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Review Article

Bilateral subdural hematomas in spontaneous intracranial hypotension: A systematic review with case illustration

Negin Safari Dehnavi[1], Fatemeh Riyahi Zaniyani[2], Sadegh Bagherzadeh[2], Alireza Khoshnevisan[2], Ushtar Amin[3], Sheyar W. Amin[4], Ashley Rogers[4], Mohsen Rostami[4], Puya Alikhani[4], Milad Shafizadeh[2]

[1]Sina Trauma and Surgery Research Center, [2]Department of Neurosurgery, Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran, Departments of [3]Neurology and [4]Neurosurgery, Brain and Spine, Morsani College of Medicine, University of South Florida, Tampa, Florida.

E-mail: Negin Safari Dehnavi - negin.safari8227@gmail.com, Fatemeh Riyahi Zaniyani - fm.riyahi1997@gmail.com, Sadegh Bagherzadeh - dr.s.bagherzadeh@gmail.com, Alireza Khoshnevisan - khoshnevisanalireza@gmail.com, Ushtar Amin - uamin@usf.edu, Sheyar W. Amin - sheyar.amin@gmail.com, Ashley Rogers - arogers1@usf.edu, Mohsen Rostami - mohsenrostami@usf.edu, Puya Alikhani - palikhan@usf.edu, *Milad Shafizadeh - milad_shafizadeh@yahoo.com



***Corresponding author:**
Milad Shafizadeh,
Department of Neurosurgery, Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran.

milad_shafizadeh@yahoo.com

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ABSTRACT

Background: Spontaneous intracranial hypotension (SIH) is a well-recognized but often underdiagnosed cause of secondary subdural hematoma (SDH), particularly in younger patients who frequently present with bilateral collections. The role of lumbar puncture (LP) remains controversial in this setting, as additional cerebrospinal fluid removal may destabilize intracranial pressure and precipitate or worsen SDH.

Methods: We performed a systematic review of SIH cases associated with bilateral SDH. When possible, we reconstructed a patient-level dataset to analyze clinical presentation, LP context, management strategies, and outcomes. We also describe a middle-aged man with orthostatic headache and fever who presented with thin, chronic-appearing bilateral SDH in the setting of SIH. Following a diagnostic LP performed to exclude meningitis, he developed a rapidly progressive, extensive bilateral mixed-density SDH requiring urgent burr-hole evacuation.

Results: Fifty-nine studies were included, totaling 485 patients with SIH, of whom 182 had bilateral SDH. From these, we assembled a patient-level cohort of 59 individuals with SIH-associated SDH. The mean age was 47 years, and 81.4% were male. Orthostatic headache was present in 76.3%, whereas coma or stupor occurred in 25.4%. In this cohort, 62.7% had no LP, 30.5% underwent LP only after SDH diagnosis, and 6.8% had clearly post-LP SDH. Management included SDH surgery in 67.8%, epidural blood patch in 67.8%, and open leak repair in 11.9%. The overall SDH recurrence rate was 27.1%, with the highest recurrence (55.6%) in patients who underwent LP after SDH recognition. No deaths were reported.

Conclusion: Bilateral SDH in the context of SIH mainly affects younger patients and requires a combined strategy of SDH evacuation (when indicated) and leak-directed therapy. Although true post-LP bilateral SDH appears uncommon, LP may reveal or amplify an already unstable low-pressure state. Given the low certainty and incomplete temporal data in many reports, our findings support cautious consideration and avoidance of routine LP solely to document opening pressure in suspected SIH.

Keywords: Burr hole drainage, Chronic subdural hematoma, Epidural blood patch, Spontaneous intracranial hypotension

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INTRODUCTION

Spontaneous intracranial hypotension (SIH) is an underrecognized disorder most commonly caused by spinal cerebrospinal fluid (CSF) leakage, resulting in reduced intracranial CSF volume and pressure.[46,58] Orthostatic headache is the hallmark symptom, but patients may exhibit a wide range of clinical and radiological features, including pachymeningeal enhancement, venous sinus distension, brain sagging, and pituitary enlargement on magnetic resonance imaging (MRI).[20,58] With broader use of MRI and advances in dynamic myelographic techniques, SIH is now identified with increasing frequency.[58-60]

One of the most significant and potentially life-threatening complications of SIH is the development of subdural hematoma (SDH). Subdural fluid collections and hematomas have been reported in up to 50% of patients with SIH.[5,18,39] often progressing from thin, chronic-appearing layers to larger, mixed-density hematomas capable of mass effect and neurological deterioration if not recognized and treated promptly.[27,39,72] The predominant mechanism involves stretching and eventual rupture of cortical bridging veins due to loss of CSF buoyancy and downward displacement of the brain, compounded by venous engorgement and altered intracranial compliance in a low-pressure state.[41,46] For neurosurgeons, SIH-associated SDH, particularly when bilateral, represents a pathophysiologically distinct condition compared with degenerative chronic SDH typically encountered in elderly patients.

