External hydrocephalus after aneurysm surgery: paradoxical response to ventricular shunting

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Object. The authors sought to investigate the mechanisms and pathophysiological effects of subdural fluid collection after surgery for aneurysmal subarachnoid hemorrhage (SAH).

Methods. The authors retrospectively analyzed the medical records of 76 patients who had undergone craniotomy. The patients included 55 with aneurysmal SAH (SAH group) and 21 with unruptured aneurysms (non-SAH group) who were used as controls. Subdural fluid collection was more common in the SAH than in the non-SAH group (38% compared with 14%, p < 0.05). Although older patients appeared to be at greater risk for subdural fluid collection in both groups (p < 0.05), this condition developed even in relatively young patients with SAH. In the SAH group most subdural fluid collection was associated with ventricular dilation (81%), and a significant correlation was seen between fluid collection and the need for subsequent shunt placement (48% compared with 21%, p < 0.05). These results point to an association between hydrodynamic dysfunction and subdural fluid collection. The course of patients with subdural fluid collection varied from spontaneous resolution to normal-pressure hydrocephalus. Seven patients with persistent subdural collections underwent shunt placement (ventriculoperitoneal [VP] shunt in six and lumboperitoneal in one), which resulted in resolution of fluid collection in all seven.

Conclusions. The results indicate that for most patients in the SAH group, subdural fluid collection represented “external hydrocephalus” rather than simple “subdural hygroma.” Decreased absorption of cerebrospinal fluid because of SAH and surgically created tears in the arachnoid membrane communicating with the subdural space were factors in the development of external hydrocephalus. The authors believe that differentiating external hydrocephalus from subdural hygroma is extremely important, because VP shunt placement can be used to treat the former but could worsen the latter.

KEY WORDS • aneurysm surgery • external hydrocephalus • subdural hygroma • subarachnoid hemorrhage

The various terms used to describe subdural fluid collection, such as external hydrocephalus,1,3,8 subdural hygroma or effusion,11 benign subdural collection,10 and extraventricular obstructive hydrocephalus,5 reflect the confusion surrounding the entity. Although the term “external hydrocephalus” has been used to describe enlargement of the subarachnoid or subdural space in the presence of increased intracranial pressure (ICP) in pediatric patients,1,3,8 a recent case report has proposed that the condition could occur in adult patients.3 Differentiation between external hydrocephalus and simple subdural hygroma may be difficult, but the former appears to be a distinct clinical entity separable from the latter. We believe that symptomatic or asymptomatic subdural fluid collection often develops after surgery for aneurysmal subarachnoid hemorrhage (SAH). In this retrospective study, we present an analysis of our clinical experience with subdural fluid collection after aneurysm surgery and propose a treatment protocol.

Clinical Material and Methods

Patient Population

The medical records of the 76 patients who underwent craniotomy for aneurysm clipping at the Dokkyo University School of Medicine between January 1995 and March 1997 were reviewed retrospectively. Fifty-five of the patients had aneurysmal SAH (SAH group) and 21 had unruptured aneurysms (non-SAH group). The latter group was used as the control. Patients who died during their hospital stay were excluded.

Management of Aneurysms

In the SAH group, patients were assigned a Hunt and Hess’ clinical grade on the day of admission. The day of SAH was defined as Day 0. No antifibrinolytic agents were used preoperatively. Twenty-four patients underwent surgery on Day 0, 17 on Day 1, five on Day 2, two on Day 3, and seven on Days 4 through 6. The aneurysm was located in the anterior communicating artery in 19 patients (34%), the middle cerebral artery in 15 (27%), the internal carotid artery in 13 (24%), and other vessels in eight (15%). Most patients harboring aneurysms in the first three locations underwent surgery via the standard pterional approach.

