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ROLE OF TRAUMATIC SUBDURAL FLUID COLLECTION IN DEVELOPING PROCESS OF CHRONIC SUBDURAL HEMATOMA

BY

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ABSTRACT

We investigated the role of traumatic subdural fluid collection in the genesis of chronic subdural hematoma (CSDH) in 43 consecutive patients with traumatic subdural fluid collection. Traumatic subdural fluid collection was found in 43 (6%) of 715 patients who underwent CT scans because of head injury. Twenty-four of the 43 patients were more than 65 years of age and this problem occurred in 30% of the 79 head-injured patients over 65 years of age. In 20 (Group 1) of the 43 patients CSDH subsequently developed. During the same period, 30 other patients were treated for CSDH. Twenty-four (Group 2) of these 30 patients had a history of head injury more than two weeks prior to the developing sings and symptoms and had hyper- or iso-dense hematomas on their first CT scan. It may have been that they too first had a traumatic subdural collection. Forty to sixty percent of the patients with asymptomatic traumatic subdural fluid collection may develop CSDH, and this may occur especially in the older patients. Careful observations after the head injury are particularly important in the aged because of the frequent occurrence of traumatic subdural fluid collection and the subsequent development of CSDH.

Key words: Chronic subdural hematoma, CT scan, Head injury, Subdural fluid collection

INTRODUCTION

Chronic subdural hematoma (CSDH) was initially noted by Virchow and thought to be due to "pachymeningitis hemorrhagica interna". Many other investigations on its causal mechanism were also performed clinically and pathologically (Trotter [1]; Nakamura [2]; Watanabe *et al.* [3]; Ito *et al.* [4, 5]; Markwalder [6]). Since CT scan became available, its development has been studied in more detail. In 1979, Yamada *et al.* [7] first reported three cases in which the CSDH developed following traumatic subdural hygroma (posttraumatic low-density subdural fluid collection). Afterwards several other reports appeared (Hyodo *et al.* [8]; Miyazaki *et al.* [9]; Fujioka *et al.* [10]; Koizumi *et al.* [11]; Naito *et al.* [12]; Takahashi *et al.* [13, 14]). However, it is not certain how often this occurs.

We therefore reviewed 43 consecutive patients with traumatic subdural fluid collection and 30 other patients with CSDH.

MATERIALS AND METHODS

Between August 1980 and March 1985,

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among the 715 patients who had CT examinations because of head injury in the Fujiyoshida City Hospital, Yamanashi Prefecture, 43 patients had traumatic hypodense subdural fluid collection soon after the head injury. The first CT scan was always taken within six days after the head injury. All patients with subdural collection had serial CT scans. Patients who developed CSDH following subdural fluid collection after the head injury were classified as Group 1 of CSDH.

During the same period, 30 other patients with CSDH were seen who had clinical signs and symptoms and hyper- or iso-dense extracerebral lesions on their first CT scan. Twenty-four of these patients had a history of head injury more than two weeks prior to the onset of the signs and symptoms. These 24 patients were classified as Group 2.

We compared the characteristics between Group 1 and Group 2 and studied their similarity. The CSDH is basically defined as a subdural blood collection encapsulated with outer and inner membranes. About one-third of the patients in Group 1 had no operation, and most patients were diagnosed as CSDH when a hyper- or iso-dense area developed on CT scan usually more than two weeks after the head injury.

Results

Traumatic subdural fluid collection was found in 43 (6%) of 715 patients who underwent CT scans because of head injury (Table 1). Twenty-four patients were more than 65 years old (an incidence of 30% in 79 patients over 65 years old), whereas 19 patients were 64 years old or less (an incidence of 3%).

The CSDH developed in 20 (47%) of the 43 patients with subdural collection, while the other 23 patients had a spontaneous resolution of their collection. Thirteen of the 24 patients more than 65 years old developed CSDH as did seven of the 19 younger patients (Table 1).

