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Risk factors of bilateral chronic subdural hematoma compared to unilateral chronic subdural hematoma

[1]Young Il Kim *MD*, [1]Dong Hoon Lee *MD*, [2]Sang Uk Kim *MD*, [1]Ho Jun Yi *MD*, [1]Seung Ho Yang *MD PhD*, [1]Il Sup Kim *MD PhD*, [1]Jae Taek Hong *MD PhD*, [1]Jae Hoon Sung *MD PhD*, [1]Chul Bum Cho *MD PhD*

[1]Department of Neurosurgery, St. Vincent's Hospital, College of Medicine, The Catholic University of Korea, Seoul; [2]Department of Neurosurgery, Deajeon St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul, Korea

Abstract

Chronic subdural hematoma (CSDH) is a common and relatively benign disease. The aim of this study was to investigate the differences between unilateral and bilateral chronic subdural hematoma in terms of predisposing factors. A retrospective analysis was made of all patients who underwent operation for CSDH at our institution between January 2010 and December 2015. Patients were divided into two groups (unilateral versus bilateral CSDH) and univariate and multivariate analysis was performed to assess demographic data, symptoms, cause of SDH, medical history, laboratory data, and initial radiologic findings. A total of 246 patients were enrolled. There were 63 (25.6%) patients with bilateral CSDH. There were no significant differences concerning sex and initial symptoms between the two groups. Only malignancy history was a significant risk factor for bilateral CSDH in both univariate and multivariate analysis (p = 0.002 and 0.001, respectively). In multivariate analysis, diabetes mellitus (OR 2.03, 95% CI: 1.05 - 3.92, p = 0.0350), malignancy (OR 5.09, 95% CI: 1.93 - 13.40, p = 0.0010), membrane septation (OR 0.50, 95% CI: 0.25 - 0.96, p = 0.0392), and brain atrophy (mild: OR 2.34, 95% CI: 1.16 - 4.71, p = 0.0164, moderate: OR 3.85, 95% CI: 1.32-11.18, p = 0.0131) were significantly associated with bilateral CSDH. The present study suggests that diabetes mellitus, malignancy, membrane septation and mild to moderate brain atrophy is independent predisposing factors of bilateral CSDH.

Keywords: Bilateral, Chronic subdural hematoma, risk factor

INTRODUCTION

Chronic subdural hematoma (CSDH) is a commonly encountered disease entity in neurosurgical department. Most CSDH patients have a good postoperative outcome. However, recurrence or complications after treatment often become an obstacle. Bilateral CSDH accounts for 16%-20% of CSDH cases.[1-3] There are previous reports that bilateral CSDH patients are prone to be elderly (> 75 years old) and to have a related coagulation problem.[4,5] The progress of disease in bilateral CSDH is more complicated than unilateral CSDH and some bilateral CSDH cases might show rapid aggravation.[6]

The predisposing factors of bilateral CSDH compared to unilateral CSDH is still uncertain and related studies are unsatisfactory. In this study, we retrospectively analyzed the differences between unilateral and bilateral CSDH in terms of predisposing factors.

METHODS

Institutional review board approval was received, and all patients were from a single institution. We retrospectively reviewed all patients who underwent surgery for CSDH at our institution between January 2010 and December 2015. All cases of CSDH were confirmed based on computed tomography (CT) scan.

Patient age, sex, symptom duration from symptom to diagnosis, type of symptoms (headache, hemiparesis, loss of consciousness, speech disturbance, gait disturbance and memory impairment), initial Glasgow Coma Scale (GCS), repeated head trauma, cause of CSDH and comorbidities including hypertension

Address correspondence to: Chul Bum Cho, Department of Neurosurgery, St. Vincent's Hospital, College of Medicine, The Catholic University of Korea, 93, Jungbu-Daero, Paldal-gu, Suwon-si, Gyeonggi-do, 16247, Republic of Korea. Tel: +82-1087581826, Email: medics0919@gmail.com

