

Risk factors associated with secondary delayed contralateral expanding hematoma in patient with bilateral acute traumatic intracranial hematomas

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Abstract

Background: Secondary delayed contralateral expanding hematoma (SDCEH) may occur after unilateral surgery in patients with bilateral acute traumatic intracranial hematomas and can lead to neurological deterioration. Identifying patients at risk may help guide early postoperative imaging and timely intervention.

Methods: We performed a retrospective cohort study of adult patients with bilateral acute traumatic intracranial hematomas who underwent unilateral surgical evacuation at a Level I trauma center between January 2021 and February 2024. Postoperative CT was obtained within 24 hours based on predefined clinical criteria. SDCEH was defined as a postoperative increase in contralateral hematoma volume or thickness by >30% requiring contralateral surgical intervention. Patients were categorized into progression (SDCEH) and non-progression groups. Variables associated with SDCEH were assessed using univariate analysis and multivariable logistic regression.

Results: Sixty-one patients were included; 29 (47.5%) developed SDCEH. On univariate analysis, midline shift, contralateral skull fracture, and postoperative coagulopathy were associated with SDCEH. In multivariable analysis, contralateral skull fracture ($p=0.048$) and postoperative coagulopathy ($p=0.011$) remained independent predictors, whereas midline shift was not significant after adjustment.

Conclusions: SDCEH occurred in nearly half of patients with bilateral acute traumatic intracranial hematomas treated with unilateral surgery. Contralateral skull fracture and postoperative coagulopathy independently predicted SDCEH. Patients with these features may benefit from intensive postoperative surveillance and early serial CT imaging to facilitate prompt detection and management.

Keywords: traumatic brain injury, epidural hematoma, subdural hematoma, skull fracture, coagulopathy

Introduction

Traumatic brain injury (TBI) is one of the major causes of disability and mortality around the world, particularly in low- and middle-income countries. According to a systematic assessment of the epidemiology of TBI from road traffic accidents (RTA), 69 million people globally are estimated to suffer a TBI each year from any cause.¹ South-east Asia and the Western Pacific are the two regions with the highest overall disease burden. According to the WHO Global status report on road safety for 2018, Thailand was one of the top two countries with an RTA mortality rate of 32.7 deaths per 100,000 people.²

It is not uncommon for neurosurgeons to encounter traumatic brain injury (TBI) patients presenting with asymmetrical bilateral intracranial hematomas. In such cases, the neurosurgeon may choose to evacuate the hematoma on only one side, particularly when the contralateral hematoma is deemed clinically insignificant or borderline. However, progression of the contralateral lesion may occur either intraoperatively or postoperatively, potentially leading to significant neurological deterioration and adversely affecting the final neurological outcome. In the neuro-intensive care unit, patient assessment is performed through clinical monitoring alone or in combination with neuromonitoring techniques, particularly intracranial pressure monitoring, to detect rising intracranial pressure or neurological deterioration that may indicate contralateral hematoma expansion. However, these neuromonitoring methods often identify the complication at a relatively late stage, resulting in delayed intervention.³ In some institutions, routine early postoperative CT scans are performed on all patients.⁴ However, this approach consumes considerable resources and

incurs unnecessary costs. To optimize practice, it is crucial to identify the factors associated with an increased risk of contralateral hematoma expansion. Recognizing these factors enables the selection of patients who would most benefit from early postoperative imaging, thereby facilitating timely detection and intervention.

This study aims to elucidate the risk factors of contralateral hematoma expansion in patients presenting with bilateral acute traumatic intracranial hematomas following unilateral surgical intervention.

MATERIAL AND METHODS

Study Design

A retrospective cohort study was conducted at a Level I referral trauma center, which treats an average of 606 TBI cases annually, including 277 operative cases. This study was approved by the Institutional Research Ethic Committee (Study code: SUR-2562-06683).

Inclusion Criteria

Adult patients (age 18 years) with bilateral acute traumatic intracranial hematomas who underwent only unilateral surgical intervention and contralateral hematomas were considered insignificant if they produced no mass effect on the ipsilateral parenchyma on the initial CT.

Exclusion criteria

Patient who underwent bilateral intervention in the same setting or absence of postoperative CT within 24 hours.

From 804 surgical cases of moderate to severe TBI between January 2021 and February 2024, 61 patients were enrolled in the study. Patients were divided into two groups: those with

secondary delayed contralateral expanding hematoma (SDCEH) (progression group) and those without SDCEH (non-progression group).

