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Factors Associated With Subdural Hygroma Following Mild Traumatic Brain Injury

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ABSTRACT

Objective: Subdural hygroma (SDG) is a complication of traumatic brain injury (TBI). In particular, the outcome and outpatient treatment period may vary depending on the occurrence of SDG. However, the pathogenesis of SDG has not been fully elucidated. Therefore, this study aimed to identify the risk factors associated with the occurrence of SDG after mild TBI.

Methods: We retrospectively analyzed 250 patients with mild TBI admitted to a single institution between January 2021 and December 2021. The SDG occurrence and control groups were analyzed according to the risk factors of SDG, such as age, history, initial computed tomography (CT) findings, and initial laboratory findings.

Results: The overall occurrence rate of SDG was 31.6% (n=79). A statistically significant association was found between preoperative diagnoses and the occurrence of SDG, such as subarachnoid hemorrhage (odds ratio [OR], 2.36; 95% confidence interval [CI], 1.26–4.39) and basal skull fracture (OR, 0.32; 95% CI, 0.12–0.83). Additionally, age ≥70 years (OR, 3.20; 95% CI, 1.74–5.87) and the use of tranexamic acid (OR, 2.12; 95% CI, 1.05–4.54) were statistically significant factors. The prognostic evaluation of patients using the Glasgow Outcome Scale (GOS) did not show any statistical differences between patients with and without SDG.

Conclusion: SDG was not associated with the prognosis of patients assessed using the GOS. However, depending on the occurrence of SDG, differences in patient symptoms may occur after mild TBI. Therefore, the early evaluation of patients with mild TBI and determination of the probability of developing SDG are important.

Keywords: Subdural hygroma; Traumatic brain injuries

INTRODUCTION

Traumatic brain injury (TBI) is a common state that occurs after a traumatic event, showing various complications, including headache, dizziness, and cognitive decline. Patients with mild TBI with an initial Glasgow Coma Scale (GCS) score of 13 points or higher recover naturally in most cases during the follow-up period; however, brain atrophy and subdural hygroma (SDG) are observed in some patients, with an incidence of 4%–6% of patients with mild TBI.[26]

Most patients with SDG do not show symptoms and do not require particular surgical treatment; however, in 5%–58% of patients with post-traumatic SDG, transition to subdural hematoma (SDH) was observed,[17] rarely along with signs of increased intracranial pressure (ICP).[19] Particularly, cognitive decline can often be observed in patients with mild TBI during the follow-up period, and there are no diagnostic guidelines clearly established yet in this regard. Therefore, regular follow-up, monitoring, and prediction are required for SDG that occurs following TBI. Currently, various studies have focused on SDG occurring after surgical treatment and its risk factors.[17,18] However, there is little interest in mild TBI; therefore, the risk factors revealed in the aforementioned studies cannot be applied to all mild TBI cases.

MATERIALS AND METHODS

Patient characteristics

This retrospective study involved 294 patients with initial GCS score of 13 points or higher mild TBI. Patients hospitalized through the emergency room of our hospital from January 2020 to December 2020. Of the 294 patients, 250 patients who met all inclusion criteria were enrolled; 15 patients who underwent surgical treatment because of aggravated hemorrhage during the follow-up period, 6 patients who died early because of medical complications, 19 patients whose symptoms shifted to chronic SDH, and 4 patients who were followed up for less than 3 months were excluded from this study. Furthermore, we excluded all cases in which surgical treatment was performed, due to the elevation of TBI grading. This study focuses on mild TBI grading only and was designed to determine the precautions during the follow-up period for patients with TBI by focusing on the risk factors for SDG that occurs following mild TBI.

