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Factors Associated With Short-Term Outcomes of Burr-Hole Craniostomy Associated With Brain Re-Expansion and Subdural Hematoma Shrinkage for Chronic Subdural Hematoma

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ABSTRACT

Objective: Chronic subdural hematoma (CSDH) is a commonly encountered neurosurgical pathology that frequently requires surgical intervention. With an increasingly aging demographic, more older people and patients with comorbidities will present with symptomatic CSDH. This study evaluated clinical and laboratory factors affecting the short-term outcomes of CSDH after surgical intervention.
Methods: We retrospectively analyzed 170 patients who underwent burr-hole trephination for CSDH in a single institution from January 2019 to December 2021. All patients were examined for risk factors and evaluated for hematoma thickness change and midline shifting on brain computed tomography (CT) scans at 3 days after burr-hole trephination.
Results: This consecutive series of patients included 114 males (67.1%) and 56 females (32.9%); mean age 72.4±12.5 years. Renal disease (p=0.044) and prior intracranial hemorrhage (p=0.004) were clinical factors associated with poorer prognosis. A statistically significant association was found between initial laboratory findings, including high creatine kinase (p=0.025) and low platelet (p=0.036) levels, and CT findings 3 days postoperatively. The 3-day mean arterial pressure and postoperative ambulation were not significantly associated with outcomes.
Conclusion: Burr-hole craniostomy is an effective surgical procedure for initial CSDH. However, patients with a history intracranial hemorrhage and abnormal laboratory findings, such as low platelet levels, who underwent burr-hole trephination had poor short-term outcomes. Therefore, these patients should be carefully monitored.

Keywords: Chronic subdural hematoma; Trephination; Recurrence

INTRODUCTION

Chronic subdural hematoma (CSDH) is a relatively common condition after trauma, presenting with various symptoms, such as headache, dizziness, impaired consciousness, and paraplegia. The total incidence of CSDH is approximately 1.72–20.6 cases per 100,000 persons per year and growing.[36]

Conflict of Interest
The authors have no financial conflicts of interest.

While conservative treatment is usually given to those with asymptomatic CSDH, surgical treatment can be required in symptomatic patients. Approximately 80%–90% of patients are completely cured following surgery, but there is a relapse rate of 10%–20%.[23,36] The relapse rate of CSDH has been extensively researched as it is a major prognostic factor. Surgery is the universal treatment option for symptomatic CSDH and the outcomes of surgery can directly impact the prognosis; however, there is a lack of research on risk factors affecting short-term surgical outcomes.

Thus, the objective of this study was to examine the relationships between several factors affecting short-term postoperative outcomes and to identify significant preoperative risk factors. To this end, we compared pre- and postoperative images from a group of patients who had undergone surgery for CSDH.

MATERIALS AND METHODS

Patient characteristics

This was a retrospective study on 172 patients who had visited the emergency room at our hospital and undergone burr-hole craniostomy with closed-system drainage for CSDH between January 2019 and December 2021. After making a single burr hole, a drainage catheter was inserted into the hole, and a closed drainage system was kept in place postoperatively. Patients with organized CSDH, bilateral lesions, or acute or subacute hematoma were excluded. Surgical procedures other than single burr-hole craniostomy were also excluded, such as patients who had undergone intraoperative lavage or double burr-hole craniostomy.

We set the independent variables as follows: 1) patient characteristics, including sex, 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), medication history (i.e., antiplatelets and anticoagulants); 2) initial laboratory findings (creatine kinase [CK], lactate dehydrogenase [LDH], calcium, phosphate, glucose, platelets, sodium, and osmolality); 3) 3-day mean arterial pressure; and 4) postoperative ambulation.

In addition, the contralateral midline shift and maximal hematoma thickness were measured from preoperative computed tomography (CT) scans and were compared with the midline shift and maximal thickness of the subdural space at the lesion site via CT scans 3-day postoperatively. The prognosis was evaluated by hematoma thickness change and the degree of brain re-expansion by midline shifting change. Here, whether the postoperative decrease in thickness was greater than or less than 60% and whether the midline recovery was greater than or less than 75% were also used as dependent variables.