(HBP), diabetes mellitus (DM), liver disease, renal disease, coronary heart disease (CHD), cerebrovascular disease (CVD), malignancy, and history of anticoagulant or antiplatelet medication were obtained from the medical records. Laboratory data investigated included platelet count, prothrombin time percentage, and international normalized ratio (INR). The initial CT findings noted included the site of the hematoma (unilateral or bilateral cerebral convexity), degree of brain atrophy, density of subdural hematoma, and presence of membrane septation containing two components of different densities with a clear boundary between them. Brain atrophy was classified into four stages: no, mild, moderate, and severe atrophy using visual rating of brain atrophy scale.[7] The density of subdural hematoma was classified into four groups; low density (<25 Hounsfield Units [HU]), iso density (25-35 HU), hyper density (>35 HU), and mixed type on the basis of CT scans.

Statistical analysis

Data are presented as the mean ± standard deviation, or median (interquartile range) for continuous variables, and number (percentage) for categorical variables. A univariate analysis was performed with Pearson's chi-square test or Fisher's exact test and Mann-Whitney U test to examine the presence of an association between variables in this study and bilateral CSDH. The Mann–Whitney U test was used for non-categorical variables (age, platelet, INR, and PT percentage). Variables with $p < 0.2$ in univariate analysis were selected for multivariate analysis. Multivariate analysis was performed using a logistic regression model to determine the independent association of the recurrence. Variables in the final model were selected according to a backward method. The relationship between each factor and bilateral CSDH is presented in terms of odds ratio (OR) and 95% confidence interval (CI). A p-value < 0.05 was considered statistically significant. Statistical analysis was performed using the MedCalc 16.2.0 software package (MedCalc Software, Mariakerke, Belgium).

RESULTS

Between January 2010 and December 2015, we treated 246 consecutive patients diagnosed with CSDH. Of these, 63 (25.6%) patients had bilateral CSDH.

The baseline characteristics are described in Table 1. The mean age was 68.59 ± 12.23 (range, 29–90) years. There were 173 men and 73 women. History of HBP, DM, liver disease, kidney disease, CHD, CVD, and malignancy were present in 117 (47.5%), 69 (28%), 19 (7.7%), 7 (2.8%), 10 (4.0%), 11 (4.4%), and 22 (8.9%) patients, respectively. Prior medication history included anticoagulant in 6 patients (2.4%) and antiplatelet drugs in 53 patients (21.5%). The most common cause of CSDH was head trauma (187 patients, 76.0%). The patients had a history of repeated head trauma were 13 (5.2%) and symptom duration was 22.7 ± 23.50 days. 205 (83.3%) patients had an initial GCS of 15 and 41 patients (16.7%) had an initial GCS below 14. The most common symptom was headache (147 patients, 59.7%). Bilateral CSDH was present in 63 patients (25.7%) compared with unilateral CSDH (183 patients, 74.3%). The most common hematoma density on CT was mixed density (109 patients, 44.3%). Midline shifting was 7.23 ± 5.47 mm (range 0-21 mm). Initial hematoma thickness was 20.01 ± 6.99 mm (range 5-40 mm). Severe brain atrophy was present in 25 patients (10.1%).

Table 2 summarizes baseline characteristics between unilateral and bilateral CSDH in 246 patients with CSDH. The unilateral CSDH group and bilateral CSDH group were compared to find out the statistical significance. Sixty three patients (25.6%) showed bilateral CSDH, whereas 183 patients (74.4%) showed unilateral CSDH. There was no significant difference in sex between two groups. Patients with bilateral CSDH tended to be advanced in age and have DM and liver disease. Malignancy history and cause of CSDH were significantly related with bilateral CSDH ($p = 0.0011$, $p = 0.0165$, respectively). Initial symptoms and laboratory investigation were not significantly related with bilateral CSDH. In terms of radiologic finding, hematoma density was not significantly related with bilateral CSDH. Patients with bilateral CSDH tended to have a radiologic finding of brain atrophy ($p = 0.0722$) on brain CT.