Definition of secondary delayed contralateral expanding hematoma (SDCEH)

Postoperative CT scans were conducted within 24 hours for patients who met any of the following criteria: 1) lack of expected improvement in postoperative neurological status; 2) deterioration of neurological status postoperatively; 3) decompressive craniectomy performed due to intraoperative brain swelling; or 4) intractable intracranial pressure elevation persisting for over 30 minutes despite cerebrospinal fluid (CSF) release. SDCEH were defined as an increase in calculated hematoma volume $[(\text{length} \times \text{width} \times \text{height}) / 2]$ or hematoma thickness by more than 30% and required surgical intervention⁵. (Figure 1)

Data collection and Statistical Analysis

The following variable (potential risk factors) were recorded, including: Clinical variables (age, gender, admission Glasgow Coma Scale [GCS]) and radiological parameters (first-operated hematoma type, contralateral insignificant hematoma type, midline shift, contralateral skull fracture, intraoperative brain swelling, and postoperative coagulopathy, which defined as INR >1.3 or platelet count $<100,000/\mu\text{L}$).

Statistical analysis was conducted using SPSS and R. Univariate analysis was utilized to assess the baseline characteristics between the progression and non-progression groups. Continuous data were presented as Mean $\pm$ Standard Deviation (SD) and analyzed using independent t-tests. Categorical variables were expressed as frequencies and percentages, analyzed via Chi-square or Fisher's exact tests. Variables with p-values < 0.20

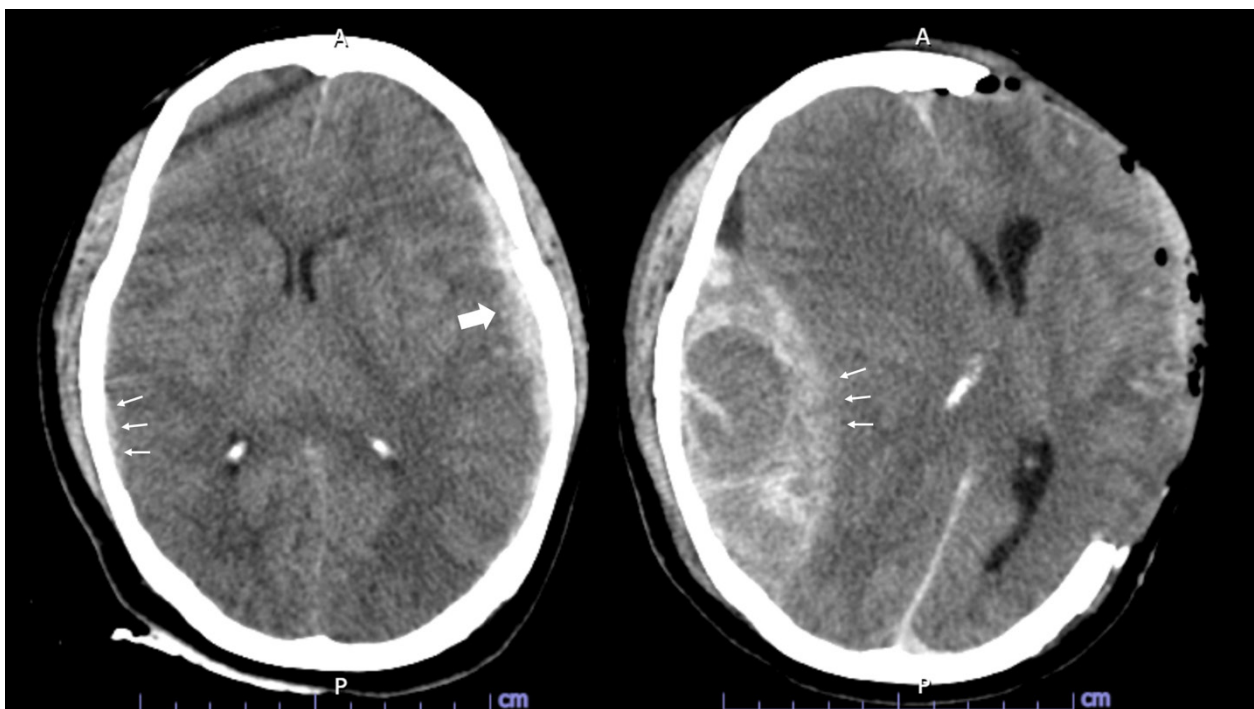


Figure 1. Representative case of bilateral acute traumatic intracranial hematomas. Left: Axial non-contrast head CT shows an acute left subdural hematoma (thick arrow), which was operated on first. Right: The initially small right epidural hematoma (thin arrows) subsequently evolved into a secondary delayed contralateral epidural hematoma (SDCEH) in the postoperative period following decompressive hemicraniectomy.

in the univariate analysis were entered into a multivariable logistic regression model to determine independent predictors, with statistical significance set at $p < 0.05$.

