolve after bur hole trephination.

**TABLE 1**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Diagnosis</th>
<th>Adm GCS Score</th>
<th>FHI/ mFHI (%)</th>
<th>Subdural Pressure (cm H₂O)</th>
<th>GOS Score</th>
<th>6-Mo Results After Bur Hole Op</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2, M concussion</td>
<td>13</td>
<td>34/34 25/33</td>
<td>12</td>
<td>5 shunt op</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>62, M mild contusion</td>
<td>13</td>
<td>29/24 31/39</td>
<td>16.5</td>
<td>5 shunt op</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>59, M concussion</td>
<td>13</td>
<td>33/39 35/43</td>
<td>23</td>
<td>5 shunt op</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>65, M mild contusion</td>
<td>15</td>
<td>31/31 36/38</td>
<td>25</td>
<td>5 shunt op</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>63, M mild contusion</td>
<td>14</td>
<td>33/37 37/44</td>
<td>27.5</td>
<td>5 shunt op</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Subdural hygroma group**

| 6        | 36, M mild contusion | 13    | 28/28 29/30   | 3             | 5 complete resolution     |           |                               |
| 7        | 60, F mild contusion | 13    | 31/31 29/31   | 3             | 5 complete resolution     |           |                               |
| 8        | 59, M concussion     | 15    | 22/22 32/32   | 3.5           | 5 8 mm remained on lt     |           |                               |
| 9        | 74, M concussion     | 15    | 26/31 28/31   | 5             | 5 complete resolution     |           |                               |
| 10†      | 81, M concussion     | 15    | 33/38 39/46   | 7             | 5 no change               |           |                               |
| 11       | 71, M concussion     | 15    | 32/33 29/32   | 7.5           | 5 complete resolution     |           |                               |
| 12       | 2, M mild contusion | 12    | 26/26 29/31   | 8             | 4 8 mm remained on lt     |           |                               |
| 13       | 69, M concussion     | 12    | 23/34 32/39   | 8             | 4 no change               |           |                               |
| 14       | 78, F mild contusion | 15    | 25/26 27/30   | 8             | 5 no change               |           |                               |
| 15       | 58, M concussion     | 13    | 27/33 27/33   | 8.2           | 4 complete resolution     |           |                               |
| 16†      | 66, M concussion     | 15    | 24/27 26/28   | 8.5           | 5 no change               |           |                               |
| 17       | 61, M mild contusion | 13    | 33/33 33/36   | 8.5           | 5 5 mm remained on lt     |           |                               |
| 18       | 67, M concussion     | 15    | 27/29 31/31   | 10            | 5 complete resolution     |           |                               |
| 19       | 74, M mild contusion | 15    | 33/34 35/38   | 12            | 5 complete resolution     |           |                               |
| 20†      | 62, M mild contusion | 13    | 30/31 25/30   | 15            | 5 no change               |           |                               |

* Adm = admission; GCS = Glasgow Coma Scale; GOS = Glasgow Outcome Scale.
† Chronic SDH developed.

**Fig. 3.** Representative CT scans obtained in a patient with mild head injury, demonstrating various stages of a subdural hygroma. A: Initial scan of bilateral subdural hygroma. B: Follow-up scan (7 days posttrauma) of increased subdural hygroma on the right side. C: Follow-up scan (21 days posttrauma) of further enlarged subdural hygroma. D: Postoperative scan (2 months postsurgery) of subsided subdural hygroma without hydrocephalus.
groma either resolves or changes to a chronic SDH. In their studies, almost one half of the subdural hygromas resolved or were reduced in size within 3 months, whereas 61.3% of the unresolved or unreduced subdural hygromas became chronic SDHs.\textsuperscript{15,16,21,22} In the present study, after bur hole trephination, chronic SDHs developed in three patients with unresolved subdural hygromas, whereas external hydrocephalus was diagnosed and subsequently treated via shunt insertion in five patients.

We suggest that subdural fluid collection is a radiological definition comprising different diseases, such as external hydrocephalus and brain atrophy, which should be treated with different methods.

Conclusions

We believe that it is extremely important to differentiate external hydrocephalus from simple subdural hygroma after brain injury because placement of a CSF shunt could be the treatment of choice for the former, whereas this same procedure would exacerbate a hygroma. Our results suggest that ventricular enlargement measured according to an mFHI on serial CT in combination with subdural pressure monitoring during bur hole trephination can be a valuable diagnostic method to distinguish between external hydrocephalus and subdural hygroma.

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References

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