Diagnostic and management challenges are especially pronounced around lumbar puncture (LP). LP may be necessary to rule out central nervous system infection or inflammatory conditions that mimic SIH; however, it neither confirms nor excludes SIH and may worsen CSF hypovolemia, thereby precipitating or accelerating SDH formation and expansion.[1,6,80] As a result, the indications, timing, and safety of LP in patients with suspected SIH and SDH remain controversial, with considerable variation in clinical practice.

In this study, we provide a systematic review and pooled patient-level analysis of reported SIH cases with bilateral SDH, with particular emphasis on LP timing and context, patterns of leak- and hematoma-directed management, and associated outcomes. We then present a patient with orthostatic headache, fever, and thin, bilateral, chronic-appearing SDH who subsequently developed extensive bilateral mixed-density SDH after a limited-volume diagnostic LP.

MATERIALS AND METHODS

Study design and reporting

This study comprises a systematic review of the literature, with pooled patient-level analysis, focusing on SIH presenting with SDH. Reporting follows the preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines.[51] The flow of records through identification, screening, eligibility, and inclusion is summarized in a PRISMA diagram, and the full PRISMA checklist is provided in Supplement 1. The review protocol was prospectively registered in the International Prospective Register of Systematic Reviews (PROSPERO) (PROSPERO; registration number CRD420251232526).

Search strategy

A comprehensive search of PubMed, Embase, Scopus, and Web of Science was conducted from database inception to September 14, 2025. The strategy combined terms related to intracranial hypotension or CSF hypovolemia and SDH or hemorrhage, with emphasis on bilateral collections. No limitations on publication date or study design were initially applied. Reference lists of included studies were manually screened to identify additional eligible reports. The full search strategies for all databases are presented in Supplement 1.

Study selection and eligibility criteria

All records were imported into a reference manager, and duplicates were removed. Two reviewers independently screened titles and abstracts, as well as full texts; disagreements were resolved through discussion, with arbitration by a third reviewer when necessary. At full-text screening, we included articles that: (1) described SIH-related bilateral SDH; (2) provided sufficient clinical and imaging detail to confirm the diagnosis; and (3) allowed extraction of key variables such as age, sex, clinical presentation, LP status, management, and outcome. We excluded studies with traumatic or postoperative SDH, unilateral SDH only, and those without primary patient data. No language restrictions were applied at the search stage; non-English full texts were excluded only when reliable data extraction was not possible. The final set of articles formed the basis for qualitative and quantitative synthesis.

Data extraction and patient-level reconstruction

Two reviewers independently extracted data using a standardized form. For each study, we recorded study characteristics (first author, year of publication, country, and design), number of SIH patients, number with bilateral SDH, and number treated surgically for SDH where available. When reports contained sufficient individual detail, patient-level data were reconstructed and entered into a dedicated dataset, including demographics, presenting symptoms, LP status and timing, characteristics of SDH, management strategies directed at the hematoma and at the underlying CSF leak, adjunctive therapies, and outcomes (recurrence,

death). Ambiguities were handled conservatively. Because operational definitions of "recurrence" differed across reports (e.g., radiologic reaccumulation vs. symptomatic deterioration or need for reintervention), we recorded recurrence as defined by the original authors and interpreted recurrence comparisons cautiously.

Because LP timing and indication varied across reports, we categorized patients into three mutually exclusive groups according to LP context: (1) No LP; (2) LP after SDH (LP performed only after SDH recognition, usually for myelography or further work-up); and (3) Post-LP SDH (bilateral SDH clearly developing after a diagnostic LP). This classification was based strictly on the temporal sequence described by the original authors. These LP-context categories were author-defined and non-standardized; because chronology was inconsistently reported across case reports, misclassification of LP timing is possible and should be considered when interpreting comparisons across LP groups.

Risk of bias assessment

The risk of bias and methodological quality were assessed using the Joanna Briggs Institute (JBI) critical appraisal checklists for case series, cohort studies, case–control studies, and case reports, applied according to the study design.[47,55] The study-level scores and predominant bias domains are summarized in Supplement 1.

Data synthesis and statistical analysis

Data were synthesized descriptively. Continuous variables (e.g., age) were summarized using means and standard deviations, and categorical variables (e.g., sex, LP context, treatment modality, recurrence, mortality) were summarized as counts and percentages. Comparative analyses across LP-context categories were exploratory and intended to generate hypotheses about the interaction between SIH, LP and bilateral SDH rather than to establish causal relationships. Meta-analysis, subgroup analysis, publication bias assessment, and sensitivity analysis were not conducted due to the unavailability of pooled estimates from the included studies.