RESULTS

Out of 61 patients, 32 (52.5%) were categorized into the non-progression group, and 29 (47.5%) developed SDCEH (progression group). The baseline characteristics and statistical analyses are presented in **Table 1**. No significant differences were observed between the two groups with respect to age, gender, admission GCS, first operated hematoma, initial contralateral insignificant hematoma, midline shift, and intraoperative brain swelling.

However, contralateral skull fracture and post-operative coagulopathy were significantly more frequent in the progression group compared with the non-progression group (62.07% vs 46.88%; $p = 0.016$ and 51.77% vs. 21.88%; $p = 0.009$, respectively).

Univariate and Multivariate Analysis (Table 2)

In the univariate analysis, midline shift ($p = 0.012$), contralateral skull fracture ($p = 0.016$), and post-operative coagulopathy ($p = 0.009$) were significantly associated with SDCEH. Age, gender, GCS at admission, and intra-operative brain swelling did not demonstrate statistically significant relationships ($p > 0.05$).

Table 1 Comparison of clinical and radiological parameters between patients with and without SDCEH.

Parameters	Total case (n=61)	Contralateral expanding hematoma		p-value
		Non-progression (n=32)	Progression (n=29)	
Age (years)				0.797
Mean (SD)	43.69 (± 18.72)	43.09 (± 17.82)	44.34 (± 19.97)	
Gender, n (%)				0.735
Male	51 (83.61)	26 (81.25)	25 (86.21)	
Female	10 (16.39)	6 (18.75)	4 (13.79)	
GCS at admission, n (%)				0.266
≤ 8	44 (72.13)	21 (65.63)	23 (79.31)	
> 8	17 (27.87)	11 (34.38)	6 (20.69)	
First-operated hematoma, n (%)				0.867
EDH	29 (47.54)	15 (46.88)	14 (48.28)	
SDH	26 (42.62)	13 (40.63)	13 (44.83)	
Contusion	6 (9.84)	4 (12.50)	2 (6.90)	
Contralateral insignificant hematoma, n (%)				0.417
EDH	12 (19.67)	8 (25.00)	4 (13.79)	
SDH	25 (40.98)	13 (40.63)	12 (41.38)	
Contusion	20 (32.79)	8 (25.00)	12 (41.38)	
SAH	4 (6.56)	3 (9.38)	1 (3.45)	

Table 1 Comparison of clinical and radiological parameters between patients with and without SDCEH.

Parameters	Total case (n=61)	Contralateral expanding hematoma		p-value
		Non-progression (n=32)	Progression (n=29)	
Midline shift (mm)				0.817
Mean (SD)	6.31 (4.67)	6.18 (5.03)	6.46 (4.32)	
Contralateral skull fracture, n (%)				0.016*
No	28 (45.90)	17 (53.13)	11 (37.93)	
Yes	33 (54.10)	15 (46.88)	18 (62.07)	
Intra-op brain swelling, n (%)				0.116
No	40 (65.57)	24 (75.00)	16 (55.17)	
Yes	21 (34.43)	8 (25.00)	13 (44.83)	
Post-op coagulopathy, n (%)				0.009*
No	38 (62.30)	25 (78.13)	13 (44.83)	
Yes	23 (37.70)	7 (21.88)	16 (55.17)	

Table 2 Risk factors for SDCEH: Univariate and Multivariate Analysis

Variables	Univariate Analysis	Correlation Coefficient and p-value (p)	Multivariate Analysis Coefficient and p-value (p)
Age (years) Mean $\pm$ SD	43.09 $\pm$ 17.82 (Non-progression) vs 44.34 $\pm$ 19.97 (Progression) 95% CI of Mean Diff: -8.54 to 11.04	r = 0.034 p = 0.797	
Gender (Male vs Female)	95% CI for OR: 0.17 to 2.45	ϕ = -0.043 p = 0.735	
GCS at admission (≤ 8 vs > 8)	95% CI for OR: 0.64 to 7.15	ϕ = 0.152 p = 0.266	
First-operated hematoma	N/A	V = 0.078 p = 0.867	
Contralateral insignificant hematoma	N/A	V = 0.211 p = 0.417	

Table 2 Risk factors for SDCEH: Univariate and Multivariate Analysis (Cont.)

Variables	Univariate Analysis	Correlation Coefficient and p-value (p)	Multivariate Analysis Coefficient and p-value (p)
Midline shift (mm) Mean $\pm$ SD	4.82 $\pm$ 2.41 (Non-progression) vs 6.64 $\pm$ 2.87 (Progression) 95% CI of Mean Diff: 0.47 to 3.17	r = 0.312 p = 0.012*	β = 0.184 p = 0.165
Contralateral skull fracture (Yes vs No)	95% CI for OR: 1.15 to 7.84	ϕ = 0.345 p = 0.016*	β = 0.281 p = 0.048*
Intra-op brain swelling (Yes vs No)	95% CI for OR: 0.81 to 5.92	ϕ = 0.208 p = 0.116	
Post-op coagulopathy (Yes vs No)	95% CI for OR: 1.45 to 11.24	ϕ = 0.392 p = 0.009*	β = 0.354 p = 0.011*

Note: * indicates statistically significant at p <0.05

95% CI, 95% confidence interval; OR, odd ratio; r, Pearson correlation; ϕ = Phi Coefficient; V = Cramer's VCoefficient; β = Beta Coefficient; SD, standard deviation.