Univariate and multivariate logistic regression analyses were done. Table 3 summarizes independent factors of bilateral CSDH in 246 patients with CSDH. Only malignancy history was a significant risk factor for bilateral CSDH in univariate logistic regression analysis ($p = 0.0021$). A statistical tendency towards a higher rate of bilateral CSDH was present for age ($p = 0.0718$), DM ($p = 0.0849$), liver disease ($p = 0.656$), spontaneous cause of CSDH ($p = 0.0670$),

Table 1: Baseline characteristics

Age, mean ± SD (range)	68.59 ± 12.23 (29-90)
Sex, men: women	173:73
Comorbidities	
Hypertension	117
DM	69
Liver disease	19
Chronic alcoholics	13
Liver cirrhosis	4
Hepatitis B	2
Kidney disease	7
CHD	10
CVD	11
Malignancy	22
Colorectal cancer	3
Stomach cancer	5
Biliary tract cancer	3
Lung cancer	2
Bladder cancer	3
Gynecologic cancer	2
Breast cancer	2
Renal cell cancer, tongue cancer	2
Prior medication	
Anticoagulation	6
Antiplatelet agent	53
History and Initial symptoms	
Cause of CSDH	
Trauma	187
Previous craniotomy history	4
Spontaneous or unknown	55
Repeated trauma history	13
[a]Symptom duration (days)	22.7±23.50 (0-90)
Initial GCS	
15	205
13-14	13
8-12	22
3-7	6
Headache	147
Hemiparesis	105
LOC	10
Speech disturbance	27
Gait disturbance	58
Memory impairment	12
Laboratory investigation	
[a]Platelet	238951 ± 79949 (52000-619000)
[a]INR	1.0837 ± 0.27 (0.89-4.53)
[a]PT percentage	91.38 ± 16.53 (16-135)
Radiologic finding	
Hematoma (unilateral: bilateral)	183.63
Hematoma density on CT	
Low density	44
Iso density	76
Hyper density	17
Mixed density	109
[a]Midline shifting (mm)	7.23±5.47 (0-21)
Membrane septation	97
[a]Hematoma thickness (mm)	20.01 ± 6.99 (5-40)
Brain atrophy	
Normal	73
Mild	85
Moderate	63
Severe	25

[a]Values are expressed as mean ± standard deviation (range)

Table 2: Summary of baseline characteristics between unilateral and bilateral CSDH in 246 patients with CSDH

