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Relations among Traumatic Subdural Lesions

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Acute subdural hematoma(ASDH), chronic subdural hematoma(CSDH) and subdural hygroma(SDG) occur in the subdural space, usually after trauma. We tried to find a certain relationship among these three traumatic subdural lesions in 436 consecutive patients. We included all subdural lesions regardless of whether they were main or not. We evaluated the distribution, age incidence and interval from injury to diagnosis of these lesions, and the frequency of new subdural lesions in each lesion. ASDH constituted 68.6 %, SDG 15.8 %, and CSDH 15.6 %. Age incidence of CSDH was similar to that of SDG, but differed from that of ASDH. Mean interval from injury to diagnosis was 0.4 days in ASDH, 13.4 days in SDG, and 51.6 days in CSDH. Focal brain injuries accompanied in 37.5 % of ASDH, 5.8 % of SDG, and no CSDH. In ASDH, 2 recurrent ASDHs, 17 SDGs and 9 CSDHs occurred. In SDG, 3 postoperative ASDHs and 8 CSDHs occurred. In CSDH, 2 postoperative ASDHs, 2 SDGs and 1 CSDH occurred. These results suggest that the origin of CSDH is not only ASDH, but also SDG in upto a half of cases. SDG is produced as an epiphenomenon by separation of the dural border cell layer when the potential subdural space is sufficient. A half of CSDHs may originate from ASDHs. ASDH may occur in CSDH by either a repeated trauma or surgery. Such transformation or development of new lesions is a function of a premorbid condition and the dynamics between the absorption capacity and expansile force of the lesion.

Key Words : *Acute subdural hematoma, Chronic subdural hematoma, Subdural hygroma, Pathogenesis, Computed tomography, Head injury*

INTRODUCTION

Acute subdural hematoma(ASDH), chronic subdural hematoma (CSDH) and subdural hygroma(SDG) occur in the so-called subdural space, usually after trauma. Although the etiology and location of these lesions are common, they have quite different clinical and radiological features. Traditionally subdural hematoma has been classified as acute, subacute, and chronic according to the time interval. Such a classification implies that CSDH is derived from ASDH. Some(Gade et al., 1990 ; Cooper, 1993 ; Hughes and Cohen, 1993) have postulated that slow leakage of blood into the subdural space from a torn bridge vein might cause CSDH in the aged. No one has clearly

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proven such assumptions, however. Recently, some-(Dolinskas et al., 1979 ; Zimmerman and Bilaniuk, 1984 ; Takahashi et al., 1985 ; Adams, 1990 ; Poljakovic et al, 1991) have questioned such assumptions. Poljakovic et al(1991) said that CSDH was not the last stage of an "old" ASDH.

Although repeated microhemorrhages play an important role in the enlargement of CSDHs(Markwalder, 1981), the origin of CSDH is still obscure(Gudeman et al., 1989 ; Adams, 1990 ; Bullock and Teasdale, 1990 ; Cooper, 1993). CSDH differs from SDG in many aspects, such as the contents of subdural fluid, radiological appearance, and clinical symptoms. However, an absolute distinction between SDGs and CSDHs is often difficult, since the subdural fluid within SDG is frequently a mixture of blood and cerebrospinal fluid(McLaurin et al., 1990), and SDGs not infrequently become CSDHs(Hyodo et al., 1980 ; Yamada et al., 1980 ; Koizumi et al., 1981 ; Takahashi et al., 1981 ; Kopp, 1988, 1990 ; Park HB et al., 1990 ; Shimoji et al., 1992 ; Whang et al., 1993 ; Lee HW et al., 1994 ; Lee KS et al., 1994, Park CK et al., 1994). Although these three traumatic subdural lesions have their unique characteristics, there should be some relationships among them. We attempted to delineate these three lesions and tried to find the relationship.

MATERIALS AND METHODS

We reviewed the charts of 2,500 consecutive cases of head injury from 1989 to 1993, and collected 436 patients(17.4 %) with traumatic subdural lesions. We included all subdural lesions regardless of whether they were main or not, but we excluded tentorial, interhemispheric, or infratentorial subdural lesions. All traumatic subdural lesions were diagnosed by computed tomography(CT). Various clinical features were retrospectively collected. CT scans were re-evaluated.

We divided these patients into ASDH(within 7 days), CSDH(over 7 days), and SDG groups according to the first subdural lesion. We evaluated the distribution, age incidence and interval from injury to diagnosis of these lesions, and frequency of new subdural lesions in each lesion.