We set the independent variables as follows: 1) patient characteristics including gender, age (over 70 years and over 80 years), history (i.e., hypertension, diabetes mellitus, cardiovascular disease, respiratory disease, renal disease, liver disease, dementia, cerebral infarction, and intracerebral hemorrhage [ICH]), medication history (i.e., antiplatelets and anticoagulants); 2) initial computed tomography (CT) diagnoses, such as epidural hemorrhage, SDH, subarachnoid hemorrhage (SAH), ICH, contusional hemorrhage, basal skull fracture, fracture compound comminuted depressed/fracture comminuted depressed, and bilateral hemorrhage; 3) initial GCS score and GCS score after 24 hours; 4) initial laboratory findings (i.e., creatine kinase, lactate dehydrogenase, calcium, phosphate, glucose, platelet, prothrombin time (PT), partial thromboplastin time, international normalized ratio, sodium, and osmole); 5) medication usage (i.e., mannitol and tranexamic acid); and 6) prognostic value, such as final Glasgow Outcome Scale (GOS) score. The dependent variable of this study is the occurrence of SDG.

SDG is defined as a case in which low-density subdural fluid of a minimum of 3 mm is observed between the intracranial surface and the cerebral cortex on CT after a traumatic event,[23] and in this study, we observed changes through the follow-up imaging study of at least 3 months. This retrospective study was approved by the Institutional Review Board of Wonju Severance Christian Hospital (CR322055).

Statistical analysis

Data are expressed according to the properties of the variable. Continuous variables are presented as means and standard deviations. Categorical variables are presented as

frequencies and percentages. To compare the 2 groups, we performed the 2-sample *t*-test or χ^2 test (Fisher's exact test), as appropriate. Logistic regression analysis was performed to identify the factors to predict SDG, and the results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs). A *p*-value less than 0.05 was considered statistically significant, and all statistical analyses were conducted using Statistical Package for the Social Sciences (version 24; IBM Corp., Armonk, NY, USA).

RESULTS

The average age of the 250 patients was 63.1 years. During the follow-up period, SDG occurred in 79 patients (31.6%), and the average age of the patients who had SDG was 70.3 years. No statistically significant gender difference in the incidence of SDG was observed, whereas there was a statistically significant difference in age between patients with and without SDG (*p*<0.001). When the patients were divided into age groups (70 years and 80 years), each group showed a statistically significant difference in the incidence of SDG; a strong correlation was observed between the occurrence of SDG and the age of 70 years (*p*<0.001). Statistically older age is a risk factor for SDG. Additionally, hypertension (*p*=0.023), cardiovascular disease (*p*=0.032), intracranial hemorrhage (*p*=0.019), and the use of antithrombic medications (*p*=0.001) correlated significantly with the incidence of SDG. A medical history of other conditions, such as diabetes mellitus, chronic kidney disease did not show a significant statistical correlation with the incidence of SDG. The presence of SAH (*p*=0.003) and basal skull fracture (*p*=0.001) on the initial brain CT scans showed statistical significance in the occurrence of SDG. Differences in the incidence of SDG were observed between patients with and without SAH (*p*=0.003) and basal skull fracture (p = 0.001) in the initial brain CT scans. The initial GCS score and the 24-hour GCS score did not show a difference between patients with and without SDG. Regarding the initial laboratory findings, a significant difference in the platelet count (*p*=0.022) was observed between the two groups, whereas no significant differences in the other items were observed. Moreover, the use of mannitol had no correlation with the occurrence of SDG, whereas the use of tranexamic acid had a significant correlation with SDG occurrence (*p*=0.014). Even though the control group showed a more favorable outcome, there was no statistical significance in the outcomes evaluated using the GOS between the two groups (**TABLE 1**). The presence of SAH, age, low platelet counts, and the use of tranexamic acid showed statistical correlation with SDG occurrence in univariate analysis.

To further evaluate overlapping variables multivariate logistic regression analysis were done. In the multivariate logistic regression analysis, the independent risk factors for the incidence of SDG included the age of 70 years and older, traumatic SAH and basal skull fracture on the initial brain CT scans, initial platelet count, and the use of tranexamic acid (**TABLE 2**, **FIGURE 1**).