	Unilateral CSDH group	Bilateral CSDH group	*p*-value
Total number (n)	183 (74.4%)	63 (25.6%)	
[a]Age (year)	70 (62.0000 to 76.0000)	74 (65.2500 to 77.7500)	**0.0578**[*]
Male/Female	129/54	44/19	0.9225[+]
Comorbidities			
Hypertension	90 (49.2%)	27 (42.9%)	0.3870[+]
DM	46 (25.1%)	23 (36.5%)	**0.0837**[+]
Liver disease	18 (9.8%)	1 (1.6%)	**0.0510**[#]
Kidney disease	5 (2.7%)	2 (3.2%)	1.0000[#]
CHD	9 (4.9%)	1 (1.6%)	0.4597[#]
CVD	10 (5.5%)	1 (1.6%)	0.2980[#]
Malignancy	10 (5.5%)	12 (19%)	**0.0011**[+]
Prior medication			
Anticoagulation	5 (2.7%)	1 (1.6%)	1.0000[#]
Antiplatelet agent	36 (19.7%)	17 (27.0%)	0.2243[+]
History and Initial symptoms			
Cause of CSDH			**0.0165**[+]
Trauma	133 (72.7%)	54 (85.7%)	
Previous craniotomy history	4 (2.2%)	0 (0%)	
spontaneous	46 (25.1%)	9 (14.3%)	
Repeated trauma history	10 (5.5%)	3 (4.8%)	1.0000[#]
[a]Symptom duration (days)	14 (4 to 30)	15 (6.25 to 30.00)	0.6750[*]
Initial GCS			0.5778[^]
15	154 (84.2%)	51 (81.0%)	
13-14	9 (4.9%)	4 (6.3%)	
8-12	16 (8.7%)	6 (9.5%)	
3-7	4 (2.2%)	2 (3.2%)	
Headache	110 (60.1%)	37 (58.7%)	0.8476[+]
Hemiparesis	83 (45.4%)	22 (34.9%)	0.1495[+]
LOC	8 (4.4%)	2 (3.2%)	1.0000[#]
Speech disturbance	20 (10.9%)	7 (11.1%)	0.9682[+]
Gait disturbance	45 (24.6%)	13 (20.6%)	0.5244[+]
Memory impairment	9 (4.9%)	3 (4.8%)	0.9605[#]
Laboratory investigation			
[a]Platelet	227000 (187000 to 280050)	251000 (183000 to 280250)	0.3880[*]
[a]INR	1.0500 (1.00 to 1.10)	1.0400 (0.98 to 1.10)	0.6285[*]
[a]PT percentage	93 (83.00 to 101.00)	93 (85.00 to 100.00)	0.5660[*]
Radiologic finding			
Hematoma density on CT			0.4680[^]
Low density	31 (13.9%)	13 (20.6%)	
Iso density	57 (31.1%)	19 (30.2%)	
Hyperdensity	11 (6.0%)	6 (9.5%)	
Mixed density	84 (45.9%)	25 (39.7%)	
Membrane septation	77 (42.1%)	20 (31.7%)	0.1487[+]
Brain atrophy			**0.0722**[^]
Normal	56 (30.6%)	17 (27.0%)	
Mild	70 (38.3%)	15 (23.8%)	
Moderate	41 (22.4%)	22 (34.9%)	
Severe	16 (8.8%)	9 (14.3%)	

[a]Values are expressed as median (interquartile range)
[*]Mann-Whitney analysis
[+]Chi-square analysis
[^]Chi-square analysis for trend
[#]Fisher's exact test

Table 3: Univariate and multivariate logistic regression analysis for risk factor for bilateral CSDH

Variables	Univariate analysis			Multivariate analysis		
	OR	95% CI	*p*-value	OR	95% CI	*p*-value
Age (per year)	1.0238	0.9979 to 1.0504	**0.0718**			
Male	1.0316	0.5522 to 1.9269	0.9223			
Comorbidities						
Hypertension	0.7750	0.4352 to 1.3801	0.3866			
DM	1.7125	0.9286 to 3.1582	**0.0849**	2.0319	1.0512 to 3.9278	**0.0350**
Liver disease	0.1478	0.0193 to 1.1311	**0.0656**	0.1559	0.0197 to 1.2353	0.0784
Kidney disease	1.1672	0.2207 to 6.1722	0.8556			
CHD	0.3118	0.0387 to 2.5117	0.2736			
CVA	0.2790	0.0350 to 2.2247	0.2282			
Malignancy	4.0706	1.6625 to 9.9665	**0.0021**	5.0927	1.9345 to 13.4065	**0.0010**
Prior medication						
Anticoagulation	0.5742	0.0658 to 5.0111	0.6157			
Antiplatelet agent	1.5091	0.7760 to 2.9346	0.2253			
History and Initial symptoms						
Cause of CSDH						
Trauma	Ref					
Previous craniotomy history	0		0.9976			
spontaneous	0.4819	0.2206 to 1.0516	**0.0670**	0.4501	0.1940 to 1.0445	0.0631
Repeated trauma history	0.8650	0.2303 to 3.2485	0.8299			
Symptom duration	1.0015	0.9895 to 1.0137	0.8075			
Initial GCS per point	0.9697	0.8488 to 1.1078	0.6510			
Headache	0.9444	0.5275 to 1.6907	0.8473			
Hemiparesis	1.1152	0.9613 to 1.2938	**0.1502**			
LOC	0.7172	0.1482 to 3.4705	0.6795			
Speech disturbance	1.0188	0.4090 to 2.5378	0.9682			
Gait disturbance	0.7973	0.3972 to 1.6005	0.5241			
Memory impairment	0.9667	0.2533 to 3.6888	0.9604			
Laboratory investigation						
Platelet	1.0005	0.9969 to 1.0040	0.7960			
INR	0.8014	0.2326 to 2.7606	0.7257			
PT percentage (%)	1.0061	0.9882 to 1.0243	0.5077			
Radiologic finding						
Hematoma density on CT						
Low density	Ref					
Iso density	1.4090	0.6416 to 3.0943	0.3929			
Hyper density	1.1200	0.5647 to 2.2214	0.7457			
Mixed density	1.8327	0.6160 to 5.4530	0.2762			
Membrane septation	0.6403	0.3492 to 1.1740	**0.1495**	0.5004	0.2591 to 0.9665	**0.0392**
Brain atrophy						
Normal	Ref					
Mild	1.7676	0.8347 to 3.7429	**0.1367**	2.3492	1.1696 to 4.7183	**0.0164**
Moderate	2.1961	0.7713 to 6.2529	**0.1406**	3.8519	1.3270 to 11.1811	**0.0131**
Severe	0.8235	0.0861 to 7.8727	0.8661			

