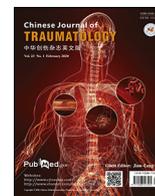




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Original Article

Risk factors associated with the progression of extra-axial hematoma in the original frontotemporoparietal site after contralateral decompressive surgery in traumatic brain injury patients

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ABSTRACT

Purpose: To introduce our experience with progressive extra-axial hematoma (EAH) in the original frontotemporoparietal (FTP) site after contralateral decompressive surgery (CDS) in traumatic brain injury patients and discuss the risk factors associated with this dangerous situation.

Methods: This retrospective study was conducted on 941 patients with moderate or severe TBI treated in Daping Hospital, Army Medical University, Chongqing, China in a period over 5 years (2013–2017). Only patients with bilateral lesion, the contralateral side being the dominant lesion, and decompressive surgery on the contralateral side conducted firstly were included. Patients were excluded if (1) they underwent bilateral decompression or neurosurgery at the original location firstly; (2) although surgery was performed first on the contralateral side, surgery was done again at the contralateral side due to re-bleeding or complications; (3) patients younger than 18 years or older than 80 years; and (4) patients with other significant organ injury or severe disorder or those with abnormal coagulation profiles. Clinical and radiographic variables reviewed were demographic data, trauma mechanisms, neurological condition assessed by Glasgow coma scale (GCS) score at admission, pupil size and reactivity, use of mannitol, time interval from trauma to surgery, Rotterdam CT classification, type and volume of EAH, presence of a skull fracture overlying the EAH, status of basal cistern, size of midline shift, associated brain lesions and types, etc. Patients were followed-up for at least 6 months and the outcome was graded by Glasgow outcome scale (GOS) score as favorable (scores of 4–5) and unfavorable (scores of 1–3). Student's *t*-test was adopted for quantitative variables while Pearson Chi-squared test or Fisher's exact test for categorical variables. Multivariate logistic regression analysis was also applied to estimate the significance of risk factors.

Results: Initially 186 patients (19.8%) with original impact locations at the FTP site and underwent surgery were selected. Among them, 66 met the inclusion and exclusion criteria. But only 50 patients were included because the data of the other 16 patients were incomplete. Progressive EAH developed at the original FTP site in 11 patients after the treatment of, with an incidence of 22%. Therefore the other 39 patients were classified as the control group. Multivariate logistic regression analysis showed that both the volume of the original hematoma and the absence of an apparent midline shift were significant predictors of hematoma progression after decompressive surgery. Patients with fracture at the original impact site had a higher incidence of progressive EAH after CDS, however this factor was not an important predictor in the multivariate model. We also found that patients with progressive EAH had a similar favorable outcome with control group.

Conclusion: Progressive EAH is correlated with several variables, such as hematoma volumes ≥ 10 mL at the original impact location and the absence of an apparent midline shift (< 5 mm). Although progressive

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EAH is devastating, timely diagnosis with computed tomography scans and immediate evacuation of the progressive hematoma can yield a favorable result.

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Introduction

The progression of extra-axial hematoma (EAH) after contralateral decompressive surgery (CDS) in patients with traumatic brain injury (TBI) has been reported for more than 20 years, and its prevalence ranges from 5% to 12%.^{1–6} However, we found that the progression of hematoma at the original frontotemporoparietal (FTP) site after CDS had a higher incidence.

Progressive EAH may present as intraoperative brain swelling, postoperative neurologic deterioration, or no signs, and can be diagnosed by postoperative routine examination. If unrecognized, progressive EAH can be serious and potentially life-threatening, and hence deserves more concern in clinical practice. Identifying the individuals at a high risk of progressive EAH can promote early detection of this lesion and prevent rapid neurological deterioration.

The aim of this study was to assess the incidence and risk factors of progressive EAH after CDS in head trauma patients and to discuss the clinical features of patients with such complication.

Methods

Inclusion and exclusion criteria

Records of 941 patients with moderate or severe TBI treated in Daping Hospital, Army Medical University, Chongqing, China from January 2013 to December 2017 were retrospectively reviewed. All the cases underwent the exam of preoperative computed tomography (CT) and analyzed by a neurologist. Their clinical and radiological data were collected. Initially 186 patients (19.8%) with original impact locations at the FTP site who underwent surgery were selected. But only those met the following criteria were included in this study: bilateral lesion, the contralateral side being the dominant lesion, and decompressive surgery on the contralateral side performed firstly. Exclusion criteria were: (1) patients underwent bilateral decompression or neurosurgery at the original location firstly; (2) although surgery was performed first on the contralateral side, patient underwent surgery again at the contralateral side due to re-bleeding or complications of the contralateral side; (3) patient aged <18 years or >80 years; and (4) patients with other significant organ injury or severe disorder or those with abnormal coagulation profiles.

