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Original Article

Risk factors for subdural hematoma in spontaneous intracranial hypotension are male sex, longer disease duration, and repeated epidural blood patches

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Abstract: Purpose: To investigate the clinical characteristics and risk factors for subdural hematoma (SDH) in patients with spontaneous intracranial hypotension (SIH), with a focus on male sex, disease duration, and repeated epidural blood patch (EBP) procedures. Methods: This retrospective cohort study included 579 consecutive SIH patients admitted between June 2019 and November 2025. Patients were divided into an SDH group (n = 101) and a non-SDH group (n = 478) based on brain imaging. Demographic data, clinical characteristics, laboratory parameters, brain imaging features, spinal imaging findings, and treatment details were collected. Results: SDH patients were more frequently male (60.4% vs. 32.4%, P < 0.001), older (45.4 ± 12.7 vs. 41.6 ± 9.5 years, P = 0.005), and had longer disease duration (52.3 ± 23.1 vs. 39.2 ± 18.3 days, P < 0.001). They exhibited higher systolic blood pressure (122.7 ± 16.4 vs. 118.8 ± 16.4 mmHg, P = 0.033) and more neurological deficits (30.7% vs. 10.3%, P < 0.001). Brain imaging showed increased frequency of brain sagging (55.5% vs. 40.8%, P = 0.007), dural enhancement (80.2% vs. 64.4%, P = 0.002), venous distension (56.4% vs. 39.1%, P = 0.001), and subdural effusion (36.6% vs. 25.5%, P = 0.023) in the SDH group. Spinal imaging revealed more identifiable leaks (93.1% vs. 77.8%, P < 0.001). The SDH group underwent more EBP procedures (1.43 ± 0.64 vs. 1.23 ± 0.51, P = 0.004). Multivariate analysis identified male sex, age, disease duration, dural enhancement, and number of EBP procedures as independent SDH risk factors (P < 0.05). Conclusion: Male sex, longer disease duration, dural enhancement on brain imaging, and repeated EBP procedures were independent risk factors for SDH in patients with SIH, highlighting a need for close monitoring in high-risk individuals.

Keywords: Spontaneous intracranial hypotension, subdural hematoma, male, disease duration, epidural blood patch

Introduction

Spontaneous intracranial hypotension (SIH) is a neurological condition characterized by low CSF pressure, typically resulting from a CSF leak. SIH has an estimated incidence of 5 per 100,000 individuals annually, although this figure may be underestimated due to underdiagnosis [1, 2]. The pathophysiology of SIH involves CSF leakage, which leads to reduced intracranial pressure and subsequent brain sagging. This mechanical stress can cause traction on pain-sensitive structures, leading to headache. This headache worsens upon assuming an upright position and improves with recumbency, though atypical headache patterns and associated symptoms such as nausea, vomiting, tinnitus, and neck stiffness are also commonly observed [3, 4].

Beyond the characteristic headache, SIH can lead to serious neurological complications, among which subdural hematoma (SDH) represents one of the most consequential. The underlying pathophysiologic mechanism involves downward displacement of the brain due to reduced CSF buoyancy, which exerts traction on intracranial bridging veins, rendering them susceptible to tearing and subsequent hemorrhage [5, 6]. The reported prevalence of SDH among patients with SIH varies considerably across studies, with hematomas often present-

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ing bilaterally and ranging from thin hygromas to large space-occupying collections requiring surgical intervention. While small, asymptomatic SDH may resolve spontaneously following successful treatment of the underlying CSF leak, larger hematomas with significant mass effect can precipitate neurological deterioration, including consciousness disturbance, focal deficits, and, in extreme cases, life-threatening brain herniation [7-9].

SIH has historically been more commonly diagnosed in women, possibly due to hormonal influences affecting connective tissue strength and CSF dynamics [10]. However, recent studies suggest that males may be at higher risk for certain complications, including SDH. This gender disparity highlights the need for further investigation into biological or behavioral factors contributing to differential risk profiles [5, 11]. Additionally, disease duration plays a role in SIH severity, with prolonged exposure to low CSF pressure possibly leading to repeated episodes of CSF leakage and exacerbating complications. Early diagnosis and intervention are crucial, but delays in seeking medical attention can prolong the disease course [12, 13]. Epidural blood patching (EBP) is a widely used treatment aimed at sealing CSF leaks, but repeated procedures may introduce additional trauma, potentially increasing SDH risk. Understanding these factors is essential for optimizing patient care and identifying those who may benefit from early or more aggressive treatment strategies [14].