There were 18 males and two females in Group 1, whose average age was 65 years (range, 27 to 84) (Table 2). The cause of the head injury was traffic accident in 13 patients. Fourteen patients had loss of consciousness for less than six hours. Traumatic subdural collection was detected on CT scan within ten days after the injury in 17 patients (average, three days). Traumatic subdural fluid collection developed bilaterally in 15 patients. Nineteen patients who were asymptomatic were followed with serial CT examinations. One patient had an external drainage of subdural fluid on the 50th day after the injury

Table 1. Development of traumatic subdural fluid collection and CSDH, associated with age

	Aged (≧65 years old)	Younger (<65 years old)	Total
Head-injured patients examined by CT scan	79	636	715
Patients with traumatic subdural collection	24	19	43
Patients with a change of subdural collection to CSDH	13	7	20

Abbreviation: CSDH=chronic subdural hematoma.

Table 2. Summary of clinical data of 20 cases followed by periodical CT scans (Group 1)

Case	Age	Sex	Side of LDA*	Cause of Head Injusy	Grade#	Accompanied Lesion	Interval from Injury to 1st Finding of HD	Side of HD	Signs and Symptoms	Op	Outcome
1	64	M	R (0)	motorcycle accident	II	cerebral contusion	18D	R	headache	+	cured
2	63	M	B (0)	automobile accident	II	–	69D	L	headache	+	cured
3	82	M	B (5)	bicycle accident	II	–	87D	R	syncope, headache	+	cured
4	67	M	L (1)	motorcycle accident	II	R-thin AEDH & ICH, Fx	27D	L	–	–	cured
5	38	M	B (9)	automobile accident	III	bloody CSF	91D	L	–	–	cured
6	80	M	B (0)	motorcycle accident	II	–	126D	L	–	–	cured
7	41	M	B (44)	pedestrian**	III	cerebral contusion	126D	R	demented	+	NC
8	84	M	L (5)	motorcycle accident	II	bloody CSF	67D	L	–	–	dead
9	69	M	B (1)	pedestrian**	II	cerebral contusion, Fx	117D	B	headache	+	cured
10	73	M	B (2)	tumble	II	–	44D	B	headache, vomiting	+	cured
11	70	M	B (6)	fall	II	ICH in occipital lobe	92D	B	gait dist.	+	cured
12	70	M	B (4)	bicycle accident	II	–	51D	R	headache, gait dist.	+	cured
13	66	F	B (1)	pedestrian**	III	–	56D	R	–	–	cured
14	56	M	B (7)	tumble	II	L-thin AEDH	27D	L	–	–	cured
15	72	M	B (2)	motorcycle accident	II	L-thin ASDH & SAH	27D	L	–	–	cured
16	82	M	B (0)	tumble	I	–	123D	B	gait dist.	+	cured
17	76	F	L (15)	tumble	I	–	43D	L	gait dist.	+	cured
18	50	M	B (12)	fall	II	–	52D, 75D	B	headache	+	cured
19	68	M	B (0)	fall	II	–	38D, 45D	B	headache	+	cured
20	27	M	L (0)	automobile accident	III	–	87D	L	–	–	cured

* Interval (days) from head injury to first appearance of low density area on CT in parentheses.
** Injured as pedestrian by traffic accident.
# Grading of head injury: I indicates no loss of consciousness (LOC) at head injury; II, LOC less than 6 hours; III, LOC more than 6 hours.
Abbnreviations: HD=high density; LDA=low density area; Op=operation; B=bilateral; R=right; L=left; C=cure; NC=no change; D=days; Fx=fracture of the skull; AEDH=acute epidural hematoma; ASDH=acute subdural hematoma; ICH=intracerebral hematoma; SAH=subarachnoid hemorrhage; CSF=cerebrospinal fluid; dist.=disturbance; M=male; F=female.

because of the prolonged impairment of consciousness. No outer or inner membrane was present at the time of the operation. He finally developed CSDH 76 days after surgery. The CSDH developed between 18 and 126 days after the head injury (average interval, 68 ± 34 days) (Fig. 1). Twelve of the 20 patients showed an increase in the size of the extracerebral lesion and, they also suffered signs and symptoms including headache, vomiting or gait disturbance and therefore were operated on. Seven of the remaining eight patients had a spontaneous resolution of the hematoma. One patient died of an accident during the observation. In nine of 15 patients with bilateral frontal hypodense areas, the hypodense area disappeared on one side and CSDH developed on the side with a persisting subdural collection. The other six developed CSDH on both sides. In two of these patients, the CSDH developed at different times on each side (Cases 18 and 19). Five patients with unilateral subdural collection showed an increased density on