The primary outcome variable was the existence of progressive original EAH at the FTP site after the treatment of CDS. Progression was defined as an increase of the initial EAH volume by 30%^{7,8} or the appearance of a new hematoma in a follow-up CT scan. Patients were classified into progression group and control group based on whether progressive EAH occurred at the original location after initial contralateral decompression.

Clinical and radiographic data

Clinical variables examined in this study included demographic data, trauma mechanisms, neurological condition assessed by Glasgow coma scale (GCS) score at admission, pupil size and reactivity, use of mannitol, and time interval from trauma to surgery (≥ 24 h or <24 h). Radiographic data such as the type and volume of EAH, Rotterdam CT Classification, presence of a skull

fracture overlying the EAH, status of basal cistern, size of midline shift and presence of associated brain lesions as well as the corresponding types were also reviewed.

CT scan

All the patients underwent head CT shortly after admission. Moreover CT examinations were conducted routinely during operation or if brain swelling or postoperative neurological deterioration presented. The purpose of intraoperative CT was to estimate the presence of progressive EAH and exclude extensive cerebral infarction or diffuse cerebral edema. The results of preoperative CT scans were scored based on the Rotterdam CT classification.

Hematoma volume

The original FTP site of the EAH was classified into 2 groups according to the hematoma volume: <10 mL and ≥ 10 mL. EAH volume was measured empirically using formula $0.5 \times \text{height} \times \text{depth} \times \text{length}$. Depth and length were measured on the CT slice having the largest clot area. The change in volume (mL) was estimated from the initial and follow-up CT scans.

Midline shift and brain lesion

The midline shift was graded as <5 mm and ≥ 5 mm. Associated brain lesions were defined as cases with subarachnoid hemorrhage, brain contusion, intraventricular hemorrhage or diffuse brain edema.

Follow-up and outcome

All patients were followed-up with a minimum of 8 months and a maximum of 3.5 years. Glasgow outcome scale (GOS) score (1 = death, 2 = persistent vegetative state, 3 = severe disability, 4 = moderate disability, and 5 = good recovery) was used to evaluate patients' outcome. The score 4–5 was regarded as favorable and 1–3 as unfavorable results. Therefore the GOS result was taken as a dichotomous variable.

Statistical analysis

Data were analyzed using SPSS version 19.0 (SPSS Inc, Chicago, USA). A univariate analysis for each variable was assessed using Students *t*-test (quantitative variables) and Pearson Chi-squared test or Fisher's exact test (categorical variables).

A logistic regression model was developed using the univariate analysis results to identify risk factors related to development of progressive EAH. A *p* value less than 0.05 was considered statistically significant and the predictors were calculated with 95% confidence interval (CI).

Results

Among the 186 selected patients with original impact locations at FTP sites and surgical treatment, 66 met the inclusion and

exclusion criteria. But 16 patients had incomplete data and were thus excluded. In the end, 50 patients were enrolled for further analysis. Eleven patients (22.0%) developed progressive EDH and were sorted in the progression group. The other 39 patients were then enrolled in the control group.

Clinical features and radiographic data for the 11 patients with progressive EAH are summarized in Table 1. Intraoperative brain swelling after contralateral hematoma evacuation was noted in 6 patients, all of whom underwent original site surgery to release mass effect immediately. Postoperative CT scans were performed in 2 patients with deteriorated neurologic condition after acute contralateral hematoma evacuation and in another 3 patients as part of routine examination after decompressive surgery. All the 5 patients underwent a second operation after the postoperative CT scan. The source of bleeding was from the middle meningeal artery of 6 patients, unknown or undocumented in the rest 5 patients. During operation, a skull fracture covering the hematoma was found in 81.8% (9/11) patients.

Comparison of clinical and radiological variables between the progression and control groups were further conducted. The former had a mean age of 50.6 years, and 81.8% (9/11) were males while those for the controls were 52.5 years and 71.8% (28/39) respectively, showing no significant difference. There were no significant difference regarding injury mechanism, GCS, pupil size, pupillary reactivity, use of mannitol, or surgery interval time between progression and control group (Table 2). Almost half of the patients (26/50, 52.0%) in this study had a favorable outcome and GOS after 6 months follow-up showed no significant difference between two groups.