In addition, the contralateral midline shift and maximal hematoma thickness were measured from preoperative CT scans and were compared with the midline shift and maximal thickness of the subdural space at the lesion site via CT scans 3-day postoperatively to analyze factors affecting the drainage of the subdural hemorrhage and brain re-expansion, respectively. We also examined the 3-month relapse rate at outpatient follow-up visits. This retrospective study was approved by the Institutional Review Board of Wonju Severance Christian Hospital (CR 323055).

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Statistical analysis

Data were expressed according to the properties of the variable. Continuous variables were presented as means and standard deviations. Categorical variables were presented as frequencies and percentages. To compare the 2 groups, we performed univariate analysis or the χ^2 test (Fisher's exact test), as appropriate. Logistic regression analysis was performed to identify the factors to predict hematoma thickness or midline shift, and the results were expressed as odds ratios with 95% confidence intervals. A *p*-value less than 0.05 was considered statistically significant and all statistical analyses were performed using Statistical Package for the Social Sciences (version 24; IBM Corp., Armonk, NY, USA).

RESULTS

Of the 172 participants, there were more male patients (114 males, 67.1% vs. 58 females, 32.9%) and the mean age was 72.4 years (**TABLE 1**). These factors did not have statistically significant effects on hematoma thickness or midline shift. Renal disease (p=0.044) and intracranial hemorrhage (p=0.004) showed significant effects on the postoperative change in hematoma thickness but did not significantly affect midline shift. Hypertension, coronary artery disease, and other comorbidities showed no significant effects on either hematoma thickness or midline shift (**TABLES 2** & **3**).

TABLE 1. Patient characteristics of patient with chronic subdural hematoma

Characteristics	Total (n=170)
Sex (male)	114 (67.1)
Age (years)	72.4±12.5
History	
Hypertension	101 (59.4)
Diabetes mellitus	44 (25.9)
Cardiovascular disease	24 (14.1)
Respiratory disease	21 (12.4)
Renal disease	15 (8.8)
Liver disease	14 (8.2)
Dementia	19 (11.2)
Infarction	17 (10.0)
Intracranial hemorrhage	32 (18.8)
Cancer	24 (14.1)
Antithrombotic medication	51 (32.1)
Laboratory finding (initial)	
Creatine kinase (IU/L)	188.5±348.4
Lactate dehydrogenase (IU/L)	267.9±77.2
Calcium (mmol/L)	9.4±0.5
Phosphate (mmol/L)	3.4±0.7
Glucose (mg/dL)	129.8±48.7
Platelet (10^{*3}/uL)	246.0±95.8
Serum sodium (mmol/L)	140.9±3.8
Osmole (mOsm/kg)	292.3±7.5
3-day mean MAP	96.0±12.1
Postoperative ambulation	91 (53.5)
Preoperative CT finding	
Hematoma thickness	19.7±5.6
Midline shifting	14.8±82.0
Recurrence	30 (17.6)

Values are presented as number (%) or mean ± standard deviation.
MAP: mean arterial pressure, CT: computed tomography.

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TABLE 2. Predictors of unfavorable prognosis after burr-hole trephination for hematoma thickness