SIH is a complex condition with significant clinical implications, particularly when complicated by SDH. Male sex, prolonged disease duration, and repeated EBP procedures emerged as key areas of interest for further investigation. In addition to these clinical factors, specific brain imaging features - particularly dural enhancement - may also predict SDH risk, but their role has not been systematically evaluated. By focusing on these aspects, future research can better elucidate the factors contributing to SDH development in SIH and inform clinical practice guidelines. Improving our knowledge of these risk factors will ultimately enhance patient care and reduce the burden of this serious complication. This study aims to address these gaps by examining the clinical features and risk predictors of SDH in patients with SIH, providing valuable insight for both clinicians and researchers.

Materials and methods

Design and patients

This retrospective cohort study was conducted at Sir Run Run Shaw Hospital, affiliated with Zhejiang University School of Medicine. Consecutive 579 patients diagnosed with SIH who were admitted between June 2019 and November 2025 were retrospectively reviewed. Inclusion criteria were: (1) diagnosis of SIH according to ICHD-3 criteria [15], requiring the presence of low intracranial pressure (cerebrospinal fluid pressure < 60 mmH_2O) and/or neuroimaging evidence of CSF leakage; (2) headache temporally associated with low intracranial pressure or CSF leakage; (3) complete medical records. Exclusion criteria were: (1) secondary intracranial hypotension due to lumbar puncture, epidural anesthesia, or craniospinal trauma; (2) patients with multiple admissions (only the first admission was included); (3) incomplete clinical or imaging data that prevented comprehensive analysis of risk factors.

Study approved by Institutional Ethics Committee of Sir Run Run Shaw Hospital, Zhejiang University School of Medicine (No. 0622, 2023). Requirement for written informed consent was waived due to the retrospective nature.

Data collection and variable definitions

Demographic and clinical data were extracted from medical records using data collection form. All data were verified by 2 researchers.

Demographic and baseline characteristics: Demographic variables included age at admission (calculated from date of birth), sex (male/female), BMI, smoking, alcohol, and comorbidities. History of prior treatment for SIH before admission was defined as documented attempts at conservative management (bed rest, hydration, analgesics) or EBP procedures performed at other institutions prior to referral. Of note, EBPs performed at outside institutions were counted only in this history variable and were not included in the count of “number of EBP procedures before SDH”.

Clinical characteristics at presentation: Disease duration was defined as interval from symptom onset (typically the first occurrence of orthostatic headache) to hospital admission, recorded in days. Headache intensity was assessed at admission using NRS, a 0 (no pain) - 10 (worst imaginable pain) scale. Orthostatic headache was defined as headache occurring within 15 minutes of assuming an upright position and improving or resolving within 30 minutes of lying down. Associated neurological deficits were systematically recorded and categorized into three types: consciousness disturbance (lethargy, somnolence, or coma), cranial nerve palsy (diplopia, facial weakness, or dysphagia), and gait disturbance (unsteadiness or ataxia).

Blood pressure was measured at admission using a calibrated automated sphygmomanometer (TERUMO Elemano ES*H5501, Terumo (Hangzhou) Co., Ltd., Hangzhou, China) after the patient had rested in a supine position for at least 10 minutes. The device was calibrated by the Zhejiang Institute of Metrology.

Laboratory measurements: Complete blood count was performed using an automated hematology analyzer (BC-6800Plus, Shenzhen Mindray Bio-Medical Electronics Co., Ltd., Shenzhen, China), with hemoglobin concentration expressed in g/L and platelet count as $\times 10^9$/L. Coagulation data, including PT and APTT, were measured using an automated coagulation analyzer (STA R MAX, STAGO, France) and reported in seconds. Fibrinogen concentration was determined using Clauss method and expressed in g/L.

Neuroimaging assessment: All patients underwent standardized neuroimaging protocols. Brain imaging was performed using either CT or MRI. CT scans were acquired using a 64-slice multidetector CT scanner (Somatom Definition AS, Siemens Healthcare, Erlangen, Germany) with contiguous 5 mm axial slices. MRI was performed on a 3.0 Tesla scanner (Magnetom Skyra, Siemens Healthcare, Erlangen, Germany) with a standardized protocol including T1-weighted, T2-weighted, FLAIR, and contrast-enhanced T1-weighted sequences after intravenous administration of gadolinium-based contrast agent (0.1 mmol/kg body weight).