Postoperative management for all patients included mild hypervolemic therapy by means of intravenous drip infusion of albuminates and low-molecular-weight dex-
Clinical summary of 76 patients with and without SFC after aneurysm surgery*

<table>
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<tr>
<th>Subgroup (no. of patients)</th>
<th>Sex (M/F)</th>
<th>Grade (I &amp; II/III/IV &amp; V)</th>
<th>Site of Aneurysm (ACoA/ICA/VA/MCA/other)</th>
<th>No. of Patients</th>
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<tr>
<td>SAH group</td>
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<td>no SFC (34)</td>
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<tr>
<td>SFC (21)</td>
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<tr>
<td>non-SAH group</td>
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<tr>
<td>no SFC (18)</td>
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<td>SFC (3)</td>
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* The data are expressed as the mean ± standard error of the mean. Abbreviations: ACoA = anterior communicating artery; H & H = Hunt and Hess clinical grade; ICA = internal carotid artery; MCA = middle cerebral artery; SFC = subdural fluid collection; — = not applicable.
† Significant differences (p < 0.05) were considered to be statistically significant.
‡ Significant difference (p < 0.05, t-test).

Data are expressed as the mean ± standard error of the mean. For statistical comparison, chi-square tests and unpaired t-tests were used when appropriate. Differences with probability values less than 0.05 were considered to be statistically significant.

Results

The incidence of subdural fluid collection postoperatively was significantly higher in the SAH than in the non-SAH group (38% compared with 14%, p < 0.05, chi-square test). A clinical summary of the 76 patients is shown in Table 1. In the SAH group, patients with subdural fluid collection were significantly older than those without (p < 0.05, t-test). No statistically significant relationships were noted between the incidence of subdural fluid collection and gender, Hunt and Hess grade, Fisher grade, or aneurysm site. In the non-SAH group, subdural fluid collections developed in three elderly patients (aged 69, 70, and 75 years old). These patients were significantly older than those without subdural fluid collection (p < 0.05, t-test).

On imaging studies, subdural fluid collection in the SAH group was characteristically associated with dilation of the lateral ventricle of both sides, despite the extraxial space-occupying lesion; such an association with internal hydrocephalus occurred in 17 (81%) of 21 patients with subdural fluid collections. In the non-SAH group the ipsilateral ventricle was compressed and smaller in size in all three cases. Preoperative CT scans obtained in these patients demonstrated brain atrophy.

In the SAH group, 48% of the patients who developed subdural fluid collection subsequently required permanent CSF diversion after development of clinical and/or radiographic findings of normal-pressure hydrocephalus (NPH) (Table 2). A significant correlation was noted between subdural fluid collection and subsequent shunt requirement (48% compared with 21%, p < 0.05, chi-square test). Of the patients requiring permanent CSF diversion in the SAH group, 10 (59%) of 17 exhibited subdural fluid collection during the initial stage postsurgery. Subdural fluid collection was not related to the GOS score.

The clinical course of subdural fluid collection is summarized in Table 3. In the non-SAH group, no subdural fluid collection produced symptoms and all resolved spontaneously (Fig. 1). In the SAH group, subdural fluid

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collection disappeared spontaneously in 10 of the 21 patients. It progressed to ventricular dilation with concomitant resolution of subdural fluid collection in three patients who developed clinical and radiographic findings typical of NPH. These patients underwent ventriculoperitoneal (VP) shunt placement. The remaining seven patients had persistent subdural fluid collection and underwent shunt placement (VP in six, lumboperitoneal in one). The subdural fluid collections disappeared during the follow-up period (range 8 days–1 year) in all patients, and the size of the ventricles decreased (Figs. 2 and 3).

Discussion

Differentiation of Subdural Fluid Collection

Subdural fluid collection is a term used to describe excess fluid in the subdural space, usually occurring after head trauma. The CSF is believed to enter the subdural space through a tear in the arachnoid membrane. Little absorption occurs in the subdural space, and the fluid collects there. The arachnoid membrane of the sylvian and chiasmatic regions is likely to tear with trauma, and a flap-valve effect could conceivably trap a significant amount of CSF. Similarly, a tear in the arachnoid membrane resulting from aneurysm surgery gives rise to postoperative subdural fluid collection.