RESULTS

Study selection and study-level characteristics

Study selection is summarized in the PRISMA flow diagram [Figure 1]. The database search identified 580 records (Embase 169, Scopus 154, PubMed 140, and Web of Science 117). After removal of 298 duplicates, 282 titles and abstracts were screened, of which 204 were excluded. A total of 59 studies were included in the qualitative synthesis. Across these reports, there were 485 patients with SIH, of whom 182 had bilateral SDH, and 118 SDH surgeries were reported.

Among the 59 studies, 12 represented larger cohorts, defined as including more than one SIH patient and reporting at least one case of bilateral SDH. These 12 series are detailed in Table 1. Together, they contributed 438 SIH patients, of whom 135 had bilateral SDH; 88 SDH surgeries were reported. Larger series were case series (including one described as a case–control study), whereas three were retrospective cohorts and one was a prospective cohort. The largest cohort, reported by Overstijns *et al.*,[50] included 272 consecutive SIH patients, of whom 75 had bilateral SDH and 23 underwent SDH surgery. Overall, the larger studies originated from Asia, Europe, and North America, reflecting broad international experience with SIH-related SDH.

Risk of bias assessment

Seven case series,[9,18,23,26,39,69,76] four cohort studies,[5,35,50,72] one case-control study,[34] and forty-seven case reports[1,3,4,6,7,11,12,13-17,19,22,25,28,29,31-33,36,38,40,42,44,45,48,49,54,55,56,57,61-63,64-66,68,70,71,73,74,77,78,79,80] were appraised using the corresponding JBI critical appraisal tools. Overall, the case series were of moderate to high methodological quality, with most of the studies clearly describing inclusion criteria and participant characteristics. However, some provided limited information on the completeness of follow-up and site-level characteristics. The cohort studies also demonstrated generally moderate to high quality (JBI scores 5–9/11), with the main concerns related to incomplete adjustment for potential confounders and incomplete or insufficiently described follow-up in a subset of studies. The single case–control study showed a moderate risk of bias (7/10), with some uncertainty regarding matching procedures and control selection. The case reports were, in general, adequately reported (JBI scores 6–8/8), but several lacked a fully detailed clinical timeline and incompletely described the post-intervention condition or adverse or unanticipated events. Detailed item-level ratings and total JBI scores for each study are presented in Supplementary Figures 1 (case series), 2 (cohort studies), 3 (case–control study), and 4 (case reports).

Patient-level cohort and clinical presentation

A patient-level dataset of 59 individuals with SIH-associated SDH, including the present case, was reconstructed to allow more granular analysis of LP status, management strategies, and outcomes. In this dataset, the mean age at presentation was 47.3 years (SD 11.7), and 48 of 59 patients (81.4%) were male. We emphasize that this apparent male predominance likely reflects sampling and publication bias inherent to case-based literature and should not be interpreted as a true epidemiologic feature of SIH. Orthostatic headache was the dominant presenting symptom and was documented in 45

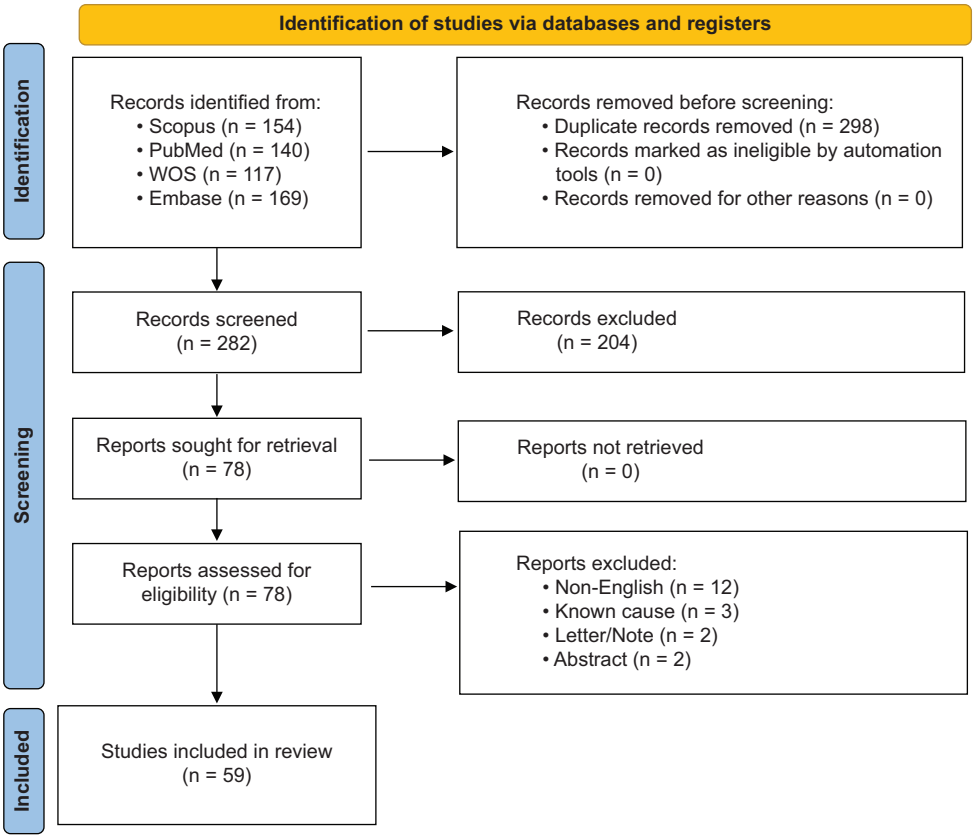


Figure 1: Preferred reporting items for systematic reviews and meta-analyses flowchart of the study selection process.