Characteristics	Hematoma thickness change		Univariate analysis	Multivariate analysis	
	<60% (n=94)	≥60% (n=76)		*p*-value	OR (95% CI)
Sex (male)	59 (62.8)	55 (72.4)	0.185	0.200	0.622 (0.301–1.285)
Age (year)	71.0±13.5	74.2±11.1	0.090	0.182	1.019 (0.991–1.049)
History					
Hypertension	56 (59.6)	45 (59.2)	0.962		
Diabetes mellitus	26 (27.7)	18 (23.7)	0.556		
Cardiovascular disease	15 (16.0)	9 (11.8)	0.444		
Respiratory disease	11 (11.7)	10 (13.2)	0.774		
Renal disease	12 (12.8)	3 (3.9)	0.044	0.064	0.276 (0.071–1.077)
Liver disease	9 (9.6)	5 (6.6)	0.480		
Dementia	8 (8.5)	11 (14.5)	0.220		
Infarction	10 (10.6)	7 (9.2)	0.758		
Intracranial hemorrhage	25 (26.6)	7 (9.2)	0.004	0.024	0.328 (0.125–0.865)
Cancer	12 (12.8)	12 (15.8)	0.574		
Antithrombotic medication	28 (31.1)	23 (33.3)	0.766		
Laboratory finding (initial)					
CK	127.6±136.1	262.0±487.2	0.025		
CK >188	14 (15.9)	22 (30.1)	0.031	0.108	1.948 (0.863–4.395)
Lactate dehydrogenase (IU/L)	270.4±83.9	265.0±68.7	0.659		
Calcium (mmol/L)	9.4±0.5	9.4±0.5	0.732		
Phosphate (mmol/L)	3.4±0.7	3.4±0.7	0.623		
Glucose (mg/dL)	132.0±50.2	127.0±47.0	0.504		
Platelet (10^{*3}/uL)		228.9±83.1	0.036		
Platelet >246K	49 (52.1)	26 (34.2)	0.019	0.012	0.413 (0.207–0.822)
Serum sodium (mmol/L)	140.7±3.8	141.1±3.8	0.501		
Osmole (mOsm/kg)		293.3±7.5	0.123		
3-day mean MAP	97.2±12.9	94.5±11.1	0.138		
Postoperative ambulation	51 (54.3)	40 (52.6)	0.833		
Preoperative CT finding					
Hematoma thickness	19.7±5.3	19.7±6.0	0.987		
Midline shifting	19.8±110.2	8.6±3.9	0.379		

OR: odds ratio, CI: confidence interval, CK: creatine kinase, MAP: mean arterial pressure, CT: computed tomography.

For the initial laboratory findings, higher levels of CK (p=0.025, p=0.034), and lower platelet levels (p=0.036) showed significant correlations with changes in hematoma thickness and midline shift. Other laboratory findings, including LDH and serum sodium levels, showed no significant correlations (**TABLES 2** & **3**)

We predicted that an increase in mean postoperative blood pressure would result in good outcomes by helping with brain expansion; however, lower 3-day postoperative mean arterial pressure (MAP) (p=0.026) showed a significant correlation with midline shift. Meanwhile, postoperative ambulation and preoperative CT findings showed no statistically significant correlations with short-term postoperative outcomes (**TABLES 2** & **3**).

To further evaluate duplicate variables, we performed a multivariate analysis which included initial platelet count and history of brain hemorrhage as independent risk factors for change in hematoma thickness and 3-day postoperative MAP as an independent risk factor for change in midline shift.

Finally, a change of at least 60% in hematoma thickness (p=0.003) and a change of at least 75% in midline shift (p=0.015), which are representative of short-term postoperative outcomes for CSDH, showed statistically significant correlations with long-term postoperative relapse (**TABLE 4**).

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TABLE 3. Predictors of unfavorable prognosis after burr-hole trephination for mid-line shifting change

Characteristics	Midline shifting change		Univariate analysis	Multivariate analysis	
	<75% (n=79)	≥75% (n=91)		*p*-value	OR (95% CI)
Sex (male)	49 (62.0)	65 (71.4)	0.193	0.099	0.546 (0.266–1.120)
Age (year)	71.2±14.4	73.5±10.6	0.249	0.385	1.012 (0.985–1.041)
History					
Hypertension	51 (64.6)	50 (54.9)	0.203		
Diabetes mellitus	22 (27.8)	22 (24.2)	0.586		
Cardiovascular disease	8 (10.1)	16 (17.6)	0.164		
Respiratory disease	12 (15.2)	9 (9.9)	0.295		
Renal disease	8 (10.1)	7 (7.7)	0.577		
Liver disease	7 (8.9)	7 (7.7)	0.782		
Dementia	8 (10.1)	11 (12.1)	0.686		
Infarction	9 (11.4)	8 (8.8)	0.573		
Intracranial hemorrhage	17 (21.5)	15 (16.5)	0.402		
Cancer	8 (10.1)	16 (17.6)	0.164		
Antithrombotic medication	23 (30.3)	28 (33.7)	0.639		
Laboratory finding (initial)					
CK	129.2±126.5	237.8±452.3	0.034	0.059	1.002 (1.000–1.004)
CK >188	11 (15.1)	25 (28.4)	0.043		
Lactate dehydrogenase (IU/L)	266.3±68.9	269.2±83.8	0.816		
Calcium (mmol/L)	9.4±0.5	9.4±0.6	0.774		
Phosphate (mmol/L)	3.4±0.7	3.4±0.8	0.599		
Glucose (mg/dL)	132.6±52.4	127.3±45.5	0.481		
Platelet (10^{*3}/uL)		236.4±94.6	0.162		
Platelet >246K	39 (49.4)	36 (39.6)	0.199		
Serum sodium (mmol/L)	141.0±3.4	141.0±4.1	0.689		
Osmole (mOsm/kg)		292.5±8.2	0.784		
3-day mean MAP	98.2±12.8	94.1±11.3	0.026	0.041	0.969 (0.941–0.999)
Postoperative ambulation	38 (48.1)	53 (58.2)	0.186		
Preoperative CT finding					
Hematoma thickness	20.6±5.3	18.8±5.8	0.042	0.052	0.942 (0.887–1.001)
Midline shifting	8.6±3.7	20.2±112.0	0.359		

