Clinical Significance of Traumatic Subarachnoid Hemorrhage

Minoru SHIGEMORI, Takashi TOKUTOMI, Masaru HIROHATA, Hikaru MARUIWA, Nobuo KAKU* and Shinken KURAMOTO

Department of Neurosurgery and *Critical Care Center, Kurume University School of Medicine, Kurume, Fukuoka

Abstract

The clinical significance of traumatic subarachnoid hemorrhage (TSAH) was studied in 20 head-injured patients. They were classified into mild (9) and severe (11) groups by their initial Glasgow Coma Scale (GCS) scores. TSAH detected by computed tomography was localized in the Sylvian fissure in four of the nine mild group patients, but was also found in other basal subarachnoid cisterns in the other five. Except for one patient who developed delayed vasospasm and communicating hydrocephalus, all had favorable outcomes in this group. Massive TSAH was noted in the basal subarachnoid cisterns in 10 severe group patients and only one had a good outcome. Delayed intracerebral hematoma in the frontal or temporal lobe in the vicinity of the Sylvian fissure was found in two mild and two severe group patients with TSAH in the Sylvian fissures. Traumatic parenchymal lesions in the brainstem were minimal in one of the two autopsied patients and no evidence of diffuse axonal injury was found in both cases. Thus, TSAH in the Sylvian fissure is suggestive of focal brain contusion around the fissures. Massive TSAH in the basal subarachnoid cisterns is not necessarily associated with severe parenchymal injury of the brainstem.

Key words: traumatic subarachnoid hemorrhage, computed tomography, brainstem injury, diffuse brain damage

Introduction

Traumatic subarachnoid hemorrhage (TSAH) is a common autopsy finding in patients with head injuries. In clinical patients, massive TSAH found on computed tomographic (CT) scans may be associated with severe, diffuse brain damage including the brainstem and the outcome of these patients is often unfavorable. In the present study, we investigated the clinical features, outcomes, and histological findings in patients with TSAH to determine its clinical significance.

Materials and Methods

The subjects of this study were 20 TSAH patients, who had no space-occupying lesions on initial CT scans. Their ages ranged from 5 to 77 years and averaged 43.1 years. No patients had serious multiple injuries. The patients were classified into a mild group with initial Glasgow Coma Scale (GCS) scores of 13 or more (9 patients with a mean age of 45.8 years) and a severe group with initial GCS scores of 7 or less (11 patients with a mean age of 40.9 years). The CT findings and outcomes were compared for these two groups.

TSAH detected on CT scans was evaluated at the pontine-interpeduncular, the ambient-quadrigeminal, and the suprasellar cisterns and the Sylvian fissure. The degree of TSAH was then classified into three grades according to the density on CT scans: mild (+), with a faint layer of blood; moderate (++) with a thin layer of blood; and massive (+++) with a thick layer of blood. The outcome was evaluated according to the Glasgow Outcome Scale scores at 3 months after injury. Good recovery and moderate disability were classified as good and severe disability and persistent vegetative state as poor. Autopsies were performed on two of the eight deceased patients and their brains including the brainstem were histologically examined.
Results

TSAH was found in the Sylvian fissures and/or the suprasellar cistern in the nine mild group patients (Table 1). Only three patients had TSAH in the pontine-interpeduncular and/or the ambient-quadrigeminal cisterns. The outcomes in this group were good for eight patients and poor for one. Delayed intracerebral hematomas were found in the temporal or frontal lobe in the vicinity of the Sylvian fissures in two patients. Delayed vasospasm and communicating hydrocephalus developed in one patient (Case 4) with TSAH in the pontine-interpeduncular, ambient-quadrigeminal, and suprasellar cisterns (Fig. 1).

The GCS scores on admission were 5 or less in eight of the 11 severe group patients (Table 2). Ten patients showed clinical evidence of primary brainstem injury, such as decerebrate posturing and abnormal ocular signs. Moderate to massive TSAH were found in the pontine-interpeduncular and/or ambient-quadrigeminal cisterns in eight patients. Six of the eight deceased patients had TSAH in all the cisterns evaluated in this study, and two patients with poor outcomes had TSAH in three major sites. Only one patient (Case 10) with localized TSAH in both the suprasellar cistern and Sylvian fissure had a good outcome. The degree of TSAH in the suprasellar cistern and Sylvian fissure did not influence the outcome. However, TSAH in the pontine-interpeduncular and ambient-quadrigeminal cisterns indicated a poor outcome. Follow-up CT scans showed delayed intracerebral hematoma in two patients (temporal lobe in one and frontal lobe in the other on the side of TSAH in the Sylvian fissure).