RESULTS

Distribution of traumatic subdural lesions

According to the first subdural lesion, 299(68.6 %) patients had ASDH, 68(15.6 %) patients had CSDH, and 69(15.8 %) patients had SDG(Table 1). Overall 44 patients had two subdural lesions at different time. Postoperative ASDH, new SDG, and new CSDH were found in 7, 19, and 18 patients, respectively. Including those lesions, ASDH shared 63.8 %(306 cases), CSDH 17.9 %(86 cases), and SDG 18.3 %(88 cases).

Age incidence of traumatic subdural lesions

Age incidence of CSDH was similar to that of SDG, but differed from that of ASDH(Fig. 1). ASDH was commonly found in 21-60 years of age. In CSDH and SDG, more than half were over 51 years of age. Age distribution of 44 patients with two different subdural lesions was similar to those of SDG and CSDH. Even the sex ratio of CSDH(M : F=4.7 : 1) was similar to that of SDG(4.8 : 1), but differed from that of ASDH(2.9 : 1)(Table 1).

Interval from injury to diagnosis

Mean interval from injury to diagnosis was 0.4 ± 1.2 days in ASDH, 13.4 ± 21.9 days in SDG, and 51.6 ± 39.2 days in CSDH(Table 1).

CT findings

Focal brain injuries accompanied in 112(37.5 %) patients with ASDH. In SDG, only four(5.8 %) patients had associated focal brain injuries. In CSDH, none had associated focal brain injuries(Table 1).

Traumatic subdural lesions were bilateral in 4

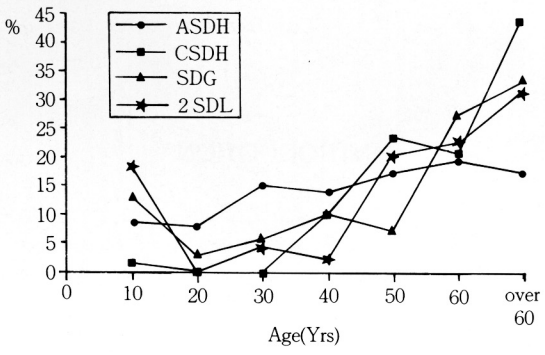

Fig. 1. Age incidences of traumatic subdural lesions(percentage in each lesion). ASDH=acute subdural hematoma ; SDG=subdural hygroma ; CSDH=chronic subdural hematoma ; 2 SDL=patients with two subdural lesions.

Table 1. Comparison of three traumatic subdural lesions.

Characteristics	ASDH(%)	SDG(%)	CSDH(%)
Total	299(68.6)	69(15.8)	68(15.6)
Sex			
Male : Female	2.9 : 1	4.8 : 1	4.7 : 1
Interval(Injury-CT ; in day)			
mean	0.4	13.4	51.6
SD	1.2	21.9	39.2
Associated FBI			
intra-axial	80(26.8)	4(5.8)	0(0.0)
extra-axial	32(10.7)	0(0.0)	0(0.0)
GCS on admission			
mean	8.7	11.3	13.5
SD	4.5	3.5	2.9
Cause of Head injury			
Slip/Fall	68(22.8)	3(4.3)	20(29.4)
Ped. TA	100(33.4)	26(37.7)	7(10.3)
Pass. TA	97(32.4)	35(50.7)	7(10.3)
Unknown	18(6.0)	3(4.3)	25(36.8)
Others	16(5.4)	2(2.9)	9(13.2)
Bilaterality	4(0.7)	52(75.4)	4(5.9)
Outcome			
Favorable	135(45.2)	44(63.8)	61(89.7)
Unfavorable	38(12.7)	21(30.4)	5(7.4)
Death	126(42.1)	4(5.8)	2(2.9)
New subdural lesions			
ASDH	2(0.7)	3(4.3)	2(2.9)
SDG	17(5.7)		2(2.9)
CSDH	9(3.0)	8(11.6)	1(1.5)

ASDH=acute subdural hematoma ; SDG=subdural hygroma ; CSDH=chronic subdural hematoma ; SD=standard deviation ; FBI=focal brain injury ; GCS=Glasgow Coma Score ; Ped. TA=pedestrian traffic accident ; Pass. TA=passenger's traffic accident

cases(0.7 %) of ASDH, in 4 cases(5.9 %) of CSDH, and in 52 cases(75.4 %) of SDG(Table 1).