The following brain imaging characteristics were evaluated by two neuroradiologists blinded to clinical data: (1) presence and location of subdural hematoma (unilateral or bilateral); (2) maximum hematoma thickness measured perpendicular to the inner table of the skull (mm); (3) presence of midline shift ≥ 5 mm; (4) brain sagging features, defined as downward displacement of the brain with effacement of suprasellar and prepontine cisterns, descent of cerebellar tonsils, and flattening of the optic chiasm; (5) dural enhancement, defined as diffuse, continuous, non-nodular enhancement of the cranial dura mater on contrast-enhanced T1-weighted images; (6) venous distension sign, defined as prominence or enlargement of the superior ophthalmic vein or dural venous sinuses; and (7) presence of subdural effusion/hygroma prior to hematoma formation, defined as thin, bilateral fluid collections over the cerebral convexities with signal intensity similar to CSF.

Spinal imaging was performed to identify and characterize CSF leaks. All patients underwent spinal MRI with heavily T2-weighted sequences (MR myelography) to detect extradural fluid collections. Leak site identification was categorized as: (1) no identifiable leak; (2) single-level leak; or (3) multiple-level leak.

Treatment modalities: Conservative treatment included strict bed rest in the horizontal position, adequate oral and intravenous hydration, and analgesics as clinically indicated (compound paracetamol or celecoxib orally once daily or as needed). Patients were advised to avoid activities that might exacerbate intracranial hypotension, including straining, bending, heavy lifting, and Valsalva maneuvers.

EBP procedures were performed by experienced anesthesiologists. Under strict aseptic technique and fluoroscopic guidance, autologous venous blood was drawn from the patient's antecubital vein and injected into epidural space at level corresponding to the suspected CSF leak (targeted EBP) or at the lumbar level when the leak site could not be identified (non-targeted EBP). The injected blood volume was determined by the anesthesiologist based on clinical response, and the procedure was terminated upon patient report of back pressure, radicular pain, or headache recurrence. The number of EBP procedures performed before the diagnosis of subdural hematoma was recorded. This variable included only EBPs performed at our institution; any EBPs performed

at outside institutions before referral were excluded from this count and captured separately under "history of prior treatment for SIH". This distinction was made to avoid double-counting and to minimize collinearity between the two variables.

History of prior treatment for SIH before admission was defined as documented attempts at conservative management (bed rest, hydration, analgesics) or EBP procedures performed at other institutions prior to referral. Length of hospital stay was recorded in days from admission to discharge.

Grouping strategy

Patients were divided into 2 groups based on presence or absence of subdural hematoma on brain imaging. The non-SDH group (n = 478) comprised patients with SIH without evidence of SDH on either initial or follow-up imaging during hospitalization. The SDH group (n = 101) comprised patients with SIH who developed SDH, documented on brain CT or MRI at any time during their clinical course.

Statistical analysis

Statistical testing employed SPSS 26.0 (IBM, Armonk, NY). Normality was assessed by Shapiro-Wilk test. All continuous measures were normal, thus shown as means ± SD and intergroup comparisons made with independent t-tests. Categorical measures were given as counts/percentages, with group differences evaluated by chi-square or Fisher's exact where appropriate.

The primary outcome was the presence of SDH on brain imaging. Primary risk factors of interest were male sex, disease duration, and number of EBP procedures; secondary variables included imaging features (dural enhancement, brain sagging) and other clinical data.

Univariate logistic regression analysis was performed to identify factors associated with the development of SDH in SIH patients. Variables with $P < 0.10$ in univariate analysis were considered candidate predictors for multivariate analysis. Ten variables were selected for inclusion in multivariate logistic regression model: age, sex, disease duration, systolic blood pressure, subdural effusion/hygroma, dural enhancement, brain sagging features, identifiable CSF leak, number of pre-SDH EBP procedures, and history of prior SIH treatment. Associated neurological deficits, midline shift ≥ 5 mm, and length of hospital stay were excluded because they represent consequences rather than predictors of SDH and could introduce reverse causality bias. Venous distension sign was excluded due to strong collinearity with dural enhancement. The categorical form of EBP procedures was not entered separately as the continuous variable was already included.

Multivariable logit modeling identified determinants of SDH. Findings were ORs (95% CIs); threshold $P < 0.05$ (two-tailed). The discrimination performance of final multivariate logistic regression model was assessed using AUC (ROC), and fit by means of the Hosmer–Lemeshow test; $P > 0.05$ resulting from this test suggested acceptable calibration.

Results

Demographic and clinical characteristics

Patients in the SDH group were significantly older than those in non-SDH group (45.38 ± 12.69 years vs. 41.58 ± 9.50 years, $P = 0.005$) and had a higher proportion of male sex (60.4% vs. 32.43%, $P < 0.001$) (**Table 1**). No significant differences were observed between 2 groups regarding body mass index, smoking, alcohol consumption, hypertension, diabetes mellitus, or cardiovascular disease (all $P > 0.05$).

