Acute Subdural Hematoma Requiring Surgery in the Subacute or Chronic Stage

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Abstract

The aim of this study was to clarify the clinical characteristics and pathophysiology of conservatively treated cases of acute subdural hematoma (ASDH) that ultimately require surgery in the subacute or chronic stage, and devise an appropriate form of management for them. A total of 50 patients with ASDH were admitted to our institution during a 5-year period. Hematoma removal in the subacute or chronic stage was performed in 8 patients. The ASDH had been caused by a fall in 5 patients. Five patients had been treated with antiplatelet agents. Fluid-attenuated inversion recovery magnetic resonance (MR) imaging demonstrated an irregularly shaped hematoma with gyrus patterns in 4 of 5 patients. Diffusion-weighted MR imaging demonstrated a two-layered hematoma structure in 3 of 4 patients. The hematoma was removed via a craniotomy, a small craniotomy, and a burr hole in 1, 1, and 6 patients, respectively. At surgery in the craniotomy case, a solid clot was located beneath the dura, and a liquid hematoma was located close to the brain. After hematoma removal, no inner membrane was observed on the brain surface. One patient had typical chronic subdural hematoma in the subacute stage, and 2 patients had so-called subacute subdural hematoma (SASDH) in the chronic stage. Although the majority of such cases can be treated by burr-hole surgery, a small craniotomy or craniotomy ought to be considered as a further surgical option if SASDH is diagnosed on the basis of clinical and radiological data, especially diffusion-weighted MR imaging.

Key words: acute subdural hematoma, subacute stage, chronic stage, surgery

Introduction

As the population of Japan ages, the number of cases of mild head trauma caused by falls or similar accidents has increased, especially in persons of advanced age.2,5 Consequently, the number of conservatively treated cases of acute subdural hematoma (ASDH) has also increased.1 In such cases, clinical deterioration with slow hematoma enlargement in the chronic stage is well known to occur as chronic subdural hematoma (CSDH), and this requires careful observation.14 However, in the subacute stage, clinical deterioration occasionally occurs associated with comparatively rapid enlargement of the hematoma, necessitating surgery, and this has recently been referred to as subacute subdural hematoma (SASDH).1,3,6,9,10,13–15

Our aim in the present study was to clarify the clinical characteristics and pathophysiology of patients with ASDH who ultimately required surgery in the subacute or chronic stage, and devise an appropriate form of management for them.

Patients and Methods

A total of 50 patients with ASDH (32 men and 18 women, aged between 12 and 92 years with a mean age of 66.5 years) were admitted to the Department of Neurosurgery, Kanmon Medical Center, during a 5-year period from April 2005 to March 2010. This study excluded patients with ASDH caused by aneurysm, arteriovenous malformation, angioma, or vasculitis because of the high risk of rebleeding in such patients. We identified those patients who showed clinical deterioration with hematoma enlargement and required surgery in the subacute or chronic stage.

From the patients’ medical records, we collected demographic and epidemiological data including details of age, sex, trauma mechanism, medical history, use of antiplatelet and/or anticoagulant agents, and presence of any systemic bleeding tendency. Clinical and radiological data were also as-

Received March 19, 2012; Accepted October 12, 2012
### Clinical data for the 8 operative cases of acute subdural hematoma in the subacute or chronic stage

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Trauma mechanism</th>
<th>Medical history</th>
<th>Antiplatelet or anticoagulant use</th>
<th>Systemic bleeding tendency</th>
<th>Interval from trauma (onset) until clinical deterioration (days)</th>
<th>Interval from trauma (onset) until MR imaging examination (days)</th>
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</thead>
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<tr>
<td>1</td>
<td>54/F</td>
<td>fall</td>
<td>schizophrenia</td>
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<td>no</td>
<td>no</td>
<td>14</td>
<td>19</td>
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<td>2</td>
<td>56/M</td>
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<td>HT, DM, HL, CI</td>
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<td>yes (antiplatelet)</td>
<td>13</td>
<td>13</td>
</tr>
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<td>84/M</td>
<td>unknown</td>
<td>CI</td>
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<td>no</td>
<td>13</td>
<td>not done</td>
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<tr>
<td>4</td>
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<td>no</td>
<td>no</td>
<td>13</td>
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<tr>
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<td>AF</td>
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<td>yes (mild)</td>
<td>21</td>
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<td>HT, CI, uAN, MI</td>
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<td>yes (antiplatelet)</td>
<td>no</td>
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<td>7</td>
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<td>primary macroglobulinemia</td>
<td>no</td>
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<td>16</td>
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<tr>
<td>8</td>
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<td>no</td>
<td>21</td>
<td>22</td>
</tr>
</tbody>
</table>