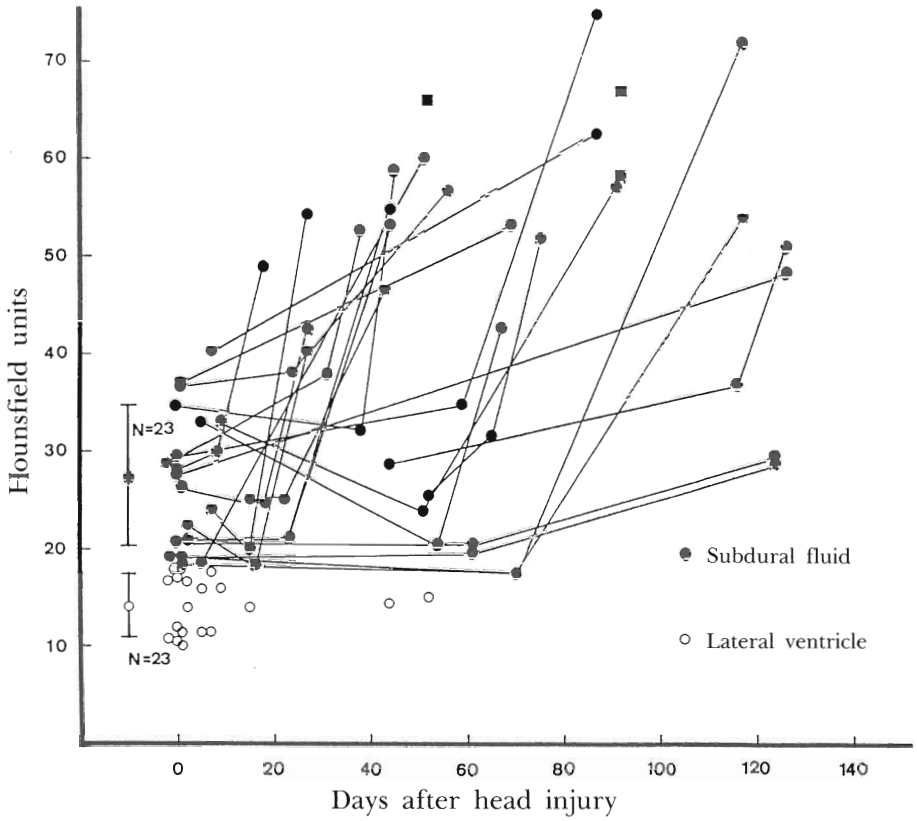

Fig. 1. Change of Hounsfields Units of Subdural Fluid Following Head Injury in Group 1. Only the side where the change of density took place is presented (26 sides in 20 patients). The Hounsfield units of the hyperdense areas are delineated with black squares in Cases 11 and 18, because those of the hypodense areas were not available. If more than three CT scans were obtained, the Hounsfield units of the subdural fluid just prior to the density increase are also shown. The Mean±S.D. of the Hounsfield units of the lateral ventricles (open circle) and subdural fluid (filled circle) at the first CT scan are shown (n=23).

the same side. In two of the 20 patients, lumbar puncture yielded bloody cerebrospinal fluid (CSF). Four patients demonstrated frontal lobe contusion and two had a single intracerebral hematoma. There were also hyperdense areas indicating contusion which was considered a contrecoup lesion on CT scan in seven of the 43 patients with traumatic subdural fluid collection.

The subdural collection which later developed CSDH had a density measurement initially of 27.1±6.7 (Hounsfield units, mean±S.D., n=23), while the ventricles were 14.3±3.0. There was a statistical significance between the Hounsfield units of the hypodense areas and those of the ventricles by the paired t-test ($p<0.01$). Three sides in two patients were not measured.