Clinical characteristics at presentation are summarized in **Table 2**. SDH group had a significantly longer disease duration (52.28 ± 23.14 days vs. 39.16 ± 18.26 days, $P < 0.001$) and higher systolic blood pressure (122.68 ± 16.44 mmHg vs. 118.84 ± 16.36 mmHg, $P = 0.033$) compared to the non-SDH group. Associated neurological deficits were markedly more frequent in SDH group (30.69% vs. 10.25%, $P < 0.001$). Diastolic blood pressure, headache intensity, and proportion of orthostatic headache did not differ significantly ($P > 0.05$).

Laboratory parameters

Laboratory values at admission are shown in **Figure 1**. There were no significant differences between the SDH and non-SDH groups in hemoglobin level, platelet count, prothrombin time, activated partial thromboplastin time, or fibrinogen concentration (all $P > 0.05$).

Table 1. Demographic characteristics of SIH patients with and without subdural hematoma

Variable	Non-SDH Group (n = 478)	SDH Group (n = 101)	t/χ^2	P
Age (years)	41.58 ± 9.50	45.38 ± 12.69	2.847	0.005
Male sex, n (%)	155 (32.43%)	61 (60.4%)	27.889	< 0.001
Body mass index (kg/m^2)	22.84 ± 3.12	23.41 ± 3.28	1.673	0.095
Smoking, n (%)	103 (21.55%)	28 (27.72%)	1.816	0.178
Alcohol consumption, n (%)	72 (15.06%)	21 (20.79%)	2.030	0.154
Hypertension, n (%)	78 (16.32%)	21 (20.79%)	1.177	0.278
Diabetes mellitus, n (%)	34 (7.11%)	10 (9.9%)	0.923	0.337
Cardiovascular disease, n (%)	28 (5.86%)	8 (7.92%)	0.609	0.435

SIH, spontaneous intracranial hypotension; SDH, subdural hematoma; BMI, body mass index.

Table 2. Clinical characteristics at presentation

Variable	Non-SDH Group (n = 478)	SDH Group (n = 101)	t/χ^2	P
Disease duration (days)	39.16 ± 18.26	52.28 ± 23.14	5.355	< 0.001
Systolic blood pressure (mmHg)	118.84 ± 16.36	122.68 ± 16.44	2.139	0.033
Diastolic blood pressure (mmHg)	73.28 ± 11.42	75.52 ± 11.46	1.789	0.074
Headache intensity (NRS 0-10)	5.92 ± 2.24	6.14 ± 2.28	0.895	0.371
Orthostatic headache, n (%)	414 (86.61%)	86 (85.15%)	0.151	0.697
Associated neurological deficits, n (%)	49 (10.25%)	31 (30.69%)	29.261	< 0.001
Consciousness disturbance	24	20		
Cranial nerve palsy	16	6		
Gait disturbance	9	5		

SDH, subdural hematoma; NRS, Numerical Rating Scale.

Brain imaging characteristics

Brain imaging characteristics are detailed in **Table 3**. Among patients with SDH, bilateral hematomas were predominant (79.21%), with a mean maximum thickness of 12.64 ± 6.48 mm. Midline shift ≥ 5 mm was present in 34.65% of SDH patients, while none in the non-SDH group exhibited this finding (P < 0.001). Brain sagging features, dural enhancement, venous distension sign, and subdural effusion/hygroma prior to SDH were all significantly more common in the SDH group than in the non-SDH group (55.45% vs. 40.79%, P = 0.007; 80.2% vs. 64.44%, P = 0.002; 56.44% vs. 39.12%, P = 0.001; and 36.63% vs. 25.52%, P = 0.023, respectively).

Spinal imaging findings and cerebrospinal fluid leak characteristics

Spinal imaging findings regarding cerebrospinal fluid leak identification are presented in **Table 4**. The distribution of leak sites differed significantly between groups (P < 0.001). The SDH group had a lower proportion of patients with no identifiable leak (6.93% vs. 22.18%) and a higher proportion with single-level leaks (81.19% vs. 62.55%), while multiple-level leaks were comparable (11.88% vs. 15.27%).