Table 1: Larger series reporting SIH with bilateral SDH.

First author, year	Country	Study design	SIH patients, *n*	Bilateral SDH, *n*	SDH surgeries, *n*
De Noronha *et al.*, 2003[18]	UK	Case series	9	4	4
Lai *et al.*, 2007[39]	Taiwan	Case series	40	6	2
Xia *et al.*, 2011[76]	China	Case series	2	2	1
Chaudhary *et al.*, 2011[9]	Canada	Case series	4	1	0
Ferrante *et al.*, 2010[26]	Italy	Case series	42	2	2
Beck *et al.*, 2014[5]	Switzerland	Prospective cohort	27	7	27
Wan *et al.*, 2016[72]	China	Retrospective cohort	15	11	15
Kim *et al.*, 2019[34]	South Korea	Case-control	8	8	1
Evran *et al.*, 2022[23]	Turkey	Case series	2	2	0
Terakado *et al.*, 2023[69]	Japan	Case series	2	2	2
Kim *et al.*, 2024[35]	South Korea	Retrospective cohort	15	15	11
Overstijns *et al.*, 2025[50]	Germany	Retrospective cohort	272	75	23

SIH: Spontaneous intracranial hypotension, SDH: Subdural hematoma, UK: United Kingdom

of 59 patients (76.3%). Neck pain occurred in 12 patients (20.3%), auditory symptoms (tinnitus or hearing disturbance) in 5 patients (8.5%), and coma or stupor in 15 patients (25.4%). Baseline characteristics and overall management patterns are summarized in Table 2, and individual patient-level data are presented in Table 3.

LP context

The temporal relationship between LP and SDH was examined using the three predefined LP-context categories. With this classification, 37 of 59 patients (62.7%) were categorized as No LP, 18 patients (30.5%) as LP after SDH,

Table 2: Baseline characteristics and overall management in the pooled SIH-SDH cohort.

Variable	Value
Patients, *n*	59
Age, mean (SD), years	47.3 (11.7)
Male, *n* (%)	48 (81.4)
Female, *n* (%)	11 (18.6)
Orthostatic headache, *n* (%)	45 (76.3)
Neck pain, *n* (%)	12 (20.3)
Auditory symptoms, *n* (%)	5 (8.5)
Coma/stupor, *n* (%)	15 (25.4)
Any SDH surgery, *n* (%)	40 (67.8)
Any EBP, *n* (%)	40 (67.8)
Open leak surgery, *n* (%)	7 (11.9)
Factor XIII replacement, *n* (%)	1 (1.7)
Intrathecal saline infusion, *n* (%)	5 (8.5)
Recurrence of SDH, *n* (%)	16 (27.1)

SIH: Spontaneous intracranial hypotension, SDH: Subdural hematoma, EBP: Epidural blood patch, SD: Standard deviation

and 4 patients (6.8%) as post-LP SDH. Thus, unequivocal post-LP SDH accounted for only a small minority of cases in this bilateral SIH-SDH dataset. In the post-LP SDH group, the LP was performed as a diagnostic procedure, usually demonstrating very low opening pressure, and the subsequent development of bilateral SDH was temporally related to the puncture. In contrast, patients in the LP after SDH group generally underwent LP and myelography to confirm SIH and localize the CSF leak after SDH recognition. In the No LP group, LP was either explicitly avoided due to concern about herniation in the presence of mass-effect SDH or not described, with the diagnosis of SIH relying on brain MRI and spinal imaging. Because these categories are author-defined and timelines were sometimes incompletely reported, classification may be imperfect and should be interpreted as approximate.

Management strategies

Management was heterogeneous and frequently combined SDH-directed and leak-directed strategies. Across the 59 patients, SDH surgery (burr-hole drainage or craniotomy) was performed in 40 (67.8%), and an epidural blood patch (EBP) was administered in 40 (67.8%). Open surgical repair of the spinal CSF leak was carried out in 7 patients (11.9%). Factor XIII replacement and intrathecal saline infusion were used as adjunctive treatments in 1 (1.7%) and 5 (8.5%) patients, respectively [Table 2].