Abbreviations, see next page.

Results

Eight patients meeting the study criteria (4 men and 4 women, ranging in age from 54 to 85 years with a mean age of 69.0 years) were identified (Table 1). The remaining 42 patients comprised 12 who underwent surgery in the acute stage and 30 who were treated conservatively without surgery. Two patients (Cases 2 and 7) had no associated trauma, and neither of them had any vascular anomaly on CT angiography or MR angiography. One patient (Case 7) had mild systemic bleeding tendency caused by primary macroglobulinemia. Three patients (Cases 1, 3, and 5) showed a reduction of the GCS score from 13 or more to 12 or less and/or presence and deterioration of motor weakness, and 5 patients (Cases 2, 4, and 6–8) showed presence and deteriora-
Table 1, contd.

<table>
<thead>
<tr>
<th>MR imaging findings</th>
<th>Interval from trauma (onset) until surgery (days)</th>
<th>Operation method</th>
<th>Operative findings</th>
<th>Histological findings</th>
<th>GOS at discharge</th>
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<tr>
<td>Gyrus patterns (FLAIR)</td>
<td>Two-layered structure (DWI)</td>
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<td>Hematoma</td>
<td>Outer membrane</td>
<td>Inner membrane</td>
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<td>craniotomy</td>
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<td>15</td>
<td>small craniotomy</td>
<td>viscous liquid</td>
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<td>not done</td>
<td>16</td>
<td>burr hole</td>
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<tr>
<td>no</td>
<td>no</td>
<td>18</td>
<td>burr hole</td>
<td>liquid</td>
<td>liquid</td>
</tr>
<tr>
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<td>not done</td>
<td>19</td>
<td>burr hole</td>
<td>liquid</td>
<td>liquid</td>
</tr>
<tr>
<td>not done</td>
<td>not done</td>
<td>22</td>
<td>burr hole</td>
<td>liquid</td>
<td>liquid</td>
</tr>
<tr>
<td>yes</td>
<td>yes</td>
<td>23</td>
<td>burr hole</td>
<td>viscous liquid</td>
<td>no</td>
</tr>
<tr>
<td>yes</td>
<td>yes</td>
<td>22</td>
<td>burr hole</td>
<td>solid and liquid</td>
<td>yes (thin)</td>
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</tbody>
</table>


Fig. 2 Case 4 treated by burr-hole surgery. A–C: Computed tomography scans, on the day of trauma showing a very thin high-density left subdural hematoma (A), 7 days after trauma showing a change to iso-density (B), and 14 days after trauma showing an increase in volume with marked midline shift and a change to low density (C). D, E: Fluid-attenuated inversion recovery (D) and diffusion-weighted (E) magnetic resonance images 13 days after trauma, showing a smooth hematoma surface without gyrus patterns and no two-layered hematoma structure, respectively.

Fig. 3 Case 7 treated by burr-hole surgery after plasmapheresis. A–C: Computed tomography scans, on the day of onset showing a thin high- and iso-density left subdural hematoma (A), 1 day after onset showing a decrease in volume (B), and 13 days after onset showing an increase in volume with midline shift and a change to low density (C). D, E: Fluid-attenuated inversion recovery (D) and diffusion-weighted (E) magnetic resonance images 16 days after onset, showing an irregularly shaped hematoma with gyrus patterns and a two-layered hematoma structure, respectively.
Fig. 4 Case 1 treated by craniotomy. A, B: Intraoperative photographs. A solid clot is located beneath the dura, and liquid hematoma is located close to the brain (A). After hematoma removal, there is no evidence of an inner membrane, torn arachnoid membrane, or an injured blood vessel on the brain surface (B). C, D: Photomicrographs of the surgical specimens showing a thin outer membrane of granulation tissue between the dura and the solid clot. Hematoxylin and eosin stain, original magnification C: ×40, D: ×400.