Treatment characteristics

Treatment characteristics and outcomes are shown in **Table 5**. Patients in the SDH group underwent significantly more epidural blood patch procedures before SDH diagnosis (1.43 ± 0.64 vs. 1.23 ± 0.51, P = 0.004) and had a higher frequency of multiple EBP procedures (P < 0.001). A history of prior treatment for SIH was more common in the SDH group (28.71% vs. 18.2%, P = 0.016). Length of hospital stay was also significantly longer in the SDH group (16.72 ± 8.14 days vs. 13.56 ± 5.98 days, P < 0.001).

Univariate and multivariate logistic regression

Univariate logistic regression analysis (**Table 6**) revealed that age (P < 0.001), male sex (P < 0.001), disease duration (P < 0.001), systolic blood pressure (P = 0.034), subdural effusion/

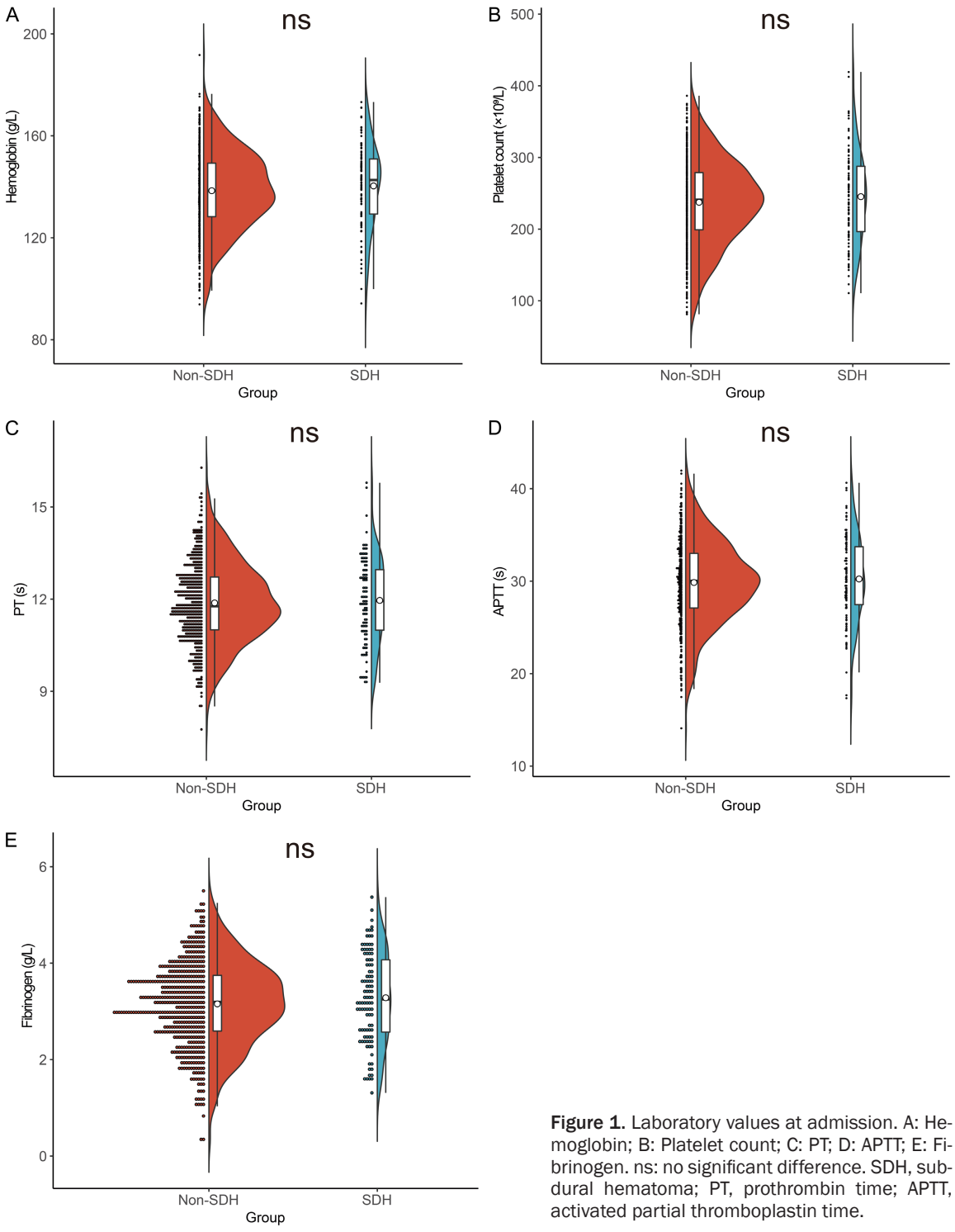

Figure 1. Laboratory values at admission. A: Hemoglobin; B: Platelet count; C: PT; D: APTT; E: Fibrinogen. ns: no significant difference. SDH, subdural hematoma; PT, prothrombin time; APTT, activated partial thromboplastin time.

hygroma (P = 0.007), dural enhancement (P = 0.003), brain sagging features (P = 0.024), identifiable CSF leak (P < 0.001), number of pre-SDH EBP procedures (P < 0.001), and history of prior SIH treatment (P = 0.018) were each significantly associated with an increased risk of SDH.