Table 1 Summary of nine mild group patients with TSAH

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Sex</th>
<th>GCS score</th>
<th>Degree of TSAH on initial CT</th>
<th>Finding of subsequent CT</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Pontine-interpeduncular cisterns</td>
<td>Ambient-quadrigeminal cisterns</td>
<td>Suprasellar cistern</td>
</tr>
<tr>
<td>1</td>
<td>35</td>
<td>M</td>
<td>15</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>54</td>
<td>M</td>
<td>15</td>
<td>+</td>
<td>#</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>73</td>
<td>F</td>
<td>15</td>
<td>#</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>62</td>
<td>F</td>
<td>15</td>
<td>+</td>
<td>#</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>40</td>
<td>M</td>
<td>14</td>
<td>#</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>37</td>
<td>F</td>
<td>14</td>
<td>#</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>13</td>
<td>M</td>
<td>13</td>
<td>#</td>
<td>#</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>39</td>
<td>M</td>
<td>13</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>59</td>
<td>F</td>
<td>13</td>
<td>+</td>
<td>#</td>
<td></td>
</tr>
</tbody>
</table>

Marked brain swelling was noted in bilateral cerebral hemispheres in one patient.

Histological study in one patient (Case 17) revealed small hemorrhagic lesions in the dorsal brainstem (Fig. 2). However, the brainstem lesions were minimal in Case 18 (Fig. 3). There were no macroscopic lesions in the corpus callosum or other cerebral white matter and no evidence of axonal retraction balls or microglial stars in these two patients.  

Discussion

TSAH is reported to be caused by the rupture of small vessels in the pia-arachnoid when the brain is moved under the dura mater by external force during injuries or by hemorrhage associated with cortical contusions. TSAH is a common autopsy finding in severe head-injured patients. It is often difficult to identify TSAH over the convexity areas by CT scanning because of an artifact due to the skull, although TSAH in the basal subarachnoid cisterns and Sylvian fissure is often demonstrated. TSAH in the basal subarachnoid cisterns has been considered to indirectly indicate primary brainstem injury in many patients. In the present study, TSAH was mainly located in the Sylvian fissures or suprasellar cistern in the mild group patients. On the other hand, moderate to massive TSAH was predominantly found in the pontine-interpeduncular and ambient–quadrigeminal cisterns in the severe group patients. Follow-up CT scans showed delayed hematoma in the temporal or frontal lobe on the same side as TSAH in the Sylvian fissure. Therefore, TSAH in the Sylvian fissure is suggestive of focal cortical contusion in the vicinity. Accordingly,
such patients should be kept under strict neurological and CT observation.

One patient developed delayed vasospasm and communicating hydrocephalus. Several authors have already reported post-traumatic vasospasm. Aruga et al. reported three cases of cerebrovascular narrowing associated with severe head injuries. Since all these patients showed marked cerebral swelling and intracranial hypertension, they proposed intracranial circulatory insufficiency associated with intracranial hypertension as the cause of such vasospasm. Suwanwela and Suwanwela reported that no TSAH was found in some patients with narrowing of the major basal arteries occurring soon after head trauma. Therefore, not only TSAH but also mechanical injuries in the vascular wall may be responsive for post-traumatic vasospasm. In addition, Wilkins stressed the importance of neurogenic factors due to hypothalamic injuries as the cause of post-traumatic vasospasm. In our patient, however, did not show any evidence of hypothalamic injuries. It is well known that vasospasm is closely related to the degree of SAH in patients with ruptured cerebral aneurysm. Since a full study with cerebral angiography was not performed in our series, it is not known whether massive TSAH was frequently associated with arterial narrowing or not. It is plausible that the delayed vasospasm in our case was induced by the same mechanism as ruptured aneurysm because there was no evidence of acute intracranial hypertension or hypothalamic injury.

Primary brainstem injuries have been considered to be associated with diffuse axonal injury in many patients. However, hemorrhagic lesions in the brainstem were rarely detected on CT scans in patients with clinical signs of primary brainstem injury. Accordingly, TSAH in the basal subarachnoid cisterns is considered an important finding suggesting primary brainstem injury. Hosaka et al. reviewed CT scans of 27 primary brainstem injuries and found the isodense brainstem in 12 cases and TSAH around the brainstem in 10. In the present study, clinical evidence of primary brainstem injury was present in 10 patients with massive TSAH around the brainstem. Histological examination in one patient certainly disclosed focal hemorrhagic lesions in the brainstem. However, lesion was minimum in the other autopsy patient. In addition, lesions in the corpus callosum as well as axonal retraction balls or microglial stars, characteristic to diffuse axonal injury, were not found in these two patients. Therefore, massive TSAH in the basal subarachnoid cisterns should be treated as an independ-

Fig. 3 Case 18. A: Gross pathological appearance of the basal brain with hemorrhage. B: HE-stained section of the midbrain, showing minimal hemorrhage in the tegmentum near the aqueduct (arrows). × 100. C: Luxol fast blue-stained sections of the brainstem, showing no significant abnormalities.
ent category of diffuse brain injury. Nakamura stated that from experimental studies in monkeys primary brainstem hemorrhage was not easily induced even by lethal cerebral injuries and death was presumably caused by cardiorespiratory insufficiency due to severe brainstem dysfunction. The death of patients with TSAH in the basal subarachnoid cisterns is not necessarily associated with parenchymal injury of the brainstem, but rather with severe brainstem dysfunction.

References

5) Freytag E: Autopsy findings in head injuries from blunt forces. Arch Path (Chicago) 75: 74-85, 1963

Address reprint requests to: M. Shigemori, M.D., Department of Neurosurgery, Kurume University School of Medicine, 67 Asahi-machi, Kurume, Fukuoka 830, Japan.