When management was stratified by LP context [Table 4], 22 of 37 patients (59.5%) in the No LP group underwent SDH surgery, and 25 (67.6%) received an EBP, while 2 (5.4%) underwent open leak repair. In the LP after SDH group (n = 18), 15 patients (83.3%) had SDH surgery, 14 (77.8%) received an EBP, and 4 (22.2%) underwent open leak repair. In the post-LP SDH group (n = 4), 3 patients (75.0%) underwent SDH surgery, 1 (25.0%) received an EBP, and 1 (25.0%) underwent open leak repair.

Recurrence and mortality

Recurrence of SDH was documented in 16–59 patients (27.1%). In the No LP group, 5–37 patients (13.5%) had recurrent SDH. In the LP after SDH group, 10–18 patients (55.6%) experienced recurrence, indicating that this subset likely represents more complex or refractory SIH with persistent CSF leakage. In the post-LP SDH group, 1–4 patients (25.0%) had recurrence. No deaths were explicitly reported in the patient-level narratives. These findings suggest that true post-LP bilateral SDH in patients with SIH is uncommon; most bilateral SIH-related SDHs occur without a preceding diagnostic LP, and patients who undergo LP only after SDH diagnosis tend to have higher recurrence rates and more intensive leak-directed investigations, reflecting more severe or persistent CSF leaks rather than LP as the primary cause. Recurrence was variably defined across reports, limiting direct comparability between LP groups.

Case presentation

A 44-year-old man presented with an occipital, orthostatic headache. On arrival, he was febrile and mildly tachycardic. His Glasgow coma scale (GCS) score was 15, the neurological examination was normal, and no papilledema was noted. He had no relevant medical history and reported occasional tobacco and alcohol use.

Laboratory testing showed an elevated C-reactive protein level of 22 mg/L; all other values were within normal limits. Because no source for the fever was identified and meningitis remained a concern, a limited-volume diagnostic LP was performed. CSF appeared light yellow and clear with +1 xanthochromia; red blood cell were 243/μL, white blood cell was 3/μL with 0% neutrophils and >90% lymphocytes; protein was 93 mg/dL, lactate dehydrogenase was 104 U/L, and glucose was 56 mg/dL (CSF-to-serum ratio = 0.39). Gram stain revealed no organisms, and cultures remained negative. These findings did not support bacterial meningitis, and attention shifted to evaluation for SIH.

Baseline non-contrast head computed tomography (CT) demonstrated thin, bilateral chronic-appearing subdural collections (approximately 6 mm) without midline shift [Figure 2a]. MRI confirmed these findings and showed additional features consistent with SIH, including brain

sagging with bilateral subdural fluid layers [Figure 2b]. CT cisternography localized a cervicothoracic (C7-T2) spinal CSF leak. An EBP was performed with initial symptomatic improvement, and the patient was discharged.

Two weeks later, he returned with reduced consciousness (GCS 12). Head CT revealed large bilateral mixed-density subacute-chronic SDH measuring 26 mm on the right and 23 mm on the left, causing sulcal effacement and mass effect but no midline shift [Figure 2c]. Bilateral burr-hole evacuations were performed approximately 1.5 cm superior to the temporal line. Postoperative CT confirmed adequate evacuation of the hematomas with expected pneumocephalus and thin residual subdural layers [Figure 2d].

The patient's neurological status improved rapidly, and he was discharged home. At 1-month follow-up, his orthostatic headache had resolved, and neurological examination was normal. At 3-, 6-, and 12-month follow-up visits, he remained asymptomatic and fully active. Interval imaging demonstrated progressive resorption of the subdural collections without recurrence, and no further intervention was required.

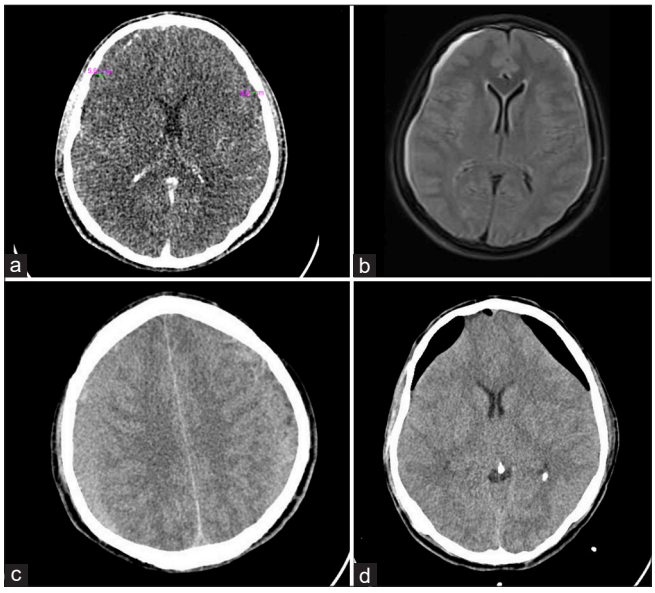


Figure 2: Evolution of bilateral subdural hematoma (SDH) in spontaneous intracranial hypotension (SIH). (a) Admission noncontrast head computed tomography (CT) shows thin bilateral chronic-appearing subdural collections (5–6 mm) without midline shift. (b) Admission axial brain magnetic resonance imaging demonstrates features consistent with SIH, including brain sagging and thin bilateral subdural fluid layers. (c) Preoperative head CT obtained on re-presentation reveals marked interval enlargement to mixed-density bilateral SDH with sulcal effacement and mass effect. (d) Early postoperative CT after bilateral burr-hole evacuation with drains confirms decompression, expected postoperative pneumocephalus, and thin residual subdural layers.