There were 19 males and five females in Group 2, with an average age of 67 (range, 40 to 82). All patients had a history of head injury, and the interval from the injury to the onset of the clinical signs and symptoms was between 18 and 80 days (average, 50±19 days). The date of head injury was not clear in one patient. The major cause

Table 3. Comparison of clinical data between Group 1 and Group 2

	No. of cases	Male/Female	Age	Interval (Days) from Injury to appearance of HD* on CT (G 1) or Symptoms (G 2)	Cause of Head Injury	
Group 1	20	18/2	65±16 (20)**	68±34 (22)**	traffic accident others	13 7
Group 2	24	19/5	67±11 (24)	50±19 (23)	traffic accident others	2 22

* Abbreviation: HD=high density.

** Mean±S.D.. There is no statistically significant difference between Group 1 and Group 2 ($p>0.1$). No. of cases in parentheses. Two cases showed bilateral changes of the density at different intervals in Group 1. One case is excluded in Group 2 because the date of head injury is not clear.

of head injury was a tumble, and 13 patients had no loss of consciousness at the time of the injury. The other 11 patients had loss of consciousness for less than six hours at the time of the injury. Hyperdense areas were seen unilaterally in 21 patients and bilaterally in three patients on the first CT scan. Twenty-two patients were treated surgically.

The other six patients with CSDH (12% of the total CSDH cases), without a past history of head injury, were three male children and three females. A 60-year-old female was under going hemodialysis. Cerebral angiography in four patients demonstrated no vascular abnormalities. All six patients were treated with surgery, and an inner membrane was identified in four.

The patients in Group 1 and Group 2 were similar in age and in the interval from the time of injury to the development of CSDH. However, the cause of the injury was different and the injury in Group 1 was more severe than that in Group 2. The severity of the injury and the medicolegal aspect of the traffic accident caused the Group 1 patients to seek treatment early and to obtain a CT scan (Table 3).

Discussion

Frontal extracerebral hypodense areas seen on CT scan after the head injury usually indicate a traumatic subdural fluid collection, when they change in size in the subsequent period or show higher Hounsfield units than the CSF in the lateral ventricles as shown in all of our patients. In several patients, this was confirmed by performing CT cisternography. The occurrence of traumatic subdural fluid collection is seen in 4 to 6.6% of the head-injured patients (Takahashi *et al.* [13]; French *et al.* [15]). In our series, the incidence (6%) was not different from that of the other authors. There was a higher incidence of subdural fluid collection in the older (over 65 years old) patients than in the younger ones.

The transformation of traumatic subdural fluid collection to CSDH is reported to have occurred in 4% to 58% of the patients (Yamada *et al.* [16]; Takahashi *et al.* [13]; Koizumi *et al.* [11]; Hyodo *et al.* [8]; St. John *et al.* [17]) (Table 4). On the other hand, Stone *et al.* [18] and French *et al.* [15] found no such changes among the 80 and 13 patients, respectively. In our series, 47% of 43 patients with subdural collection developed CSDH. The reason for the difference in the incidence among these reports is not clear. However, in the series of Stone *et al.* [18], not all patients had CT examinations and surgical intervention

Table 4. Incidence of transformation of traumatic subdural fluid collection to chronic subdural hematoma

Author, year	No. of Patients with TSDC	No. of Patients who developed CSDH following TSDC
French *et al.* (1978)	13 (0)*	0
Yamada *et al.* (1980)	24 (9)	12 (7)*
Hyodo *et al.* (1980)	24 (?)	2 (0)
Takahashi *et al.* (1981)	26 (13)	15 (7)
Koizumi *et al.* (1981)	38 (20)	4 (4)
St. John *et al.* (1981)	25 (11)	1 (1)
Stone *et al.* (1981)	80 (?)	0
Ohno *et al.* (1986)	43 (27)	20 (15)

Abbreviations: TSDC=traumatic subdural fluid collection; CSDH=chronic subdural hematoma.
* No. of patients more than 60 years old in parentheses.

was usually done early. In the series of French *et al.* [15], the average age of 13 patients was 26 years, much younger than the patients in the Japanese reports (Hyodo *et al.* [8]; Koizumi *et al.* [11]; Takahashi *et al.* [13]; Yamada *et al.* [16]). A high incidence of the development of traumatic subdural fluid collection in the elderly was noted and the development of CSDH following subdural collection also occurred much more frequently in the elderly in the Japanese reports, as shown in Table 4. In our series, 27 of 43 patients with subdural collection and 75% of the 20 patients who then developed CSDH were over 60 years old. Although careful follow-up is indicated in all patients with asymptomatic traumatic subdural fluid collection, this is particularly important in the aged.