Hosmer & Lemeshow test, Chi squared 5.0783, $P = 0.6504$
Backward method

hemiparesis (p=0.1502), membrane septation (p = 0.1495), and brain atrophy (p = 0.1367). Multivariate logistic regression analysis, we found that DM (OR 2.03, 95% CI: 1.05-3.92, p = 0.0350), malignancy (OR 5.09, 95% CI: 1.93-13.40, p = 0.0010), membrane septation (OR 0.50, 95% CI: 0.25-0.96, p = 0.0392), and brain atrophy (mild: OR 2.34, 95% CI: 1.16-4.71, p = 0.0164, moderate: OR 3.85, 95% CI: 1.32-11.18, p = 0.0131) were significantly associated with bilateral CSDH. DM, malignancy and brain atrophy are risk factors and membrane septation is a protective factor for bilateral CSDH. Spontaneous bleeding as the cause of CSDH have the tendency of protective factor for bilateral CSDH (OR 0.45, 95% CI: 0.19-1.04, p = 0.0631).

DISCUSSION

When the veins that bridge the subdural space are excessively stretched, they rupture and venous blood escapes into the subdural space.[8] CSDHs start with hemorrhage into the subdural space. The blood becomes encapsulated by a membrane of neovascularization at the inner surface of dura mater. Membrane fragility results from an inflammatory reaction to a chemical mediator in the subdural fluid collection. The sinusoid channels in the neomembrane result in frequent micro-bleeding. Finally, encapsulation of unclotted and liquefied hematoma occurs by splitting of the neomembrane.[9]

The pathophysiology of bilateral CSDH is not well understood. Confusion can arise when assessing whether bilateral craniostomy in one session gives a better clinical outcome than does unilateral craniostomy only with larger size CSDHs.

Many risk factors for the development of CSDH have been described in the literature.[10,11] Some factors for bilateral CSDH have been reported.[1,2,12] Several risk factors for bilateral CSDH have been reported; they include old age, coagulopathy, medication with antiplatelet agent and anticoagulant agent, and hemodialysis.[5] But there are few advanced studies on the predictive factors for bilateral CSDH related to clinical history and radiologic finding, such as membrane septation, brain atrophy, hematoma thickness, and hematoma density on CT through multivariable regression analysis. This prompted us to examine the predictive factors for bilateral CSDH. Brain atrophy, malignancy, DM and membrane septation were independent predictors for bilateral CSDH.