DISCUSSION

Symptoms and signs, diagnosis, treatment strategies, and prognosis of SDG
SDG is the abnormal accumulation of cerebrospinal fluid (CSF) in the subdural space due to arachnoid membrane rupture,[17,26] which was defined in a neuroradiological textbook by Osborn[27] as hypodense, CSF-like, crescentic extra-axial collections that purely consist of CSF, have no blood products, lack encapsulating membranes, and show no enhancement

TABLE 1. Univariate analysis of mild TBI patient characteristics and SDG occurrence

Characteristics	Total (n=250)	SDG (n=79)	No SDG (n=171)	p-value
Sex (male)	176 (70.4)	54 (68.4)	122 (71.3)	0.630
Age (year)	63.1±15.3	70.3±12.0	59.7±15.6	<0.001
Age ≥70	84 (33.6)	42 (53.2)	42 (24.6)	<0.001
History				
Hypertension	113 (45.2)	44 (55.7)	69 (40.4)	0.023
Diabetes mellitus	53 (21.2)	20 (25.3)	33 (19.3)	0.279
Cardiovascular disease				
Respiratory disease	31 (12.4)	15 (19.0)	16 (9.4)	0.032
Renal disease	25 (10.0)	8 (10.1)	17 (9.9)	0.964
Liver disease	12 (4.8)	3 (3.8)	9 (5.3)	0.758*
Dementia	28 (11.2)	5 (6.3)	23 (13.5)	0.097
Infarction	12 (4.8)	7 (8.9)	5 (2.9)	0.056*
Intracranial hemorrhage	15 (6.0)	8 (10.1)	7 (4.1)	0.084*
Cancer	20 (8.0)	11 (13.9)	9 (5.3)	0.019
Antithrombotic medication	21 (8.4)	7 (8.9)	14 (8.2)	0.858
Initial diagnosis	51 (20.4)	26 (32.9)	25 (14.6)	0.001
Subdural hemorrhage	133 (53.4)	45 (57.7)	88 (51.5)	0.361
Epidural hemorrhage	43 (17.3)	11 (14.1)	32 (18.7)	0.372
SAH	135 (54.2)	53 (67.9)	82 (48.0)	0.003
Contusional hemorrhage	89 (35.7)	31 (39.7)	58 (33.9)	0.374
ICH	17 (6.8)	7 (9.0)	10 (5.8)	0.364
Basal skull fracture	50 (20.1)	6 (7.7)	44 (25.7)	0.001
FCCD/FCD	10 (4.0)	0 (0)	10 (5.8)	0.033*
Bilateral hemorrhage	69 (27.7)	27 (34.6)	42 (24.6)	0.100
Hemorrhage increased	24 (10.8)	12 (15.8)	12 (8.2)	0.085
Initial GCS	14.6±0.7	14.5±0.7	14.6±0.7	0.545
24 hours GCS	14.5±1.0	14.3±1.1	14.6±0.9	0.074
Laboratory finding (initial)				
CK	271.2±318.0	294.1±393.0	260.5±277.0	0.439
LDH	371.6±233.5	313.7±155.7	319.3±262.3	0.861
Calcium	9.1±0.6	9.1±0.6	9.1±0.6	0.891
Phosphate	3.0±0.8	3.0±0.7	3.0±0.9	0.695
Glucose	145.6±59.2	143.8±53.9	146.5±61.6	0.742
Platelet	227.8±74.7	211.9±60.3	235.1±79.5	0.022
PT	12.1±2.5	11.9±1.0	12.2±3.0	0.336
PTT	26.2±2.9	25.8±2.3	26.4±3.2	0.143
INR	1.0±0.2	1.0±0.1	1.1±0.3	0.325
Natrium	139.7±3.3	139.7±3.1	139.7±3.4	0.917
Serum osmole	292.6±7.2	293.3±7.0	292.3±7.3	0.286
Medications				
Mannitol	42 (16.8)	18 (22.8)	24 (14.0)	0.085
Tranexamic acid	180 (72.0)	65 (82.3)	115 (67.3)	0.014
GOS				0.102
Favorable outcome	234 (93.6)	71 (89.9)	163 (95.3)	
Unfavorable outcome	16 (6.4)	8 (10.1)	8 (4.7)	