DISCUSSION

This report highlights the intersection of diagnostic uncertainty and pathophysiological vulnerability in SIH complicated by bilateral SDH. Our index case demonstrates how a clinically justified, limited-volume diagnostic LP performed to exclude meningitis can be followed by rapid progression from thin bilateral chronic-appearing collections to large, mixed-density bilateral SDH necessitating bilateral burr-hole evacuation. The systematic review and pooled patient-level analysis provide context for this scenario and clarify the relative contribution of LP versus the underlying CSF leak.

Alternative diagnostic pathways can often reduce reliance on diagnostic LP in suspected SIH. Brain MRI with contrast (e.g., pachymeningeal enhancement, venous sinus distension, and brain sagging) and dedicated spinal imaging may establish a high pre-test probability of SIH without CSF sampling.[8,10] The "venous distension sign" has also been described as a helpful MR feature supporting intracranial hypotension.[24] When leak localization is required, contemporary myelographic techniques, such as CT myelography (including dynamic or decubitus protocols) and digital subtraction myelography, can identify leaks and CSF-venous fistulas.[21,37,43] MR myelography or radionuclide cisternography may be useful when initial studies are nondiagnostic or when leak localization remains unclear.[2,30] We have added this context to emphasize that LP is rarely necessary as a "pressure check" and should be reserved for evaluation of competing diagnoses.

Pathophysiology of SDH in SIH and the effect of LP

This report highlights how, in SIH with bilateral SDH, a low-pressure intracranial state makes patients vulnerable to deterioration when LP is performed. In our case, a low-volume LP for suspected meningitis was followed by rapid progression from thin bilateral collections to large, mixed-density SDH requiring urgent bilateral burr-hole evacuation. Within the reconstructed patient-level cohort, this appears to be an extreme expression of an already unstable low-pressure state, with LP acting mainly as a potential precipitant rather than the primary cause of bilateral SDH. In this fragile configuration, even modest CSF removal can further reduce intracranial volume, increase caudal brain displacement, and promote venous tearing, consistent with earlier reports of post-LP SDH in unrecognized SIH.[68,75]

In our patient, the evolution from thin collections to sizeable mixed-density hematomas after LP mirrors these observations. However, unequivocal post-LP SDH accounted for only 6.8% of bilateral SIH-SDH cases, whereas most patients either did not undergo LP or had LP only after SDH recognition, indicating that the underlying CSF leak

Table 3: Patient-level characteristics, LP context, management, and recurrence in reported SIH-SDH cases.