Clinical findings

Age

In general, brain atrophy is closely related to the aging process. Age is expected to have the clinical significance of bilateral CSDH. We could not find an age-related effect. Age certainly had the tendency of association with bilateral CSDH (p = 0.0578). Previous reports showed the results of significant association between age and bilateral CSDH. We speculate that the reason of this difference is as follows; In terms of age, the mean age was 68.59 years old in our study. There were 27 patients below 50 years of age (5 patients with bilateral CSDH). A few of these patients below 50 years of age could influence the significant association between age and bilateral CSDH. However, age was strongly associated with bilateral CSDH.

Diabetes mellitus

DM was an independent predictive factor for bilateral CSDH (p = 0.0350). The blood of patients with DM has a high osmotic pressure and increased platelet aggregation, so DM may play a role in decreasing rebleeding in patients with CSDH.[13] On the other hand, DM may play a role in increasing the risk of bleeding of CSDH. However there was no explanation for the role of DM in the development of bilateral CSDH.[14] Exudation due to capillary vasculopathy caused by DM plays an important role in the re-expansion of the CSDH cavity.[15] Hyper viscosity of the subdural blood is expected to draw more water into the hematoma and increase the risk of bleeding.[16] We suggest that the cause of higher incidence of bilateral CSDH of patients with DM may be well-developed neovascularization of the inner and outer neomembrane, which are a bleeding source of CSDH compared with non-diabetics. In case of diabetic retinopathy, hyperglycemia instigates retinal vascular endothelial dysfunction; thus, retinal ischemia and increased vascular permeability are major reasons for diabetic retinopathy.[17] We speculate that inner and outer neomembrane of patients with DM and CSDH may be like diabetic retinopathy. It is likely for patients with DM, minimal subdural hematoma (early stage of CSDH development) tends to increase the amount of subdural hematoma rather than decreasing it. For this reason, minimal acute stage subdural hematoma seems to develop bilateral CSDH rather than unilateral CSDH because of well-developed neovascularization of the global

cerebral dura membrane. Neovascularization degree of the inner and outer neomembrane in patients with DM may be more developed than in patients without DM. Patients with DM usually take various medications, such as antiplatelet agent, antihypertensive drug, and antihyperlipidemia drug. Such various medication histories may exert a bad influence on coagulation capacity of CSDH in terms of the development of bilateral CSDH rather than unilateral CSDH.

Liver disease

In this study, liver disease tended to be associated with unilateral CSDH in both univariate analysis and multivariate logistic regression analysis (p = 0.0510 and p = 0.0784, respectively). The etiology of liver disease in CSDH patients is summarized in Table 1. Mean platelet count and PT (mean ± standard deviation (range)) were 187,105 ± 79,351/μl (62,000 - 345,000/μl) and 78.26 ± 21.03% (range, 30 - 126%). Platelet count was within normal range and PT was impaired. We suppose that this difference between platelet count and PT is a typical finding in liver disease because prothrombin is one of the clotting factors made by the liver.[18]

Unilateral CSDH associated with liver disease is expected to be related with hemostasis. Impaired prothrombin function is detrimental to hemostasis. It is important to explain why impaired hemostasis function causes unilateral CSDH more than bilateral CSDH. We suggest that minimal bleeding leak at subdural space in patients with liver disease develops more rapidly. Early development of symptomatic CSDH leads to early diagnosis of CSDH. Symptomatic CSDH originates from mass effect of CSDH to brain parenchyma. The degree of midline shifting was greater in unilateral CSDH than in bilateral CSDH.[4] Unilateral CSDH tends to be diagnosed early compared to bilateral CSDH. We speculate that rapid development of CSDH may be closely related with impaired hemostasis function.

Malignancy

Malignancy tended to be associated with bilateral CSDH in both univariate analysis and multivariate logistic regression analyses (p = 0.0021 and p = 0.0010, respectively). Data concerning the etiology of malignancy in CSDH patients are presented in Table 1. Mean ± standard deviation (95% CI) age of CSDH patients with and without malignancy, were 74.59 ± 9.33 years (70.45 to 78.72) and 68.00 ± 12.34 years (66.37 to 69.62), respectively. There was a significant difference of mean age between CSDH patients with malignancy and CSDH without malignancy (p = 0.0156) using independent t-test. We suggest that the reason for tendency of bilateral CSDH associated with malignancy may be age. There is another point of view on this issue. It is suspected that malignancy facilitate the development of neovascularization of cerebral dura in case of CSDH because of overexpression of vascular endothelial growth factor. However, many forms of research are needed to support this opinion.