Causes of injury and Glasgow Coma Score on admission

Traffic accidents were responsible for 197 cases(65.8 %) of ASDH and 61 cases(88.4 %) of SDG. In CSDH, traffic accidents occupied only 20.6 %(14 cases), and the most common cause of injury was unknown in 36.8 %(25 cases)(Table 1). The mean Glasgow Coma Score on admission was 8.7 in ASDH, 11.3 in SDG, and 13.5 in CSDH(Table 1).

Management and course

From 1987, we tried to treat small ASDHs conservatively when the thickness of the hematoma was smaller than the skull bone thickness(about 3 mm in the printed CT film or 1 cm in true thickness). Rationale and details of our management have been reported elsewhere(Lee KS et al., 1987, Lee KS et al., 1987 ; Park JK et al., 1990 ; Lee KS et al., 1992 ; Lee KS et al., 1994). In 110(36.8 %) of 299 patients with ASDH, we removed the hematoma by immediate surgery. In 110 operated cases, seven new subdural lesions(2 cases of recurrent ASDH, 2 cases of SDG and 3 cases of CSDH) occurred(Fig. 2). We managed the remaining 189(63.2 %) patients with ASDH conservatively, at first. Twenty-five in 189 conservatively managed patients required surgery due to either enlargement of the hematoma(14 cases) or delayed new lesions(7 SDGs and 4 CSDHs). In 164 conservatively managed patients, 10 new subdural lesions(8 cases of SDG, and 2 cases of CSDH) occurred(Fig. 2). Fifty-seven(34.8 %) patients in 164 conservatively managed patients expired. Finally 107 patients(35.8 %) recovered without surgery. Overall, 2 recurrent ASDHs(0.7 %), 17 SDGs(5.7 %) and 9 CSDHs(3.0 %)

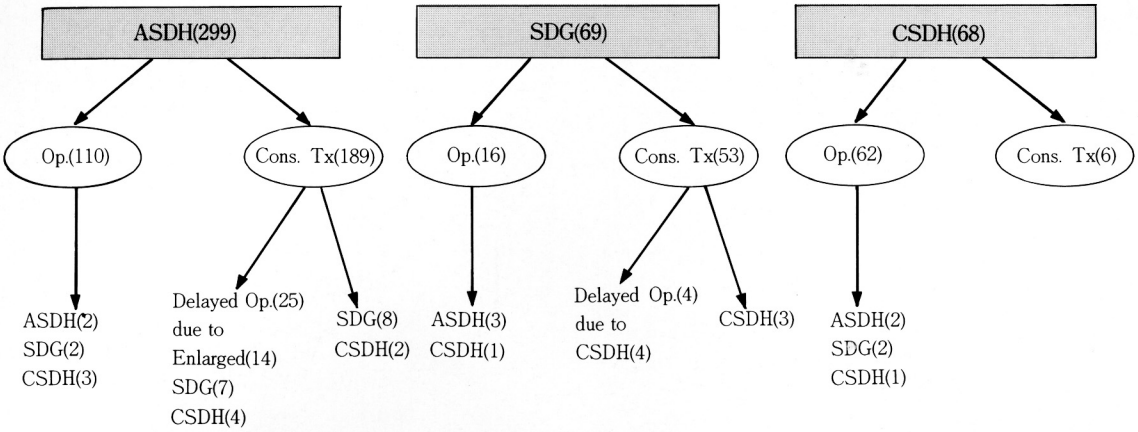

Fig. 2. Management and fate of traumatic subdural lesions. ASDH=acute subdural hematoma ; SDG=subdural hygroma ; CSDH=chronic subdural hematoma ; Op=operation ; Cons. Tx=conservative treatment.

occurred in ASDH(Table 2). The mean age of patients with immediate surgery was 43.3(SD 16.8), that of patients with delayed surgery was 41.9(SD 27.8), and that of conservatively managed patients was 40.2(SD 21.1) years of age.

For SDGs, we operated only when the size of the lesion enlarged in serial CT scans. Surgical intervention was done in 16(23.2 %) patients. In 16 operated cases, four new lesions(3 cases of recurrent ASDH and 1 case of CSDH) occurred(Fig. 2). At first, we managed conservatively for 53 patients with SDG. Four in 53(7.5 %) conservatively managed patients required surgery due to subsequent development of CSDHs. In 49 conservatively managed patients, three cases of CSDH occurred(Fig. 2). Overall, three postoperative ASDHs(4.3 %) and eight CSDHs(11.6 %) occurred in SDG(Table 2).