Our clinical observations indicate that extraaxial CSF collections are commonly observed after early aneurysm surgery post-SAH (38%). Most patients in the SAH group appeared to have developed external hydrocephalus rather than simple subdural hygroma. Although the mechanisms and pathophysiological effects of external hydrocephalus are still unclear, we postulate that two key factors are important to the development of this condition: decreased CSF absorption and a tear in the arachnoid membrane. An SAH interferes with the absorption of CSF and during the initial accumulation increased ICP drives fluid through the surgically created arachnoid tear, resulting in subdural fluid collection. In some cases, epidural and subcutaneous fluid accumulation may even occur as a result of further CSF egress through the dural suture. Notably, extraaxial CSF collections did not tend to occur while continuous external drainage (cisternal, ventricular, or lumbar) was in place.

The mechanism and course of subdural fluid collection is diagrammed in Fig. 4. Although radiographic similarities exist between external hydrocephalus and simple subdural hygroma, the former appears to be a distinct clinical entity that can be clearly differentiated from the latter. First, in most patients with external hydrocephalus, when CSF accumulates in the subdural space, the lateral ventricles also usually increase in size, despite the presence of subdural fluid collection, indicating disturbed CSF absorption. Second, although our data indicate that older patients seem to be at increased risk for development of either condition, external hydrocephalus developed even in relatively young patients (the youngest was 43 years old).
old). In contrast, simple subdural hygroma occurred almost exclusively in elderly patients with significant cerebral atrophy. Craniocephalic disproportion underlies development of hygroma in this context, as in cases of infants with subdural fluid collection.

**Course and Treatment of External Hydrocephalus**

The reported incidence of acute or chronic hydrocephalus after SAH ranges from 6 to 30% in the literature. Hydrocephalus is probably caused by occlusion of CSF pathways and fibrosis of arachnoid granules, leading to dysfunction in CSF absorption. Excessive CSF accumulates in the area of least resistance within the cranial cavity, which varies between patients. Our study indicates that the CSF initially tends to accumulate over the convexities (external hydrocephalus). In some cases, a shift of CSF among intracranial compartments could occur, resulting in contralateral or interhemispheric subdural fluid collection and/or ventricular dilation. In approximately two-thirds of patients with external hydrocephalus, the collection resolved spontaneously. Three cases evolved into internal hydrocephalus with clinical symptoms of NPH, which subsequently required placement of VP shunts.

However, some patients had persistent subdural fluid collection, with clinical signs of high- or normal-pressure hydrocephalus. How should we treat these patients? Whereas some surgeons may argue that observation with serial CT scans is needed to confirm complete disappearance of subdural fluid collection before shunt placement, prolonged hydrodynamic disturbance may cause irreversible brain damage. Placement of subdural–peritoneal shunts may be useful for treating subdural fluid collection, but is not likely to provide permanent CSF diversion. Our initial concern was that placement of a VP shunt in patients with this condition might increase subdural CSF accumulation, but this did not occur in any case. In all patients in this study, the subdural fluid collection resolved and ventricular size decreased after shunt placement. This paradoxical response appears to reflect the pathophysiological conditions underlying external hydrocephalus: shunt placement relieves the increased ICP that drives CSF into the subdural space. Subdural hygroma, in contrast, is caused primarily by a vacuum effect on CSF precipitated by sinking of the atrophic brain.

**Conclusions**

We believe that it is extremely important to differentiate external hydrocephalus from simple subdural hygroma after aneurysm surgery because placement of VP shunts could be the treatment of choice for the former, whereas it would exacerbate a hygroma.

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References


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Fig. 4. Schematic drawing showing proposed pathophysiological origins and treatment of subdural fluid collections. Upper: Simple subdural hygroma usually occurs only in patients with atrophic brains. The CSF enters the subdural space through a tear in the arachnoid membrane. The ipsilateral ventricle is compressed by the extraaxial fluid collection and decreases in size. Most external accumulations resolve spontaneously. If the lesion becomes symptomatic, subdural external drainage or subdural–peritoneal (SP) shunt placement is the treatment of choice. Lower: In external hydrocephalus, defective CSF absorption increases ICP, driving CSF into the subdural space through the tear in the arachnoid membrane. Internal hydrocephalus is associated with most cases; the ventricles dilate despite the presence of subdural fluid collection. The subdural fluid collection is transient in some patients with or without progression to internal hydrocephalus (NPH). Placement of ventricular shunts in the presence of subdural fluid collection could resolve external as well as internal hydrocephalus.