Cause of chronic subdural hematoma

There was an inverse correlation between spontaneous cause of CSDH and bilateral CSDH (OR: 0.4819, 95% CI: 0.2206 to 1.0516). Mean ± standard deviation (95% CI) symptom duration of CSDH patients with traumatic cause and CSDH with spontaneous or unknown cause was 25.45 ± 24.54 days (21.91 to 29.00) and 14.00 ± 17.73 days (9.20 to 18.79), respectively. There was a significant difference of mean age between CSDH patients with traumatic cause and CSDH with spontaneous or unknown cause (p = 0.0014) using independent t-test. We suggest that symptom duration (from initial symptom episode to diagnosis of CSDH) in spontaneous or unknown CSDH is shorter than in traumatic CSDH. Therefore, rapid progress to mass effect of CSDH seems to be more frequent in spontaneous CSDH.

Radiologic finding

Brain atrophy

The degree of brain atrophy was an independent predictive factor for bilateral CSDH. Patients with brain atrophy tend to show poor brain re-expansion after operation for CSDH. We speculate that bilateral CSDH may be a post-operative risk factor for recurrent CSDH. Brain atrophy is closely associated with aging. CSDH tends to occur in elderly people because brain atrophy causes enlargement of the subarachnoid space and stretching of the bridging veins; these situations provoke tearing of the bridging veins and bloody cerebrospinal fluid in the subdural space after mild head injury.[19]

The degree of brain atrophy is classified into four stages according to the visual rating of brain atrophy scale.[7] We found that the values of odds ratio is higher in moderate brain atrophy (OR: 3.8519, 95% CI: 1.3270 to 11.1811) compared

to mild brain atrophy (OR: 2.3492, 95% CI: 1.1696 to 4.7183). We suggest that the incidence of bilateral CSDH rather than unilateral CSDH may be higher when the degree of brain atrophy is more severe. Diagnosis of bilateral CSDH can be delayed due to lack of specific symptoms such as hemiparesis. Consequently, delayed diagnosis of SDH is more frequent in bilateral CSDH compared to unilateral CSDH. This is similar to brain atrophy, where delayed diagnosis of CSDH can be a cause of statistical significance between brain atrophy and bilateral CSDH.

Membrane septation of hematoma

An inverse correlation was evident between membrane septation of intrahematoma and bilateral CSDH (OR: 0.5004, 95% CI: 0.2591 to 0.9665). To our knowledge, there have been no reports about the relationship between membrane septation of intrahematoma and bilateral CSDH. We speculate that bilateral CSDH arises from brain atrophy rather than neovascularization of the inner and outer dura membrane. Membrane septation of intrahematoma seems to be a marker of well-developed neovascularization of the dura membrane. It is likely that the progress patterns toward CSDH after initial development of SDH may be quite different although the initial events of SDH between unilateral and bilateral CSDHs were same (rupture of stretched bridge vein in subdural space). In case of bilateral CSDH, pre-existing brain atrophy facilitates CSDH bilaterally after rupture of bilateral stretched bridge vein at the same time or at the different time, while in case of unilateral CSDH, neovascularization of dura membrane causes CSDH unilaterally. This is not to say that the cause of unilateral CSDH is only neovascularization of the dura membrane. It is doubtful that the influence of neovascularization of CSDH may be stronger in unilateral CSDH than in bilateral CSDH. Consequently, this hypothesis seems to be a reason for the higher incidence of membrane septation in association with unilateral CSDH.

The present study was a retrospective study and is therefore subject to potential sources of bias and variation. A prospective study with a larger number of cases is needed.

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DISCLOSURE

Conflict of interest: None

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