Chronic Subdural Hematoma May be Preceded by Persistent Traumatic Subdural Effusion

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Abstract

The incidence of traumatic subdural effusion (TSE) was analyzed to clarify the relationship with subsequent chronic subdural hematoma (CSH) in 500 patients with head injury evaluated over a 36-month period. TSE occurred in 108 patients (21.6%), and CSH developed in 29 (5.8%) of these. The incidence of TSE was high, although only hospitalized patients were included because of the necessity for serial computed tomography. TSE frequently developed into CSH, and all CSH followed TSE. Therefore, TSE is closely associated with CSH and subdural effusion is probably a preliminary stage necessary for the formation of CSH.

Key words: subdural effusion, chronic subdural hematoma, computed tomography, head injury, subdural hygroma

Introduction

Traumatic subdural effusion (TSE) or traumatic subdural hygroma is an accumulation of cerebrospinal fluid (CSF) between the dura and the arachnoid membrane caused by trauma, which Mayo first reported in 1894 using the term "subdural hydroma," although McConnell subsequently proposed the terms "subdural effusion" or "subdural fluid collection" irrespective of cause. TSE was previously thought to be rare compared with chronic subdural hematoma (CSH). However, computed tomography (CT) has revealed that the incidence of TSE is much higher. Recently, a relationship between TSE and CSH has been proposed. In this study, the incidence of TSE was analyzed and clarified its relationship with CSH.

Materials and Methods

This study included 500 consecutive patients with head injury (363 males and 137 females) admitted to Shimada Municipal Hospital between January, 1989 and December, 1991 and followed by serial CT. TSE occurred in 108 patients (21.6%); the diagnosis was chiefly based on the CT finding of a crescent-shaped hypodense region in the cerebral convexity. Thirteen patients with this indication of TSE persisting without CT changes for several months but remaining asymptomatic had been excluded because differentiation from brain atrophy or preceding subdural hygroma (effusion) was difficult. CSH occurred in 29 patients (5.8%), and not in any of the excluded ones. The diagnosis of CSH was based on operative findings in 16 cases and only CT scans in 13.

The age distribution, sex ratio, site and severity of head injury were analyzed. The site of head injury was divided into five categories: frontal, temporal, parietal, occipital, and multiple. Patients with an unknown site of head injury were excluded. The duration of consciousness disturbance and skull fracture are generally accepted as an index of the severity of head injury. The cases were classified into two types: Type A, no neurological deficit despite a brief loss of consciousness following head injury; and Type B, consciousness disturbance continuing for more than 1 hour, and/or some neurological deficits following head injury.

The differences between the whole series, TSE and CSH groups were tested with the \( \chi^2 \) test. In the case of significance, results between individual groups were compared by the \( \chi^2 \) test with modified level of significance according to the Bonferroni method.
Results

Patients with TSE tended to be older (3–90 yrs; mean, 61.9 yrs) than all head injury patients (0–90 yrs; 41.6 yrs). Patients aged over 60 years made up 34.0% of the whole series, 63.9% for the TSE group and 69.0% for the CSH group. The majority of patients with TSE or CSH were aged over 60 years, but there was no significant difference between the two groups. The percentage of males in the series (72.6%) did not significantly differ from that in the TSE group (78.7%).

Figure 1 shows a comparison of the site of head injury. In the whole series and the TSE group, head injury in the frontal and occipital regions was frequent. If the frontal and occipital injuries are combined into a single category (head injury in the sagittal direction), the percentage of patients did not significantly differ for all patients (73.2%) and the TSE group (74.4%). Thus, there was no relationship between the occurrence of TSE and site of the head injury. A similar result was also obtained for the CSH group.

The percentage of Type A injury, 88.8% for all patients, 90.7% for the TSE group and 93.1% for the CSH group, did not significantly differ. The incidence of skull fracture also did not significantly differ between all patients (31.8%), the TSE group (39.8%) and the CSH group (31.0%).

Illustrative Case Reports

Case 1: A 61-year-old male fell off a moving motorcycle in an accident with a car and struck the frontal region of his head. He sustained brief loss of consciousness after the accident, but on admission his consciousness was clear without evidence of focal neurological deficit. The initial CT scans revealed no abnormalities (Fig. 2A). Ten days later, however, follow-up CT scans revealed an obvious subdural effusion on the left (Fig. 2B). He showed no neurological deterioration. Repeat CT scans 25 days after the accident showed decreased effusion (Fig. 2C). Twenty-eight days later, no effusion persisted on CT scans (Fig. 2D).

Case 2: A 17-year-old male fell off a motorcycle in an accident with a car and struck his occipital region. On admission, CT scans showed no abnormal findings (Fig. 3A). Twelve days later, repeat CT scans showed small bilateral subdural effusions (Fig. 3B). The serial CT scans showed that the effusions became smaller and disappeared after 72 days (Fig. 3C).

Case 3: A 77-year-old male was hit by a car while riding a bicycle and was struck on the parietal region. The initial CT scans on admission showed small bilateral subdural effusions (Fig. 4A). The next day, CT scans revealed that the effusions had increased (Fig. 4B). However, CT scans on the 14th day showed reduction of the effusions. Thirty days after the injury, serial CT scans showed that the right subdural effusion had become hyperdense,
leading to the diagnosis of subdural hematoma (Fig. 4C). The hyperdense lesion reduced and disappeared (Fig. 4D). No symptoms were noted during the course.

Case 4: A 51-year-old male struck his head against the windshield of his car during an accident with another car. The CT scans on admission disclosed small bilateral subdural effusions (Fig. 5A). CT scans on the 16th day showed slightly increased effusions (Fig. 5B). Sixty days after the injury, serial CT scans showed a marked CSH on the left (Fig. 5C). He complained of headache and right hemiparesis, so a craniotomy was performed to evacuate the hematoma. The postoperative course was uneventful.