After adjusting for confounding factors in the multivariate model, contralateral skull fracture (β = 0.281, p = 0.048) and post-operative coagulopathy (β = 0.354, p = 0.011) remained independent, statistically significant predictors of SDCEH. Conversely, midline shift lost its statistical significance once other covariates were controlled (β = 0.184, p = 0.165).

DISCUSSION

Secondary delayed contralateral expanding hematoma (SDCEH) is a critical complication following unilateral surgical evacuation of bilateral acute traumatic intracranial hematomas and is strongly associated with poor outcomes.⁶ In this cohort, we observed an incidence of 47.5% (29/61), highlighting the clinical importance of identifying

patients at risk for progression. This incidence was substantially higher than previously reported rates of 7.7–22%, which may reflect a more severe injury mechanism in our cohort than in prior reports.^{6–8} Our multivariate analysis identified postoperative coagulopathy and contralateral skull fracture as independent, statistically significant risk factors, while the significance of midline shift was attenuated after adjusting for these variables.

Postoperative coagulopathy demonstrated the strongest statistical association with SDCEH (p = 0.011). Patients experiencing systemic failure of blood coagulation after craniotomy are highly susceptible to microvascular oozing. We hypothesize that when the mass effect of the index hematoma is relieved via unilateral surgery, the resultant change in intracranial hemodynamics, coupled

with systemic coagulopathy, facilitates rapid expansion of intact contralateral hemorrhagic lesions. This emphasizes that hematoma expansion is not merely a mechanical pressure-drop phenomenon but is heavily mediated by the patient's underlying hemostatic profile.

Contralateral skull fracture was also established as a statistically significant independent risk factor ($p = 0.048$). The presence of a fracture on the contralateral side indicates a high-energy kinetic impact that directly affected that specific region. This structural disruption likely weakens the vascular integrity of the meningeal vessels and underlying parenchyma. Consequently, these vessels become predisposed to re-bleeding or expansion during the acute postoperative phase, particularly as the brain undergoes global shifts following surgical decompression.

Interestingly, while midline shift demonstrated statistical significance in the univariate analysis ($p = 0.012$), its significance was not maintained in the multivariate model ($p = 0.165$). This suggests that global mass effect, while a marker of injury severity, may act as a secondary bystander rather than an independent initiator of contralateral expansion when compared to more specific mechanical (skull fracture) and systemic (coagulopathy) risk factors.

Our findings are supported by several authors who have proposed mechanisms for postoperative contralateral hematoma progression, particularly the loss of the tamponade effect of the index hematoma, which may permit continued bleeding from contralateral vascular injury. This hypothesis is consistent with our observation that skull fractures were more frequent among patients who developed contralateral expanding epidural hematoma (EDH).^{6,8} Another mechanism was purposed

by Kurland et al, he stated that overt or latent coagulopathy, together with subthreshold kinetic energy imparted to penumbral micro vessels, initiates maladaptive molecular cascades that culminate in microvascular failure, tissue ischemia, and delayed petechial hemorrhages, which then coalesce into hemorrhagic progression.^{9,10} Our observation that postoperative coagulopathy and contralateral skull fracture are key risk factors aligns with these established mechanisms.

This study has several limitations. Its single-center, retrospective design introduces selection and information bias and limits generalizability. The small sample and low event rate reduced power, yielding wide confidence intervals and limiting multivariable modeling, interaction testing, and subgroup analyses. Perioperative care was not fully standardized introducing treatment- and detection-related confounding especially the proper time to repeat scan in high risk group. Data were abstracted from routine records with potential missingness and measurement error. Prospective, multicenter studies with standardized perioperative and imaging protocols as well as adequate sample size are needed to validate and refine SDCEH risk factors.

CONCLUSION

Secondary delayed contralateral expanding hematoma is an intricate complication driven primarily by postoperative coagulopathy and contralateral skull fracture. Clinicians must shift focus from purely anatomical variables toward proactive monitoring of coagulation status and structural impact marks. Intensive neurological vigilance and early serial CT scans are strictly recommended for this high-risk subset of patients.

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Conflicts of interest

Kriangsak Champawong: None

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Author's disclosure

The author(s) have no competing interest to disclose

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