Multivariate logistic regression analysis (**Table 7**) identified male sex (OR 3.03, 95% CI 1.85-4.98, P < 0.001), age (OR 1.04, 95% CI 1.01-1.06, P = 0.006), disease duration (OR 1.04, 95% CI 1.02-1.05, P < 0.001), dural enhancement (OR 2.29, 95% CI 1.29-4.08, P = 0.005), and number of pre-SDH EBP procedures (OR

Table 3. Brain imaging characteristics

Variable	Non-SDH Group (n = 478)	SDH Group (n = 101)	χ^2/Fisher	P
Subdural hematoma location, n (%)				
Unilateral	/	21 (20.79)		
Bilateral	/	80 (79.21)		
Maximum hematoma thickness (mm)	/	12.64 ± 6.48		
Midline shift ≥ 5 mm, n (%)	0 (0%)	35 (34.65%)	/	< 0.001
Brain sagging features, n (%)	195 (40.79%)	56 (55.45%)	7.288	0.007
Dural enhancement, n (%)	308 (64.44%)	81 (80.2%)	9.397	0.002
Venous distension sign, n (%)	187 (39.12%)	57 (56.44%)	10.252	0.001
Subdural effusion/hygroma prior to SDH, n (%)	122 (25.52%)	37 (36.63%)	5.167	0.023

SDH, subdural hematoma.

Table 4. Spinal imaging findings and cerebrospinal fluid leak characteristics

Variable	Non-SDH Group (n = 478)	SDH Group (n = 101)	χ^2	P
Leak site identification, n (%)			14.983	< 0.001
No identifiable leak	106 (22.18%)	7 (6.93%)		
Single-level leak	299 (62.55%)	82 (81.19%)		
Multiple-level leak	73 (15.27%)	12 (11.88%)		

SDH, subdural hematoma; CSF, cerebrospinal fluid.

Table 5. Treatment characteristics and outcomes

Variable	Non-SDH Group (n = 478)	SDH Group (n = 101)	t/χ^2	P
Number of EBP procedures before SDH	1.23 ± 0.51	1.43 ± 0.64	2.965	0.004
EBP procedures before SDH, n (%)			14.234	< 0.001
1 procedure	392 (82.01%)	66 (65.35%)		
2 procedures	76 (15.9%)	30 (29.7%)		
≥ 3 procedures	10 (2.09%)	5 (4.95%)		
History of prior treatment for SIH, n (%)	87 (18.2%)	29 (28.71%)	5.751	0.016
Length of hospital stay (days)	13.56 ± 5.98	16.72 ± 8.14	3.695	< 0.001

SDH, subdural hematoma; SIH, spontaneous intracranial hypotension; EBP, epidural blood patch.

Table 6. Univariate logistic regression analysis of factors associated with subdural hematoma in SIH patients

Variable	Coefficient	Std_Error	Wald Z	*P*_Value	OR	95% CI
Age (per year)	0.037	0.011	3.361	< 0.001	1.04	1.02-1.06
Male sex (vs. female)	1.156	0.226	5.123	< 0.001	3.18	2.05-4.98
Disease duration (per day)	0.034	0.006	5.755	< 0.001	1.04	1.02-1.05
Systolic blood pressure (per mmHg)	0.015	0.007	2.125	0.034	1.02	1.00-1.03
Subdural effusion/hygroma	0.591	0.221	2.678	0.007	1.81	1.17-2.79
Dural enhancement	0.804	0.267	3.009	0.003	2.24	1.35-3.87
Brain sagging features	0.523	0.232	2.258	0.024	1.69	1.07-2.65
Identifiable CSF leak	1.342	0.407	3.297	< 0.001	3.83	1.84-9.31
Number of EBP procedures before SDH	0.700	0.207	3.382	< 0.001	2.01	1.35-3.04
History of prior SIH treatment	0.593	0.250	2.375	0.018	1.81	1.10-2.93

SIH, spontaneous intracranial hypotension; SDH, subdural hematoma; β, regression coefficient; SE, standard error; OR, odds ratio; CI, confidence interval; CSF, cerebrospinal fluid; EBP, epidural blood patch.