In CSDH, 62(91.2 %) patients underwent operations. In 62 operated cases, five new lesions(2 cases of postoperative ASDH, 2 cases of SDG and 1 case of recurrent CSDH) occurred(Fig. 2). In five patients(7.4 %), ASDH with CSDH was found with two different episodes of trauma. Initial conservative management was successful for 6 patients(8.8 %) with CSDH. None of them required surgery. Overall, two postoperative ASDHs(2.9 %), two SDGs(2.9 %) and one CSDHs(1.5 %) occurred in CSDH(Table 2).

In ASDH, the site of new subdural lesion was the same as the site of the initial lesion in all recurrent ASDH, and six of nine CSDHs, but only two of seventeen SDGs. In SDG, it was different in all ASDH, but the same in all CSDHs. In CSDH, it was different in all ASDH and one of two SDGs.

DISCUSSION

ASDH, CSDH, and SDG are traumatic lesions of the subdural space. Although these three lesions have their own unique characteristics, an absolute distinction is difficult in certain situations. Temporal classification of the subdural hematomas by interval is quite confusing and arbitrary(Teasdale and Galbraith, 1981). The intervals used to distinguish between acute, subacute, and chronic vary widely according to the author. An absolute distinction between SDGs and CSDHs is not always possible, since the subdural fluid within SDG is frequently a mixture of blood and cerebrospinal fluid(McLaurin et al., 1990), and SDGs not infrequently become CSDHs(Hyodo et al., 1980 ; Yamada et al., 1980 ; Koizumi et al., 1981 ; Takahashi et al., 1981 ; Park HB et al., 1990 ; Shimoji et al., 1992 ; Whang et al., 1993 ; Lee HW et al., 1994 ; Lee KS et al., 1994, Park CK et al., 1994). A significant number of transitional lesions exist between ASDH and CSDH, or between SDG and CSDH. Thus, there should be some relations among them. We suggest here that the relationships among these three traumatic subdural lesions are as in Fig. 3.

ASDH usually results from tear of bridge veins or cortical vessels, or cortical laceration after trauma(Bullock and Teasdale, 1990). ASDH is considered to be the most important cause of death in severely head

Table 2. Summary of Cases with two different subdural lesions.