Discussion

Based on the period from trauma until the onset of symptoms, subdural hematoma has long been categorized as ASDH (3 days or less), SASDH (from 4 to 20 days), or CSDH (21 days or more). There is a great difference between ASDH and CSDH; however, the distinction between SASDH and CSDH is unclear, especially in terms of pathophysiology. Consequently, SASDH accompanied by CSDH is sometimes reported as subacute and chronic subdural hematomas because most patients in this category are treatable by burr-hole surgery. Recently, subdural hematomas that show reduction in the acute stage but enlarge comparatively rapidly in the subacute stage in conservatively treated patients with ASDH have been referred to as SASDH. In addition, along with the aging of the Japanese population, the number of aged patients with ASDH caused by mild head trauma has also been increasing, as has the number of those patients being treated conservatively in view of their mild clinical conditions and bleeding tendency induced by antiplatelet and/or anticoagulant use. Therefore, the majority of reported clinical studies of SASDH have been conducted in Japan. SASDH has been reported to occur in approximately 10% of ASDH cases treated conservatively. In the majority of reported clinical studies of SASDH have been conducted in Japan. SASDH has been reported to occur in approximately 10% of ASDH cases treated conservatively.
present study, SASDH was found in 7 (18.4%) of 38 patients, indicating that it is not uncommon. We also found that ASDH cases treated surgically in the subacute or chronic stage showed the following features: a thin (immature) outer membrane and a liquid or viscous liquid hematoma, or occasionally a solid clot, but no inner membrane, thus possibly requiring a craniotomy or small craniotomy on occasion (Cases 1–3 and 5–8), and a hematoma with gyrus patterns and/or a two-layered structure on MR imaging (Cases 1, 2, 7, and 8); a liquid hematoma with thick (mature) outer and inner membranes, which was treatable by burr-hole surgery (Case 4), and a hematoma without gyrus patterns and a two-layered structure on MR imaging (Case 4). In addition, two (Cases 6 and 8) of the former cases (so-called SASDH) showed clinical deterioration in the chronic stage, whereas the latter (typical CSDH) showed clinical deterioration in the subacute stage.

A number of previous studies have reported the characteristics of SASDH evident on CT scan. These characteristics have included a comparatively thick high-density hematoma in the acute stage, expanding along the skull vault and including low-density components, an enlarged hematoma in the subacute stage showing a change to iso- or low density, a low prevalence of complicating traumatic lesions including brain swelling, and frequent occurrence of brain atrophy. On the other hand, the characteristics of SASDH evident on MR images have included gyrus patterns (a feature indirectly suggesting absence of an inner membrane) on the brain surface side of a hematoma, and a two-layered hematoma structure consisting of a high-intensity component (solid or jelly-like clot) and a low-intensity component (liquid hematoma) on diffusion-weighted MR images. The latter feature in particular has been reported to be useful for diagnosis and surgical decision-making in patients with SASDH. With regard to the CT density of SASDH, the present findings were similar to those of the previous studies, cerebral contusion was found in only 1 patient, and brain atrophy was evident in half of the present patients. Four patients had gyrus patterns and no inner membrane, but 1 patient lacking such patterns did have an inner membrane, suggesting that this feature reflects the characteristic absence of an inner membrane in SASDH. Intraoperatively, 3 patients in whom diffusion-weighted MR imaging demonstrated a two-layered hematoma structure were diagnosed as having SASDH, but 1 patient lacking this structure was diagnosed as having typical CSDH. In Cases 1 and 8, diffusion-weighted MR imaging was able to clearly distinguish between a solid clot and a liquid hematoma, corroborating the operative findings, and was considered to be useful for preoperative examination of subacute and chronic subdural hematomas.