Table 7. Multivariate logistic regression analysis of independent risk factors for subdural hematoma in SIH patients

Variable	Coefficient	Std_Error	Wald Z	*P*_Value	OR	95% CI
Age (per year)	0.035	0.013	2.744	0.006	1.04	1.01-1.06
Male sex (vs. female)	1.109	0.253	4.381	< 0.001	3.03	1.85-4.98
Disease duration (per day)	0.035	0.006	5.397	< 0.001	1.04	1.02-1.05
Systolic blood pressure (per mmHg)	0.015	0.008	1.877	0.060	1.02	1.00-1.03
Brain sagging features	0.412	0.250	1.647	0.100	1.51	0.93-2.47
Dural enhancement	0.830	0.294	2.819	0.005	2.29	1.29-4.08
Subdural effusion/hygroma	0.371	0.268	1.386	0.166	1.45	0.86-2.45
Identifiable CSF leak	0.480	0.340	1.994	0.157	1.62	0.83-3.15
Number of pre-SDH EBP procedures	0.757	0.234	3.234	0.001	2.13	1.35-3.38
History of prior SIH treatment	0.379	0.292	1.299	0.194	1.46	0.83-2.59

SIH, spontaneous intracranial hypotension; SDH, subdural hematoma; β, regression coefficient; SE, standard error; OR, odds ratio; CI, confidence interval; CSF, cerebrospinal fluid; EBP, epidural blood patch. Model performance: C-statistic = 0.812 (95% CI 0.769-0.855); Hosmer-Lemeshow $\chi^2 = 8.24$, df = 8, P = 0.411.

2.13, 95% CI 1.35-3.38, P = 0.001) as independent risk factors for SDH in SIH patients. Systolic blood pressure, brain sagging features, subdural effusion/hygroma, identifiable CSF leak, and history of prior SIH treatment were not independently associated with SDH risk in the multivariate model (all P > 0.05).

The final multivariate logistic regression model demonstrated good discrimination with a C-statistic of 0.812 (95% CI 0.769-0.855). The Hosmer-Lemeshow goodness-of-fit test yielded a chi-square value of 8.24 (degrees of freedom = 8, P = 0.411), indicating acceptable calibration and no significant lack of fit.

Discussion

Spontaneous intracranial hypotension (SIH) is a condition characterized by low CSF pressure, often due to a CSF leak. It frequently presents with orthostatic headaches but can also lead to more severe complications such as SDH. Development of SDH in patients with SIH is a critical issue that requires careful evaluation and management. This study aimed to identify clinical characteristics and risk factors associated with occurrence of SDH in patients with SIH. Our findings highlight several key differences between patients with and without SDH, providing insights into potential mechanisms underlying these observations.

Male sex was more prevalent in SDH group compared to non-SDH group. This gender disparity suggests that hormonal or anatomical differences may play a role in pathophysiology of SDH in SIH. Males tend to have larger brains relative to skull size, which could result in greater traction on bridging veins when there is a reduction in CSF volume. Additionally, hormonal influences such as testosterone levels might affect vascular fragility and the propensity for hemorrhage [16-18].

Another notable finding was longer disease duration observed in SDH group. Prolonged exposure to low CSF pressure likely increases the risk of venous distension and rupture, leading to the formation of SDH. Chronic CSF leakage causes sustained brain sagging, which stretches the bridging veins over time. This mechanical stress can weaken the vessel walls, making them more susceptible to rupture under normal physiological pressures [19]. Moreover, prolonged disease duration may be associated with repeated episodes of CSF leakage, further exacerbating the risk of SDH [20]. Understanding the temporal relationship between CSF leakage and SDH development is crucial for early intervention and prevention strategies.

Although systolic blood pressure was higher in SDH group on univariate analysis, it did not retain significance in the multivariate model, suggesting that its apparent effect was likely confounded by other variables such as age and disease duration. Therefore, routine blood pressure control may not be primary strategy for SDH prevention in SIH, though it remains

clinically prudent for general cardiovascular health [21-23].

Associated neurological deficits were more frequent in the SDH group, indicating that SDH can cause significant neurological impairment. The presence of neurological deficits suggests that SDH may exert mass effect, leading to midline shift and compression of adjacent structures. This mass effect can disrupt normal brain function, resulting in symptoms such as cognitive decline, motor weakness, and sensory disturbances [24-26]. Early detection and treatment of SDH are essential to prevent permanent neurological damage. Neuroimaging plays a critical role in identifying SDH and guiding appropriate interventions.