Our results showed that there was no general clinical difference between Group 2 in which the developing process was not followed and Group 1 followed by CT scan. Since the cause of the injury in Group 2 was mainly a tumble or fall without loss of consciousness and the injuries were relatively less severe, patients did not visit our clinic to undergo CT scan until the development of clinical signs and symptoms. If they had had CT examination early after the injury, the frontal extracerebral hypodense area might have been recognized. Twelve patients with clinical signs and symptoms (the other eight patients were asymptomatic) in Group 1 showed the same clinical course and signs and symptoms as the patients of Group 2. Namely, patients, who were diagnosed as CSDH angiographically or operatively before the introduction of CT scan, may correspond to the symptomatic patients in Group 1 and all the patients in Group 2, and an asymptomatic CSDH proved to be present in the CT era.

Thus, it seems reasonable to interpret that the development of CSDH following traumatic subdural fluid collection reflects the developing process of nearly all CSDH patients with an episode of head injury. Namely, it is likely that the development of CSDH is usually preceded by the occurrence and the persisting existence of traumatic subdural collection as follows: Subdural fluid collection occurs in the frontal region soon after the head injury. Some fluid collection resolves spontaneously without developing signs and

symptoms and in some it changes to CSDH with a hemorrhage from the neomembrane (as shown by Group 1), although what difference exists between them remains unknown. Some of those who developed CSDH show spontaneous resolution, while the others with CSDH become symptomatic as equivalents of all patients of Group 2 and part of Group 1. Therefore, patients with spontaneous resolution of asymptomatic subdural collection or CSDH may form a common and large denominator. Considering the results in this study, as there were 12 symptomatic patients in Group 1 among the 43 patients with traumatic subdural fluid collection, three to four times as many potential candidates for CSDH as symptomatic patients are estimated.

The Hounsfield units of the frontal hypodense areas were significantly higher than those of the lateral ventricles, as shown in Group 1. In three of the 20 patients in Group 1, they were as high as 40 and two patients showed bloody CSF by lumbar puncture. Seven of the 43 patients with subdural collection revealed hyperdense areas on the frontal and/or temporal lobes indicating contrecoup lesions on CT scan. According to Stone *et al.* [18],j 53 of 57 patients with posttraumatic subdural hygroma furnished bloody CSF. These findings indicate that the subdural fluid is not merely the CSF but CSF mixed with blood to various extent (DaCosta *et al.* [19]).

Although the mechanism of subdural collection of blood and CSF is not clearly shown, there are a few explanations such as "flap valve action" via the arachnoid tear of the Sylvian fissure (DaCosta *et al.* [19]; French *et al.* [15]; Stone *et al.* [18]) and subdural effusion for delayed collection of subdural fluid (Stone *et al.* [18]). However, since, as described above, subdural fluid collection may often accompany the cerebral contusion and occurs highly in the aged with a tendency of brain atrophy, it is conjectural that blood and CSF flow from the subarachnoid space through the arachnoid tear, probably at the contusional site or the bridging site of the veins on the parietofrontal cerebral surface, and collect by the "flap valve action" (Koizumi *et al.* [11]).

About a one-half of the patients with traumatic subdural fluid collection showed a spontaneous resolution relatively soon after the head injury in our follow-up study by CT scan. However, the other one-half in which the subdural fluid collection persisted for a long time had a density change in the subdural fluid by the hemorrhage from the neomembrane (Group 1), although it remains unknown how the hemorrhage occurred and when the inner membrane was formed. Among the 20 patients with such a density change (Group 1), eight patients showed a spontaneous resolution of CSDH during the observations, without any clinical symptoms. Accordingly, although a change in the hypodnese area to the hyper- or iso-dense area causes a serious condition to be treated on the one hand, such a change also seems to be a healing process as a natural history of long-persisting traumatic subdural fluid collection on the other hand.

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