OR: odds ratio, CI: confidence interval, CK: creatine kinase, MAP: mean arterial pressure, CT: computed tomography.

TABLE 4. Correlation between postoperative change values and chronic subdural hematoma recurrence

Characteristics	Recurrence (n=30)	No recurrence (n=140)	*p*-value
Hematoma thickness change ≥60%	6 (20.0)	70 (50.0)	0.003
Midline shifting change ≥75%	10 (33.3)	81 (57.9)	0.015

DISCUSSION

Due to population aging, the incidence of CSDH is gradually increasing.[8,28] According to Feghali et al.,[8] the peak age of CSDH is also increasing towards 80–89 years. CSDH is generally a condition affecting the elderly after mild trauma.[2,7,13,17,29] and while it can be completely cured with surgery, there is a high rate of postoperative relapse.[2,15] The postoperative prognosis for CSDH is diverse and there have been various studies on the factors affecting prognosis during treatment after the initial diagnosis of CSDH. However, the results of these studies are inconsistent. Given that treatment methods for CSDH and its prognosis are important topics in neurosurgery, efforts are needed to improve the situation.

In CSDH, a laminated, encapsulated collection of fluid, blood, and products of hemolysis forms between the arachnoid mater and the dura mater.[6] Characteristically, the initial diagnosis is made from head CT scans and the hematoma appears as a crescent-shaped layer in the subdural space.[22,34] Hemosiderin and hemochrome, produced from the degradation of hemoglobin, generally appear hypodense in CT scans.[34] However, while CSDH usually

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appears as a homogenous hypodense mass in CT scans, it can have diverse presentations. Nakaguchi et al.[24] categorized CSDH into homogeneous, laminar, separated, and trabecular types, based on the density regularity. This is because the appearance changes depending on the extent of coagulative and fibrinolytic activities in the natural progression of CSDH,[19,24,25] and different appearances have been reported to show differences in re-bleeding and recurrence rates.[3,10,16,19,24,26,30,31]

Patients with symptomless CSDH are generally managed conservatively through careful follow-up observation,[8,22] whereas surgical intervention is performed for patients with radiological evidence of pressure on the brain and clinical symptoms. Various surgical interventions have been attempted, including twist-drill craniostomy, craniotomy, single or double burr-hole craniostomy, membranectomy after dura incision, and hematoma irrigation,[14] but Markwalder et al.[21] reported that single burr-hole craniostomy was the most commonly used method. In single burr-hole craniostomy, with the patient under general anesthetic, a single burr hole is drilled into the skull, followed by membranectomy and drain catheter insertion.[8,21]

CSDH has low morbidity and mortality after surgical intervention.[2,15] Nevertheless, the relapse rate is an important factor in the prognosis of patients with CSDH. There have been several previous studies on CSDH relapse. According to Cofano et al.,[4] age, use of antithrombotic agents, and laterality were not correlated with postoperative risk of CSDH relapse; burr-hole craniostomy showed the lowest relapse risk of the analyzed surgical methods; and drain catheter insertion and dexamethasone were also significantly correlated with reduced risk of relapse. In a meta-analysis by Zhu et al.,[37] 32 relapse risk factors were reported, including sex, age, use of antithrombotic agents, use of drainage, and differences in initial CT scans (laterality, hematoma width, midline shift, etc.). Male sex, bilateral hematoma, and no drainage were reported to be especially convincing as risk factors, with class I evidence.