Brain imaging characteristics provided valuable insight into the underlying pathology. Bilateral hematomas were more common in the SDH group, suggesting a diffuse process affecting multiple regions of the brain. Midline shift was exclusively present in the SDH group, highlighting the severity of this complication. Features such as dural enhancement, venous distension sign, and subdural effusion/hygroma were also more prevalent in the SDH group. Dural enhancement may reflect inflammation or reactive changes in response to chronic CSF leakage, while venous distension indicates increased intracranial venous pressure. Subdural effusions or hygromas represent another form of fluid accumulation, which can predispose to SDH formation. These imaging findings underscore the complex interplay between CSF dynamics and vascular integrity in the pathogenesis of SDH [27, 28].

Spinal imaging revealed differences in distribution of CSF leaks between the 2 groups. Patients with SDH had a lower proportion of no identifiable leaks and a higher proportion of single-level leaks. This finding suggests that the location and extent of CSF leakage may influence the risk of SDH. Single-level leaks may cause more localized pressure gradients, leading to focal areas of brain sagging and increased tension on bridging veins. Conversely, multiple-level leaks may distribute the pressure changes more evenly, reducing the risk of SDH [29-31]. Identifying the precise site of CSF leakage is crucial for targeted treatment approaches, such as epidural blood patching. However, an alternative explanation for this observation is detection bias. Patients in the SDH group often had a more complicated clinical course, which may have prompted more frequent or repeated spinal imaging compared to the non-SDH group. Such enhanced imaging surveillance would naturally increase the likelihood of identifying a CSF leak site, and account for the lower proportion of “no identifiable leak” and the higher proportion of single-level leaks observed in the SDH group. Thus, although leak characteristics may contribute to SDH risk, observed differences should be interpreted with caution.

Treatment characteristics highlighted the impact of repeated EBP procedures on SDH risk. Patients in the SDH group underwent more EBP procedures before SDH diagnosis, suggesting that repeated interventions may contribute to the development of SDH. While EBPs are effective in sealing CSF leaks, they can also introduce trauma to the dura and surrounding tissues. Repeated punctures may cause microtears or exacerbate existing vascular weaknesses, increasing the likelihood of hemorrhage [32-34]. Therefore, optimizing the timing and frequency of EBP procedures is essential to balance benefits of CSF leak repair with risks of complications.

In terms of risk factors, our multivariate analysis identified male sex, age, disease duration, dural enhancement, and number of pre-SDH EBP procedures as independent predictors of SDH. These findings provide a framework for identifying high-risk patients who may benefit from closer monitoring and more aggressive management strategies. For instance, older male patients with long-standing SIH and evidence of dural enhancement on imaging should be considered at higher risk for SDH. Similarly, patients requiring multiple EBP procedures should be carefully evaluated for signs of impending SDH. Multivariate analysis adjusted for age and sex, among other variables, to account for baseline imbalances. The fact that both age and male sex remained independent risk factors after adjustment suggests that these factors have a direct association with SDH risk beyond simple confounding.

Notably, brain sagging features and subdural effusion/hygroma were significantly associated with SDH in univariate analysis but lost significance in the multivariate model. This attenua-

tion may be explained by their strong collinearity with dural enhancement and disease duration, both of which are more direct indicators of chronic CSF leakage and intracranial hypotension severity. Alternatively, these imaging findings may represent intermediate phenotypes on the causal pathway rather than independent predictors of SDH, and their effect may be mediated through the variables that remained in final model.

Despite these important insights, this study has several limitations. Its retrospective design introduces potential biases and confounding factors that may affect the interpretation of results. Prospective studies are needed to validate these findings and establish causal relationships. Second, the generalizability of our results may be limited by specific patient population and clinical practices at our institution. Larger multicenter studies would enhance the external validity of our findings. Finally, future research should focus on elucidating molecular and cellular mechanisms underlying the development of SDH in SIH. Understanding these mechanisms will inform development of novel therapeutic strategies and improve patient outcomes.

Conclusion

This study suggests that several clinical characteristics, including male sex, longer disease duration, and a higher number of epidural blood patch procedures, may be associated with an increased risk of developing SDH in patients with SIH. Presence of dural enhancement on brain imaging also appears to be a risk factor. These findings indicate that prolonged exposure to low cerebrospinal fluid pressure, along with repeated interventions, could contribute to development of SDH. This highlights the importance of careful monitoring and management strategies for high-risk patients to potentially reduce incidence of this serious complication.

Disclosure of conflict of interest

None.

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