Radiological findings revealed that the most common lesion of progressive EAH at the original site was epidural hematoma (EDH) (45.5%, 5/11), with an average original volume of 14 mL. Patients with progressive hematoma tended to have a greater Rotterdam CT classification score, but this difference did not reach statistical significance. The original EAH volume at the original FTP site was found to be < 10 mL in 29 (58%) patients and ≥ 10 mL in 21 (42%) patients. The incidence of progressive EAH in patients with a volume ≥ 10 mL was significantly higher than that in those with a volume < 10 mL ($p = 0.008$). Hematoma progression at the original location was observed in 7/16 (43.8%) patients with a midline shift < 5 mm, showing a great difference ($p = 0.003$). Moreover, the variable skull fracture at the original location reached statistical significance, as patients with skull fracture at the original location had a higher risk of hematoma progression ($p = 0.025$). While the variables of type of the original hematoma, associated brain lesion and basal cistern status showed no significant difference between two groups. Figs. 1 and 2 showed representative CT scan images of two patients with progressive EAH after CDS.

Multivariate logistic regression results showed that only the volume of the original hematoma (Wals 5.11, OR 3.43, 95% CI 1.18–10.0, $p=0.024$) and midline shift (Wals 5.48, OR 6.64, 95% CI 1.36–32.44, $p=0.019$) were independent predictors of progressive EAH. Thus, patients with a larger original hematoma volume (≥ 10 mL) and the absence of an apparent midline shifts (< 5 mm) had respectively 3.43 and 6.64 times higher risk of progressive EAH than those with the opposite CT features.

Discussion

Delayed progression of EAH after CDS is an uncommon phenomenon, which has been reported in the literature. However, it is a severe and potentially lethal complication if unrecognized. In our research, the incidence of post-decompressive effects of EAH at the original FTP site reached as high as 22%. Therefore it is necessary to give a high suspicion or awareness of this dangerous entity for an early diagnosis in clinical practice.

There are many explanations for the pathogenesis of delayed progression of EAH following CDS, including loss of tamponade effect, abnormal vasomotor mechanisms, and acute coagulopathy.^{9,10} Moreover, the contralateral EAH was not detected through radiographic examination, but developed into a natural process with no relation to the surgical evacuation.^{11,12} The main reason seems to be that the balance between the damaged blood vessels and reactive intracranial hypertension is disrupted.¹⁰ The sources of EDH bleeding consisted of rupture of the meningeal artery branch (6/11 in the present study) or skull fracture. In addition, it has been suggested that rapid displacement of the brain after decompressive craniectomy and hematoma clearance can cause shear stress in the contralateral bridging vein, leading to rupture of these bridging veins and formation of contralateral SDH.^{13,14} Though it was considered that EDH and SDH have very different pathophysiologies, we failed to found any significant difference of the type of the original hematoma in predicting hematoma progression.

The current study focused mainly on injuries at the original FTP site which was found to have a higher incidence of progressive EAH than other parts. There are abundant dural vessels at this site. Moreover, the mean age of the progression group was 50.6 years, an age range characterized by decreased elasticity and increased fragility of vessels.¹⁵ In addition, we found original skull fractures overlying or nearby the site of hematoma in 81.8% patients in progression group. It is speculated that the dura mater may be more likely to be torn or avulsed because it becomes more and more attached to the skull with age.^{14,16} As a result, elderly patients are more vulnerable to rupture of the bridging veins after evacuation of the hematoma. In addition, it has been suggested

Table 1
Characteristics of the 11 patients with progressive extra-axial hematoma after contralateral decompressive surgery.

| Case | Gender/Age (years) | GCS score at admission | Signs presented | Original location | Fracture type | Volume of original hematoma (mL) | GOS score |
|------|--------------------|------------------------|----------------------------|-------------------|---------------------|----------------------------------|-----------|
| 1 | F/79 | 6 | Brain swelling | EDH + SDH | Linear fracture | 15 | 4 |
| 2 | M/57 | 4 | Brain swelling | EDH | Linear fracture | 13 | 1 |
| 3 | M/29 | 3 | Neurological deterioration | EDH + SDH | Comminuted fracture | 15 | 1 |
| 4 | M/58 | 10 | Brain swelling | EDH | Linear fracture | 22 | 5 |
| 5 | F/26 | 15 | None | SDH | None | 12 | 5 |
| 6 | M/55 | 10 | Brain swelling | EDH | Comminuted fracture | 13 | 4 |
| 7 | M/48 | 5 | Brain swelling | SDH | Linear fracture | 18 | 1 |
| 8 | M/49 | 7 | Neurological deterioration | EDH + SDH | Linear fracture | 7 | 3 |
| 9 | M/42 | 9 | None | EDH + SDH | Comminuted fracture | 20 | 2 |
| 10 | M/56 | 6 | None | EDH | Linear fracture | 5 | 1 |
| 11 | M/58 | 15 | Brain swelling | EDH | None | 14 | 4 |

GCS: Glasgow coma scale; EDH: epidural hematoma; SDH: subdural hematoma; GOS: Glasgow outcome scale.