No.	Age	Sex	SDL1(side)	ASL1	SDL2(side)	ASL2	I-D1	I-D2	Treatment	GOS
1	65	M	ASDH(R)	NO	ASDH(R)	NO	3	5	CO, CO	D
2	28	M	ASDH(L)	NO	ASDH(L)	HC	0	0	CE, Cons	D
3	79	M	ASDH(L)	AIR	SDG(B)	VE	0	24	Cons, Cons	SD
4	5	M	ASDH(L)	NO	SDG(B)	NO	0	6	Cons, BH	MD
5	49	M	ASDH(L)	NO	SDG(B)	NO	0	2	Cons, BH	SD
6	1	F	ASDH(R)	HC	SDG(B)	NO	0	13	Cons, Cons	SD
7	75	F	ASDH(R)	NO	SDG(B)	NO	0	6	Cons, Cons	MD
8	55	M	ASDH(R)	NO	SDG(B)	NO	0	37	Cons, BH, SD-P	SD
9	52	M	ASDH(L)	NO	SDG(B)	NO	0	28	Cons, SD-P	SD
10	1	F	ASDH(R)	NO	SDG(B)	VE	0	26	Cons, SD-P, BH	VS
11	1	M	ASDH(R)	NO	SDG(B)	NO	0	2	Cons, Cons	SD
12	49	M	ASDH(R)	HC	SDG(R)	NO	0	50	Cons, Cons	SD
13	44	M	ASDH(R)	NO	SDG(L)	HC	0	12	Cons, Cons	MD
14	59	F	ASDH(L)	NO	SDG(R)	NO	0	22	CE, SD-P	VS
15	52	M	ASDH(L)	NO	SDG(R)	NO	0	15	Cons, Cons	MD
16	50	M	ASDH(R)	NO	SDG(L)	NO	0	5	Cons, Cons	SD
17	7	M	ASDH(R)	NO	SDG(B)	NO	0	24	Cons, SD-P	SD
18	69	M	ASDH(R)	NO	SDG(L)	NO	0	35	CE, Cons	D
19	68	M	ASDH(L)	NO	SDG(L)	NO	0	21	Cons, Cons	D
20	54	M	ASDH(R)	ICH	CSDH(R)	NO	1	15	CO, CO	D
21	72	M	ASDH(R)	NO	CSDH(R)	NO	2	21	Cons, BH, Cons	GR
22	26	M	ASDH(R)	NO	CSDH(R)	NO	0	15	Cons, Cons	MD
23	1	M	ASDH(L)	NO	CSDH(B)	VE	2	15	Cons, BH, SD-P	MD
24	63	M	ASDH(L)	HC	CSDH(L)	NO	1	12	Cons, BH	MD
25	48	M	ASDH(R)	HC	CSDH(L)	NO	0	22	CO, BH	D
26	70	M	ASDH(R)	HC	CSDH(R)	NO	0	13	Cons, BH	MD
27	55	M	ASDH(L)	NO	CSDH(L)	NO	0	23	CO, Cons	D
28	8	M	ASDH(R)	NO	CSDH(L)	NO	0	15	Cons, Cons	SD
29	48	F	SDG(R)	NO	ASDH(L)	NO	72	88	SD-P, CO	VS
30	54	M	SDG(B)	NO	ASDH(L)	NO	?	?	BH, CO	GR
31	73	M	SDG(B)	NO	ASDH(L)	NO	17	19	BH, CO	D
32	80	F	SDG(B)	NO	CSDH(B)	NO	3	74	BH, Cons	SD
33	45	M	SDG(R)	NO	CSDH(B)	NO	38	110	BH, BH	GR
34	65	M	SDG(B)	NO	CSDH(L)	SDG(R)	1	82	BH, Cons	MD
35	72	M	SDG(B)	NO	CSDH(L)	NO	3	79	Cons, Cons	GR
36	53	M	SDG(B)	ICH	CSDH(R)	SDG(L)	4	91	Cons, Cons	SD
37	1	M	SDG(B)	NO	CSDH(L)	SDG(R)	11	115	BH, Cons	SD
38	37	M	SDG(B)	NO	CSDH(L)	NO	26	107	BH, Cons	SD
39	56	F	SDG(B)	NO	CSDH(L)	NO	13	77	Cons, Cons	SD
40	70	M	CSDH(R)	NO	ASDH(L)	NO	?	?	BH, CO	GR
41	74	M	CSDH(R)	NO	ASDH(L)	NO	30	35	BH, CO	VS
42	57	M	CSDH(R)	NO	SDG(R)	NO	33	60	CE, BH	SD
43	50	M	CSDH(L)	NO	SDG(R)	NO	38	42	BH, Cons	GR
44	48	M	CSDH(L)	NO	CSDH(L)	NO	?	?	BH, Cons	SD

SDL1=first subdural lesion ; SDL2=second subdural lesion ; ASL1=associated lesion in the first CT ; ASL2=associated lesion in the second CT ; R=right ; L=left ; B=bilateral ; I-D1=interval from injury to diagnosis of first subdural lesion ; I-D2=interval from injury to diagnosis of second subdural lesion ; GOS=Glasgow Outcome Scale ; ASDH=acute subdural hematoma ; SDG=subdural hygroma ; CSDH=chronic subdural hematoma ; HC=hemorrhagic contusion ; ICH=intracerebral hematoma ; VE=ventricular enlargement ; Cons=conservative treatment ; CO=craniotomy ; CE=craniectomy ; BH=burr hole drainage ; SD-P=subduroperitoneal shunt ; GR=good recovery ; MD=moderate disability ; SD=severe disability ; VS=vegetative state ; D=death