Author, year	Country	Age (years)	Sex	LP context	SDH surgery	EBP	Leak surgery	Recurrence
Nakajima *et al.* 1996[49]	Japan	52	M	No LP	Yes	Yes	No	No
Sato *et al.* 1997[57]	Japan	42	F	No LP	Yes	Yes	No	Yes
Murakami *et al.* 2000[48]	Japan	43	M	No LP	Yes	Yes	No	No
Inenaga *et al.* 2001[31]	Japan	55	M	Post-LP SDH	Yes	No	Yes	Yes
Ishihara *et al.* 2001[32]	Japan	58	F	LP after SDH	No	No	No	No
Binder *et al.* 2002[6]	USA	43	M	No LP	Yes	Yes	No	No
Page *et al.* 2002[51]	Japan	57	F	No LP	No	Yes	No	No
de Noronha *et al.* 2003[18]	UK	66	M	No LP	Yes	No	No	No
		49	M	No LP	No	No	No	Yes
		39	M	No LP	No	Yes	No	No
		39	F	No LP	No	No	No	No
Tsai *et al.* 2005[70]	Taiwan	55	F	LP after SDH	No	Yes	No	No
Platz *et al.* 2007[53]	Switzerland	42	M	LP after SDH	Yes	No	No	No
Zada *et al.* 2007[79]	USA	44	M	No LP	No	Yes	No	No
Lai *et al.* 2007[39]	Taiwan	58	M	No LP	Yes	Yes	No	No
Chen *et al.* 2008[11]	Taiwan	27	M	No LP	No	Yes	No	No
Buvanendran *et al.* 2008[7]	USA	56	M	LP after SDH	Yes	Yes	No	Yes
Ferrante *et al.* 2009[25]	Italy	62	M	No LP	Yes	Yes	No	No
Srimanee *et al.* 2009[64]	Thailand	43	M	No LP	Yes	No	No	No
Yamakawa *et al.* 2010[77]	Japan	35	F	No LP	Yes	No	No	No
Dhillon *et al.* 2010[19]	Canada	50	M	LP after SDH	Yes	Yes	No	No
Chen *et al.* 2011[12]	Taiwan	33	M	No LP	Yes	No	No	No
Kuramae *et al.* 2011[38]	Japan	28	M	No LP	Yes	Yes	Yes	No
Kim *et al.* 2011[33]	South Korea	38	F	No LP	No	Yes	No	No
Xia *et al.* 2011[76]	China	66	M	LP after SDH	No	Yes	No	No
		34	F	LP after SDH	Yes	Yes	No	No
Fontaine *et al.* 2012[28]	France	46	F	LP after SDH	Yes	Yes	No	No
Ahmad *et al.* 2013[1]	Singapore	33	M	No LP	No	Yes	No	No
Chotai *et al.* 2013[15]	India	41	F	No LP	Yes	Yes	Yes	No
Dhillon *et al.* 2013[19]	Canada	67	M	No LP	Yes	Yes	No	Yes
Aladakatti *et al.* 2014[3]	India	32	M	No LP	No	Yes	No	No
Davis *et al.* 2014[17]	USA	42	F	No LP	Yes	Yes	No	No
Tana *et al.* 2014[68]	Italy	53	F	Post-LP SDH	Yes	Yes	No	No
Merali *et al.* 2018[45]	Canada	36	F	No LP	Yes	Yes	No	No
Uzunköprü *et al.* 2018[71]	Turkey	38	F	No LP	No	Yes	No	No
Williams *et al.* 2019[74]	USA	68	M	No LP	No	Yes	No	No
Podkovik *et al.* 2020[54]	USA	46	F	No LP	Yes	Yes	No	No
Yamamoto *et al.* 2020[78]	Japan	36	F	LP after SDH	Yes	Yes	No	No
Matsuoka *et al.* 2020[44]	Japan	55	M	LP after SDH	Yes	Yes	No	No
Wei *et al.* 2021[73]	China	40	F	No LP	No	Yes	No	No
Liu *et al.* 2021[42]	China	34	F	LP after SDH	Yes	Yes	No	No
Subramaniam *et al.* 2021[65]	India	38	M	LP after SDH	Yes	Yes	Yes	Yes
Kitagawa *et al.* 2021[36]	Japan	52	F	LP after SDH	Yes	Yes	No	No
Barros *et al.* 2021[4]	USA	63	M	No LP	Yes	Yes	No	No

(Contd...)

Table 3: *(Continued).*

Author, year	Country	Age (years)	Sex	LP context	SDH surgery	EBP	Leak surgery	Recurrence
Choi *et al*. 2022[14]	South Korea	52	M	LP after SDH	Yes	Yes	No	No
Swain *et al*. 2022[66]	India	40	F	LP after SDH	Yes	Yes	No	No
Evran *et al*. 2022[23]	Turkey	40	M	No LP	No	Yes	No	No
		39	M	No LP	No	Yes	No	No
Terakado *et al*. 2023[69]	Japan	55	M	LP after SDH	Yes	Yes	No	No
		54	M	LP after SDH	Yes	Yes	No	No
Chiu *et al*. 2023[13]	Taiwan	39	F	LP after SDH	Yes	Yes	No	No
Shah *et al*. 2024[61]	India	35	M	No LP	Yes	Yes	No	No
Ganjifard *et al*. 2024[29]	Iran	38	F	No LP	No	Yes	No	No
Cirillo *et al*. 2024[16]	Italy	49	M	LP after SDH	Yes	Yes	No	No
Pineda *et al*. 2024[52]	Philippines	37	M	No LP	Yes	Yes	No	No
		43	M	No LP	Yes	Yes	No	No
Shahzad *et al*. 2025[62]	Pakistan	41	F	LP after SDH	Yes	Yes	No	No
Present case 2025	Iran	44	M	LP after SDH	Yes	Yes	No	No

LP: Lumbar puncture, SDH: Subdural hematoma, EBP: Epidural blood patch, SIH: Spontaneous intracranial hypotension

Table 4: LP context, management, and recurrence in patients with SIH-associated SDH.

LP category	Patients, *n* (%)	SDH surgery, *n* (%)	EBP, *n* (%)	Leak surgery, *n* (%)	Recurrence, *n* (%)
No LP	37 (62.7)	22 (59.5)	25 (67.6)	2 (5.4)	5 (13.5)
LP after SDH	18 (30.5)	15 (83.3)	14 (77.8)	4 (22.2)	10 (55.6)
Post-LP SDH	4 (6.8)	3 (75.0)	1 (25.0)	1 (25.0)	1 (25.0)

LP: Lumbar puncture, SIH: Spontaneous intracranial hypotension, SDH: Subdural hematoma, EBP: Epidural blood patch

and intracranial hypotension are the dominant drivers of hematoma formation, with LP acting as a trigger in only a minority of patients.