Most previous studies have focused on risk factors for long-term prognosis, such as postoperative relapse. However, there have been no studies examining how the risk factors for long-term prognosis also affect short-term postoperative outcomes. As such, the objective of this study was to investigate the effects of risk factors associated with CSDH prognosis in previous studies on short-term postoperative outcomes. Mehta et al.[22] reported that perioperative anticoagulant/antiplatelet therapy, poor postoperative brain re-expansion, presence of thick membranes, persistent midline shift, and pneumocephalus are important factors with regard to the relapse and prognosis of CSDH.[18] Complex, interrelated, pathophysiological processes, such as inflammation, membrane formation, angiogenesis, and fibrinolysis, act to further increase hematoma volume after trauma.[6] By draining several inflammatory factors involved in these mechanisms out of the hematoma (such as interleukin-1, -6, -8, and -10), it is possible to reduce the relapse rate of CSDH, which is an important factor affecting patients' long-term outcomes.[1,12,20,22,32] Thus, the short-term postoperative radiological prognosis offers insight into patients' long-term prognosis.

Similarly, brain re-expansion is known to be an important prognostic factor. Jang et al.[14] reported that depressed brain volume of over 50 cm^3 in 7-day postoperative CT scans is an important factor in relapse and can help determine postoperative treatment strategies for patients with CSDH. Stanisic et al.[30] Reported that pneumocephalus, cerebral atrophy, and increased brain surface elastance are factors that perpetuate the subdural space and hinder

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brain re-expansion.[5,9,14,35] Pneumocephalus is suspected to affect relapse by preventing brain expansion and enabling the hematoma cavity to persist.[14,27] Cerebral atrophy is correlated with relapse because it maintains the subdural space due to excessive fibroblastic processes and suppresses brain expansions.[11,14,33] Finally, Jang et al.[14] reported that persistent mechanical compression of the brain surface in patients who have undergone hematoma evacuation increases brain surface elastance, causes a persistent subdural cavity, and results in a higher relapse rate.

Based on the above results, we believe that hematoma drainage and brain re-expansion are important factors affecting the short-term postoperative outcomes of CSDH. We found that patients with a history of renal disease or brain lesions do not respond well and patients with low initial platelet counts responded well to subdural hemorrhage drainage. Meanwhile, 3-day MAP and postoperative ambulation, which were initially expected to affect brain re-expansion, showed no statistically significant effects.

Limitations and strengths

This was a single-center, retrospective study on a relatively small sample of 170 patients. Patients were followed up for 6 months after discharge, but follow-up was stopped if patients showed no relapse within 3 postoperative months. Therefore, we were unable to observe the relapse rate of patients over long-term follow-up. In addition, all patients were evaluated based on CT scans and only hematomas with a homogenous appearance were included. In other words, we did not evaluate patients using different modalities, such as magnetic resonance imaging, or different stages of CSDH progression. In addition, since the patients were evaluated only by imaging tests on the third postoperative day, further evaluation of changes in imaging tests may be required. Nevertheless, the strength of this study is that it is the first, to the best of our knowledge, to examine factors affecting short-term postoperative prognosis (brain expansion and subdural hematoma shrinkage) for CSDH.

CONCLUSION

Patients with symptomatic CSDH are usually treated surgically. As such, there have been several studies on the need for follow-up and for evaluating factors related to postoperative prognosis. In this study, we showed that low platelet counts were closely related to postoperative good prognosis and a history of renal disease or brain lesions were related to poorer prognosis. This suggests that patients with CSDH with the above characteristics will need to receive a detailed explanation of the risks before surgery and should be even more closely followed up and monitored.

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