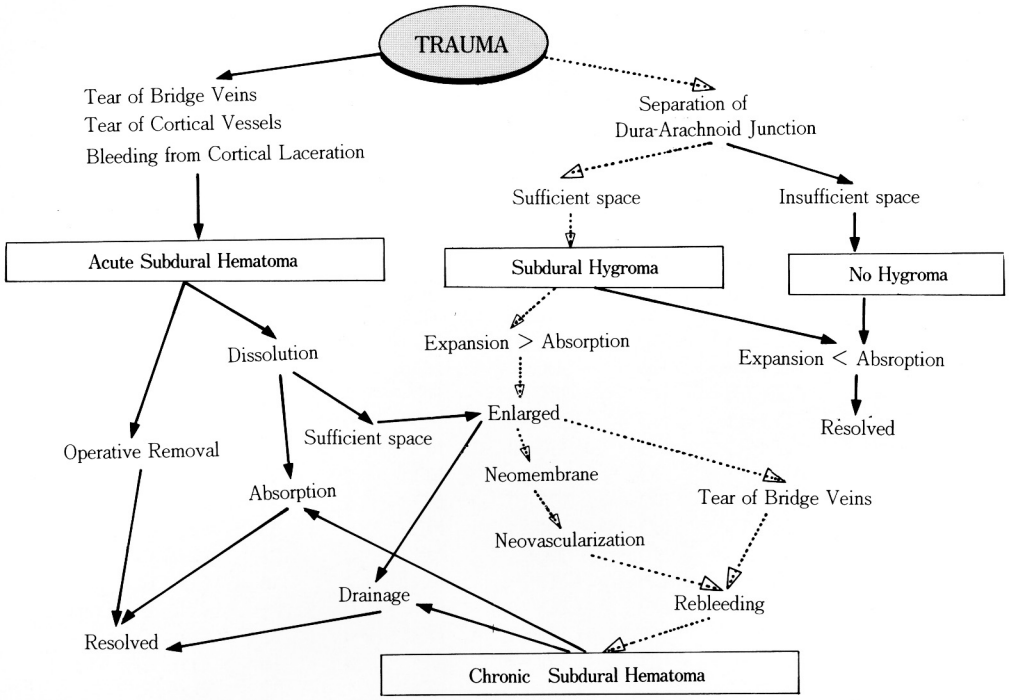

Fig. 3. Schematic representation of relationships among traumatic subdural lesions.

injured patients due to high incidence, high mortality, and head injury severity(Gennarelli and Thibault, 1982). Most ASDHs act as a mass lesion, but, not infrequently ASDH is an epiphenomenon of diffuse axonal injury or hemispheric swelling(Lobato et al., 1988 ; Sahuquillo-Barris et al., 1988 ; Cooper, 1993 ; Gennarelli, 1993). If the ASDH acts as an expanding mass, the victim will expire unless it is removed surgically. If not, the hematoma will eventually resolve. The natural history of unoperated ASDH is not well known. Most of the ASDH will resolve into liquefied clot within 4-6 weeks(McCormick, 1985 ; Adams, 1990), and will be absorbed. Resolution of ASDH without surgery is expected to be common in young healthy adults with relatively small ASDH. However, small ASDHs in young adults do not progress to a CSDH(Dolinskas et al., 1979). If CSDH results from ASDH, the age incidence of CSDH should be similar to that of ASDH. The age incidence of CSDH was more similar to that of SDG in this study, however. Contrary to ASDH, CSDH and SDG rarely had associated focal brain injuries. The severity of head injury is most often severe in ASDH, while it is mild or trivial in CSDH. ASDH is almost always unilateral, while CSDH is often bilateral.

If there is a sufficient subdural potential space either by cortical atrophy or loss of brain tissue after trauma, absorption will be delayed and a neomembrane of subdural hematoma will be formed. After the 10th day, the formation of membrane enclosing the hematoma has been observed(Frowein and Firsching, 1978). Subsequent enlargement of CSDH is caused by repeated microhemorrhages from neomembrane(Markwalder, 1981). By this route, however, only 3 % of ASDH changed into CSDH in this study.

The origin of SDG was a matter of debate, too(Bullock and Teasdale, 1990 ; Cooper, 1993). Now, we believe that separation of the dura-arachnoid interface with sufficient potential subdural space is the important precondition for SDG. We have reported the proposed pathogenesis of SDG elsewhere(Lee KS et al., 1994). So-called subdural space is not present in normal conditions, but a minute trauma could separate the dura-arachnoid interface, the so-called dural border cell layer(Sachenmayr and Friedman, 1978 ; Haines et al., 1993). Any pathologic condition induc-

ing cleavage of tissue within the dural border cell layer can induce proliferation of dural border cells with production of neomembrane(Friedman and Sachenmayr, 1978 ; Sachenmayr and Friedman, 1978 ; Haines et al., 1993). Once neomembrane is formed, hyperpermeable capillaries will be followed with time-(Hasegawa et al., 1992). If the absorption takes time, hemorrhage into the subdural fluid would occur either by tearing of bridge veins or bleeding from neomembrane. The majority of SDG are asymptomatic(Lee KS et al., 1994) and most SDG will disappear when the brain expansion or absorption exceeds effusion(Kopp, 1988 ; Lee KS et al., 1994). Kopp(1988) reported that more than 85 % of SDG were resolved within 3 months. In the opposite situation, it will be enlarged and may cause delayed recovery, or rarely clinical deterioration. The complexity of SDG depends on the variety of premorbid condition, the dynamics between absorption and expansion, duration of observation, and indication and rate of surgery, besides variety of the primary head injury in lesional types and severity. We expect that a significant number of CSDH will be produced by this mechanism.