Categorical variables are presented as frequencies and percentages and continuous variables are presented as means and standard deviations.
TBI: traumatic brain injury, SDG: subdural hygroma, SAH: subarachnoid hemorrhage, ICH: intracerebral hemorrhage, FCCD: fracture comminuted compound depressed, FCD: fracture comminuted depressed, GCS: Glasgow Coma Scale, CK: creatine kinase, LDH: lactate dehydrogenase, PT: prothrombin time, PTT: partial thromboplastin time, INR: International Normalized Ratio, GOS: Glasgow Outcome Scale.
*Fisher's exact test.

following contrast administration. Most patients with SDG do not present symptoms; however, SDG can be accompanied by various complications,[19] including headache, dizziness, vomiting, ataxia, epilepsy, and facial palsy due to increased ICP. Moreover, a study reported that brain parenchymal atrophy and neurodegeneration following TBI make abnormal accumulation of CSF in subdural space and increase the incidence of Alzheimer's

TABLE 2. Logistic regression to calculate OR for SDG occurrence in risk factors

Variables	OR	95% CI	p-value
Age ≥70 years	3.196	1.740–5.872	<0.001
Cerebral hemorrhage history	2.628	0.986–7.006	0.053
Traumatic subarachnoid hemorrhage	2.355	1.263–4.393	0.007
Basal skull fracture	0.318	0.122–0.832	0.020
Initial platelet count	0.996	0.991–1.000	0.044
Use of tranexamic acid	2.188	1.054–4.544	0.036

OR: odds ratio, SDG: subdural hygroma, CI: confidence interval.
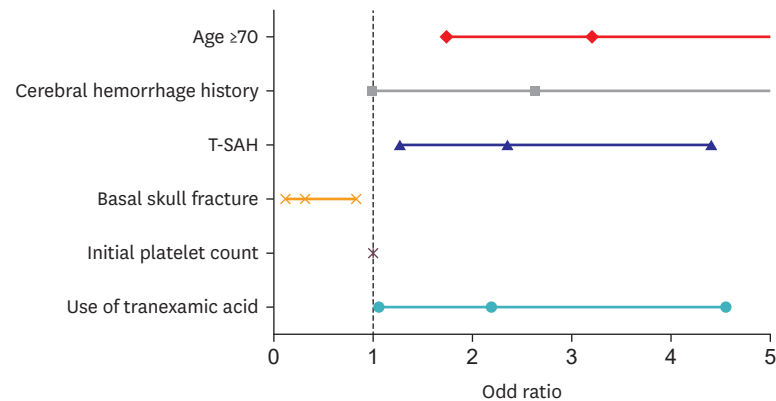
FIGURE 1. Forest plot of the relationship between SDG occurrence risk after mild TBI and initial patient's characteristics.
SDG: subdural hygroma, TBI: traumatic brain injury, T-SAH: traumatic subarachnoid hemorrhage.

disease and dementia,[8] in which it was stated that the frequency of dementia is 1.5–3 times higher after TBI.[8,31]

Generally, SDG is diagnosed using CT and magnetic resonance imaging (MRI), showing homogeneous isodense CSF collections in the subdural space on CT scan and isointense CSF collections on MRI.[5,7,11,15,16,30,32,34] In children, it should be distinguished from benign enlargement of the subarachnoid space that shows CSF collections in the subarachnoid space because of the immaturity of the arachnoid villi,[12,34] which can be differentiated by the shape of the blood vessels in the subarachnoid space through an MRI study.

Traumatic SDG shows various follow-up results over time. According to previous studies[4,24,25,33] it disappeared completely in 20.7% of patients with traumatic SDG and decreased in amount in 25.9% within 3 months, and the amount was maintained in 7.2%, whereas the disease progressed to chronic SDH in 32.8%. The study reported that in half of patients with SDG, the disease disappeared or decreased in amount within 3 months, and 61.3% of those whose SDG did not reduce progressed to chronic SDH. Furthermore, it has been reported that there were differences in the degree of conversion depending on studies, which were at least 5%–58% converted to chronic SDH.[20-22,26,34] The mechanism of the transition to chronic SDG is not fully clarified; however, Case[3] and Wittschieber et al.[34] reported that when normal ICP is maintained, SDG disappears naturally; however, in an environment with decreased ICP, the amount of SDG increases and neovascularization occurs in the dural border zone, explaining the transition to SDH due to continuous microbleeding in such a fragile vessel. Cha et al.[4] claimed that follow-up and conservative treatment should be performed if there is no transition to chronic SDH because SDG disappears in many cases even with a large amount. Cha et al.[4] and Ju et al.[14] reported that surgical treatment of SDG