SASDH may be confused with CSDH because the former can often be treated by burr-hole surgery; however, SASDH occasionally requires a small craniotomy or craniotomy. Therefore, it is important to diagnose SASDH precisely on the basis of clinical and radiological findings. In this study, 2 middle-aged patients (Cases 1 and 2) were treated using a craniotomy or craniotomy, and 5 elderly patients (Cases 3–6 and 8) and 1 patient (Case 7) with systemic bleeding tendency were treated using burr-hole surgery. None of the 8 patients required reoperation. Especially in Case 1, a comparatively massive hard solid clot beneath the dura and a liquid hematoma close to the brain were observed, but no inner membrane was evident on the brain surface intraoperatively, indicating that the solid hematoma would be difficult to be removed via a burr hole. In addition, a thin (≤1 mm) outer membrane differing from the so-called hemorrhagic outer membrane of CSDH was evident histologically between the dura and the solid clot. On the basis of the characteristic operative and histological findings in Case 1, SASDH was considered to differ from CSDH distinctly in terms of the pathogenesis of hematoma enlargement. In Case 1, a liquid hematoma, which was considered to be the major cause of a mass effect, had collected close to the brain, where an inner membrane was absent. Therefore, we considered that hematoma enlargement in Case 1 might have been caused by leakage of cerebrospinal fluid through a torn arachnoid membrane, which could not be confirmed intraoperatively. On the other hand, in Cases 2 and 7, which were both non-traumatic spontaneous SASDH, we considered that it would have been difficult for the hematoma to have enlarged as a result of cerebrospinal fluid leaking through a torn arachnoid membrane, suggesting that the pathogenesis of hematoma enlargement in patients with SASDH is not a simple mechanism.

SASDH, which is a distinct pathophysiological entity, should be separated from CSDH. However, it is possible that SASDH may show transformation to CSDH. In addition, in conservatively treated patients with ASDH, we found that typical CSDH could occur in the subacute stage and that SASDH occurred in the chronic stage. Therefore, SASDH and CSDH should be defined not according to the period from trauma (onset) until clinical deterioration but according to the pathogenesis of hematoma enlargement, especially in the context of surgical decision-making. Most patients with SASDH can be treated by burr-hole surgery. However, for patients...
with SASDH showing a thick, extensive, and hard solid clot, a small craniotomy or craniotomy is required. On the other hand, although typical CSDH can be removed via a burr hole, occasionally this might not be possible for CSDH complicated by acute hemorrhage (so-called acute-on-CSDH) or CSDH with SASDH (transformation of SASDH to CSDH).

One limitation of this retrospective study was the uneven use of MR imaging. MR (especially diffusion-weighted) imaging was not carried out for all patients, and the interval from MR imaging examination until surgery was comparatively long in Cases 4 and 7. Therefore, in order to address the relationship between MR imaging features and operative findings effectively, further prospective studies with a well-defined protocol for examination and treatment will be required. On the other hand, in Case 7, a solid and liquid hematoma might have changed to a viscous liquid hematoma during the period between MR imaging examination and surgery, and therefore we believe that any high-intensity component in a subdural hematoma evident on diffusion-weighted MR imaging is likely to be a solid clot. At present, however, it is difficult to confirm preoperatively whether a solid clot is hard.

In summary, subdural hematomas that become enlarged and require surgery in the subacute or chronic stage in conservatively treated patients with ASDH include both SASDH and CSDH. Accordingly, these patients should first be diagnosed as having subacute and chronic subdural hematomas, which are often treatable by burr-hole surgery, and then a small craniotomy or craniotomy ought to be considered as a further surgical option if SASDH is diagnosed on the basis of clinical and radiological data, especially diffusion-weighted MR imaging.

Conflicts of Interest Disclosure

The authors have no personal financial or institutional interest in any of the drugs, materials, or devices in the article. All authors who are members of The Japan Neurosurgical Society (JNS) have registered online Self-reported COI Disclosure Statement Forms through the website for JNS members.

References


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Neurol Med Chir (Tokyo) 53, May, 2013