Case 5: A 53-year-old female was hit by a car while walking on the street and suffered injury to the occipital region. She had suffered left hemiparesis caused by cerebral embolism following surgery for mitral valve stenosis 1 year before. The initial CT scans on admission showed a small acute subdural hematoma (Fig. 6A). CT scans on the following day showed a subdural effusion but the acute subdural hematoma had disappeared (Fig. 6B). The CT density of this effusion was higher than that of CSF. Two weeks later, follow-up CT scans showed that the effusion showed the same density as CSF (Fig. 6C). Two months after the injury, the subdural effusion became a hematoma (Fig. 6D). This case is an exceptional one. The acute subdural hematoma was thought to have been diluted by CSF.

Discussion

The diagnosis of TSE is complicated by the need for differentiation from brain atrophy and CSH. The diagnosis of TSE is possible based only on CT scans if serial CT scans are available. TSE occurs several hours to 1 week after head injury, so
the diagnosis of TSE was only made after chronological CT observations from the early to chronic stage after head injury.\textsuperscript{5,14,27} In contrast to previously reported incidences of TSE (3–6.6%), this study found a high incidence (21.6%), although only hospitalized patients could be examined because of the necessity for serial CT scanning.\textsuperscript{3,5,22,27,30} This higher incidence for TSE is chiefly attributable to the availability of improved CT which allows definite diagnosis even in patients such as Case 2. The analysis of the age distribution showed a higher incidence of TSE in the elderly (in particular > 60 yrs). However, the age distribution did not significantly differ between the TSE and CSH groups.

Various mechanisms for the development of TSE have been proposed: 1) a tear in the arachnoid membrane,\textsuperscript{3,22,27,33} 2) effusion through vessels damaged by trauma in the dura mater, arachnoid or parenchyma,\textsuperscript{22,27,34} 3) an osmotic pressure mechanism, and 4) extension from the subdural hematoma.\textsuperscript{7,22,23,26,27,32,35,38} At present, the first hypothesis is thought the most likely, because many tears in the arachnoid membrane have been found during operation.\textsuperscript{18,22,26} Arachnoid tear is likely to occur in the Sylvian fissure or chiasmatic region due to coup or contrecoup injury because the arachnoid membrane adheres strongly to the sharp sphenoidal wing.\textsuperscript{3,22} In this study, however, the severity and site of head injury did not correlate with TSE or CSH.\textsuperscript{29} Some investigators support the second hypothesis based on the delayed onset of TSE (\(\geq 1\) wk after head injury) and the frequent highly proteinaceous quality of subdural effusion fluid.\textsuperscript{5,22,30,35} This study found no delayed onset (\(\geq 1\) wk after head injury) in any patient. In past reports, most TSE cases were clinically “silent” and tended to resolve spontaneously.

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**Fig. 5** CT scans in Case 4. A: On the first day after injury, showing small bilateral subdural effusions. B: Sixteen days after injury, the effusions had slightly increased. C: Sixty days after injury, showing a subdural hematoma on the left.

**Fig. 6** CT scans in Case 5. A: On the first day after injury, showing a small acute subdural hematoma (arrow) on the right. B: On the following day, showing a subdural effusion and disappearance of the acute subdural hematoma. The density of this effusion was slightly higher than the CSF density. C: Two weeks after injury, the subdural effusion showed the same density as CSF. D: Two months later, the subdural effusion had changed into a subdural hematoma.
without surgical treatment.\textsuperscript{5,18,22,26,27,35,36} In this study, only two TSE patients required surgical treatment.

Twenty-nine (26.9\%) of 108 patients with TSE developed CSH in this study. Although the natural history of CSH is almost completely known, the etiology is still controversial.\textsuperscript{15,19-21,25,28,24,37} CSH is usually classified into two groups: traumatic and non-traumatic (no history of head injury). Various cases of non-traumatic CSH have been reported. The etiology of CSH is difficult to identify from the various factors. The presence of blood in the subdural space may not be a sufficient condition for progression to CSH, and other factor(s) may be necessary.\textsuperscript{1,9,24,31} Attempts to induce CSH in experimental animals were not successful until 1972, when Watanabe et al.\textsuperscript{31} produced a clinical form of subdural hematoma by inoculating a blood clot mixed with CSF into the subdural space of dogs and monkeys. Two years later, Apfelbaum et al. concluded that CSF is unnecessary for the formation of membrane around a subdural hematoma, but could not reproduce or explain the growth of a subcutaneous implant of blood and CSF mixture to several times the original size. Membrane very similar to that of CSH was also formed in TSE (in particular \(\geq 3\) wks after head injury).\textsuperscript{1,3,11,12,22,21,25}

This study found that TSE frequently developed into CSH and further that all cases of CSH had preceding TSE. The author's institute is the only neurosurgical institution in Shimada City, which allows a continuous follow-up of patients with head injury once registered. Therefore, it seems likely that TSE is closely associated with CSH. Although no cases of non-traumatic subdural hematoma have been studied, I suggest that subdural effusion is an essential preliminary stage for the formation of traumatic CSH, and conclude that CSF is not necessary for the formation of the CSH membrane, but is required for expansion of CSH.

References

3) DaCosta DG, Adson AW: Subdural hydroma. \textit{Arch Surg (Chicago)} 43: 559-567, 1941
9) Gardner WJ: Traumatic subdural hematoma with particular reference to the latent interval. \textit{Arch Neurol Psychiat} 27: 847-858, 1932
20) Nakamura N: The relationship between head injuries and chronic subdural hematoma. \textit{No To Shinkei} 18:


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