did not improve the cognitive level or ability of affected patients but improved headache and excitability by increasing the ICP. Surgical treatment includes percutaneous subdural puncture, craniotomy, simple puncture drainage, and permanent subdural peritoneal shunting, and among those, simple puncture drainage and permanent subdural peritoneal shunting are mainly used.[2,6,18]

Pathophysiology of SDG

The mechanism of SDG has not been clearly identified; however, many studies have explained it as a phenomenon that occurs because of arachnoid membrane disruption and abnormal CSF flow. According to electron microscopic analysis, there is a lack of collagen fiber in the border zone between the dura and arachnoid space compared with the border zone between the periosteum and dura[9,10,17,29]; thus, the structure is vulnerable to trauma. An article reported that traumatic membrane damage forms a one-way valve for CSF flow,[17,28] leading to the incidence of SDG. Apart from injuries due to trauma, SDG can also occur because of surgical disruption, and after decompressive craniectomy (DC), SDG occurs in more than 90% of patients.[1,17] Additionally, Graham and Sharp[8] reported neurodegeneration and SDG aggravation caused by brain atrophy. When traumatic axonal demyelination occurs, the abnormal accumulation of tau protein occurs because of intra-axonal microtubule destruction, which activates microglial cells, leading to axonal apoptosis. Without demyelination, amyloid precursor protein secretion increases in injured axonal bulbs, and the protein is degraded by cleaving enzymes into β-amyloid, which is stored in axons, causing the formation of extracellular β-amyloid plaques in the long term. The mentioned plaques also induce apoptosis of neighboring cells and mitochondrial damage, and in the aforementioned study, brain white matter atrophy was explained by 2 mechanisms.

Risk factors for SDG

SDG that occurs after surgical treatment reaches its peak of occurrence approximately 3–4 weeks after DC and shows a naturally decreasing pattern between 14 and 17 weeks.[1,17] Several studies focused on risk factors related to this. Jeon et al.[13] and Kim et al.[17] claimed that the risk factors for SDG include midline shifting (>5 mm), SAH, delayed hydrocephalus, basal cistern compression, and arachnoid membrane tearing. Furthermore, Kim et al.[17] reported that when SDH, SAH, and cortical opening are observed on preoperative CT scans, it is highly likely that postoperative SDG will occur. However, such risk factors are applied to patients after surgery; thus, these risk factors cannot be applied to all mild TBI cases. In this study, it was found that the age of 70 years and older had a strong relationship with the occurrence of SDG after trauma.

Limitations and strengths

This was a single-center, retrospective study involving a small sample size of 296 patients, in which there was a 3-month follow-up process after discharge, and then, the follow-up was closed when there were no findings of aggravated SDG. Hence, changes in the cognitive abilities of the patients could not be observed through long-term follow-up. Moreover, the follow-up examination was performed using CT in most patients; thus, MRI and biomarker-related studies were not conducted. However, note that this is the first study that examined the risk factors associated with SDG that occurs following mild TBI.

CONCLUSION

For mild TBI, a long-term follow-up is almost never performed for diseases or complications commonly occurring after a traumatic event. However, cognitive decline is commonly observed after mild TBI, and studies are being conducted to determine the causes and report the necessity of follow-up. Through this study, we found that the age of 70 years and older is strongly related to the incidence of SDG, and for such patients, considering the necessity of the long-term follow-up for more than a year and regular cognitive ability tests is necessary. There is still no clear diagnostic method for cognitive decline and dementia occurring during the follow-up period after mild TBI. Therefore, it is considered that if further molecular biological studies are conducted on the mechanism of SDG incidence and biomarkers, such as tau protein and β-amyloid, it will be helpful for preventing post-TBI complications and alleviating the symptoms through the long-term follow-up and monitoring.

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