Nearly a half of patients with CSDH have no history of head injury(Bullock and Teasdale, 1990 ; Cooper, 1993), and CSDHs are seen only uncommonly following severe brain injury(Gudeman et al., 1989). These facts imply that the injury itself is trivial or minor. Such a trivial injury, however, can produce a SDG, a potent precursor of CSDH.

Although there is general agreement that repeated microhemorrhages are responsible for the enlargement of chronic SDHs, still the origin of CSDH is obscure(Gudeman et al., 1989 ; Adams, 1990 ; Bullock and Teasdale, 1990 ; Cooper, 1993). Poljakovic et al.(1991) investigated the histopathological features of subdural hematomas and concluded that CSDH was not the last stage of an "old" acute SDH. They insisted that CSDH and ASDH were different entities, considering their etiopathogenetic and clinical pictures, and especially their CT and histopathological appearances. We found that CSDH was similar to SDG instead of ASDH in this study. SDG and CSDH occur most often at the extremes of life(Zimmerman and Bilaniuk, 1984 ; Duhaime and Sutton, 1992). Even the age distribution of 44 patients with two different subdural lesions was similar to those of SDG and CSDH. These distributions suggest premorbid conditions for development of these lesions. In the infant, the brain is quite compressible and in the elderly, brain atrophy leads to potential space, where any fluid can be easily collected(Duhaime and Sutton, 1992). We found that 11.6 % of SDG changed into CSDH, in this study. Such a transformation is not rare. Ohno et al(1987) reported that the incidence of CSDH from SDG was as high as 58 %. The incidence of 11.6 % in this study seems to be low. However, since more than 85 % of SDG will resolve within 3 months spontaneously(Kopp, 1988), it means more than 70 % of unresolved SDG. Mean interval from injury to diagnosis is short in ASDH, intermediate in SDG, and long in CSDH. This difference in intervals also supports our supposition. The occurrence rate of CSDH from SDG(11.6 %) was about four times higher than that(3 %) of ASDH. However, the prevalence rate of ASDH was over four times higher than that of SDG. Overall number of CSDH from SDG(8 cases) was similar to those from ASDH(9 cases).

ASDH may be produced from CSDH by a repeated trauma or operative removal of CSDH. Patients with CSDH are prone to another accident. CSDH with recent bleeding is not rare. SDGs also occur after operative removal of CSDHs. Thus, all three traumatic subdural lesions may change into another lesion under certain conditions.

Finally, our retrospective study may not be enough to solve this issue. Although a prospective study would be ideal to prove this issue, it seems to be very hard due to the following reasons. A minute trauma could separate the dura-arachnoid interface. The majority of SDG are asymptomatic(Lee KS et al., 1994) and resolve spontaneously(Kopp, 1988). Thus, a significant number of patients may not visit doctors. So, detection of the study population at the beginning is hampered. Nearly a half of patients with CSDH have a history of chronic alcoholism(Cooper, 1993) and frequently they forgot the injury. We do not know how many patients with SDG recover without any medical attention. Such unrecognized SDGs may constitute a half of CSDH.

In conclusion, all three traumatic subdural lesions have a certain relationship. ASDH usually results from tear of bridge veins or cortical vessels, or cortical laceration after trauma. The origin of CSDH seems to be SDG in a half of cases. Although the occurrence rate of CSDH from SDG was higher than that of ASDH, the prevalence rate of ASDH was higher than that of SDG. SDG is produced by separation of the dura-arachnoid interface when there is sufficient potential subdural space. A half of CSDHs may be

transformed from ASDHs, when the potential subdural space is sufficient. ASDH and SDG may be produced from CSDH. Such transformation or development of new subdural lesions is a function of premorbid condition and the dynamics between the absorption capacity and expansile force of the lesion.

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