Management patterns and the "dual-track" principle

The management patterns observed in our review support a "dual-track" strategy for SIH-related bilateral SDH: (1) treating the hematoma to relieve mass effect, and (2) addressing the underlying CSF leak to prevent recurrence. In the pooled cohort, SDH surgery was performed in 67.8% of patients and EBP in 67.8%, while open leak repair and adjunctive therapies (Factor XIII replacement, intrathecal saline) were reserved for complex or refractory cases. These findings are consistent with series in which non-geriatric chronic SDH was ultimately attributed to spinal CSF leak.[26,28,77]

Our case illustrates this dual-track principle. Bilateral burr-hole evacuation was necessary to relieve mass effect, but sustained clinical and radiological improvement occurred only after targeted treatment of the cervicothoracic leak. In several published series, failure to recognize and treat the leak led to recurrent SDH despite technically adequate hematoma evacuation, emphasizing the need for systematic leak localization, particularly at cervicothoracic levels, where leaks may be overlooked without dedicated imaging.[5,67,72]

Leak sealing, recurrence, and the role of LP context

An important insight from the pooled data is that early symptomatic improvement after EBP does not necessarily equate to complete leak closure. Some studies report persistent epidural fluid collections or demonstrable leaks on imaging or at surgery despite marked clinical improvement, whereas others describe rapid radiographic resolution after targeted patching or surgical repair of focal leaks.[5,34,35,46,58]

In our cohort, recurrence of SDH occurred in 27.1% of patients overall. Notably, recurrence was most frequent (55.6%) in the LP after SDH group, which likely represents patients with more severe or persistent CSF leaks prompting repeated investigations and interventions. In contrast, recurrence rates were lower in the No LP and post-LP SDH groups. These findings argue against LP as the principal culprit for poor outcomes and instead point toward underlying leak complexity and incomplete leak control as the key determinants of recurrence.

Implications for neurosurgical practice

Several practical implications emerge for neurosurgeons:

1. Suspect SIH early in younger patients with bilateral or atypical SDH and no clear trauma, notably with orthostatic headache, pachymeningeal enhancement, or spinal epidural fluid treat SIH as the default diagnosis until proven otherwise.
2. Avoid routine LP as a "pressure check" In suspected SIH with SDH or mass effect. Avoid LP performed solely to document low opening pressure; it may worsen downward brain shift and enlarge the hematoma.
3. Use LP only when there is a real alternative diagnosis. Reserve low-volume LP for cases where CNS infection or another competing diagnosis is genuinely plausible, and couple it immediately with leak-focused imaging (e.g., CT myelography) and expedited leak-targeted treatment.
4. Run SDH and leak treatment in parallel. Drain symptomatic or sizeable SDH when indicated, but simultaneously localize and treat the spinal CSF leak. In stable patients with smaller SDH and clear SIH, consider early, aggressive leak-directed therapy with close imaging rather than immediate SDH surgery.
5. Follow these patients closely because EBP may not fully or immediately seal the leak, arrange early clinical review and follow-up imaging (MRI or CT) to detect new or recurrent SDH, particularly if symptoms recur or fluctuate.

Strengths and limitations

Strengths of this study include the combination of a well-documented illustrative case with a broad, PRISMA-compliant systematic review and reconstruction of a patient-level dataset, enabling more nuanced exploration of LP context, management strategies, and outcomes. Limitations include the predominance of case reports and small series, heterogeneity in reporting and imaging protocols. Furthermore, some variables (e.g., leak site, size, and chronicity; exact LP volume) were inconsistently reported, limiting more detailed causal inferences. Nonetheless, the converging patterns across multiple independent reports support the clinical recommendations outlined above. In addition, reporting and publication bias are likely, and confounding by disease severity and management selection cannot be excluded; therefore, our LP-context comparisons are exploratory and do not support causal inference about LP and SDH progression.

CONCLUSION

Bilateral SDH is an important manifestation of SIH and typically reflects an underlying spinal CSF leak rather than a primary chronic SDH. In our case and pooled analysis, definite post-LP bilateral SDH was uncommon; most patients either did not undergo LP or had LP only after SDH was recognized, suggesting that the low-pressure state and persistent leak are the main drivers of hematoma formation and recurrence. Because the available evidence is largely retrospective and case-based, we cannot infer causality between LP and SDH progression. Instead, we recommend avoiding routine LP performed solely to document opening pressure when SIH is suspected, especially in the presence of subdural collections or mass effect. When LP is clinically required to evaluate a competing diagnosis, it should be performed with minimal CSF removal, close monitoring, and prompt progression to leak-localizing imaging and definitive leak-directed therapy. Management should combine timely treatment of symptomatic SDH with active leak localization and definitive leak closure, followed by close clinical and radiological follow-up.

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