Note: The patients without special signs are detected by routine computed tomography scan.

Table 2
Comparison of clinical and radiological variables between patients with or without progressive extra-axial hematoma.

| Clinical and radiological variables | Total cases (n = 50) | Progression (n = 11) | Control (n = 39) | p value |
|---|----------------------|----------------------|------------------|---------|
| Age (years) | – | 50.6 ± 14.7 | 52.5 ± 18.9 | NS |
| Male/Female | 37/13 | 9/2 | 28/11 | NS |
| Mechanism of trauma | | | | NS |
| Fall | 31 (62.0) | 8 (72.7/27.3) | 23 (59.0) | |
| Traffic accident | 17 (34.0) | 3 (27.3) | 14 (35.9) | |
| Impact and other causes | 2 (4.0) | 0 (0) | 2 (5.1) | |
| GCS | – | 8.2 ± 4.1 | 8.3 ± 3.1 | NS |
| Pupil size (mm) | – | 3.2 ± 1.1 | 2.9 ± 0.8 | NS |
| Pupillary reactivity | | | | NS |
| Normal | 18 (36.0) | 4 (36.4) | 14 (39.9) | |
| Dull | 20 (40.0) | 4 (36.4) | 16 (41.0) | |
| Absent | 12 (24.0) | 3 (27.3) | 9 (23.1) | |
| Mannitol | | | | NS |
| Used | 27 (54.0) | 5 (45.5) | 22 (56.4) | |
| Not used | 23 (46.0) | 6 (54.5) | 17 (43.6) | |
| Surgery interval time (h) | | | | NS |
| <24 | 27 (54.0) | 5 (45.5) | 22 (56.4) | |
| ≥24 | 23 (46.0) | 6 (54.5) | 17 (43.6) | |
| Outcome | | | | NS |
| Favorable | 26 (52.0) | 5 (45.5) | 21 (53.8) | |
| Unfavorable | 24 (48.0) | 6 (54.5) | 18 (46.2) | |
| Rotterdam CT classification | – | 3.2 ± 1.5 | 3.0 ± 1.6 | NS |
| Volume of hematoma at original location (mL) | | | | 0.008 |
| <10 | 29 (58.0) | 2 (18.2) | 27 (69.2) | |
| ≥10 | 21 (42.0) | 9 (81.8) | 12 (30.8) | |
| Type of original hematoma | | | | NS |
| None | 11 (22.0) | 0 (0) | 11 (28.2) | |
| EDH | 21 (42.0) | 5 (45.5) | 16 (41.0) | |
| SDH | 10 (20.0) | 2 (18.2) | 8 (20.5) | |
| EDH + SDH | 8 (16.0) | 4 (36.4) | 4 (10.3) | |
| Skull fracture at the original location | | | | 0.025 |
| None | 24 (48.0) | 2 (18.2) | 22 (56.4) | |
| Fracture | 26 (52.0) | 9 (81.8) | 17 (43.6) | |
| Associated brain lesion | | | | NS |
| None | 8 (16.0) | 3 (27.3) | 5 (12.8) | |
| tSAH or brain contusion | 26 (52.0) | 4 (36.4) | 22 (56.4) | |
| tSAH and brain contusion/multifocal brain contusion | 11 (22.0) | 4 (36.4) | 7 (17.9) | |
| IVH or diffuse brain swelling | 5 (10.0) | 0 | 5 (12.8) | |
| Basal cistern | | | | NS |
| Normal | 16 (32.0) | 3 (27.3) | 13 (33.3) | |
| Compressed | 26 (52.0) | 5 (45.5) | 21 (53.8) | |
| Absent | 8 (16.0) | 3 (27.3) | 5 (12.8) | |
| MLS (mm) | | | | 0.011 |
| <5 | 16 (32.0) | 7 (63.6) | 9 (23.1) | |
| ≥5 | 34 (68.0) | 4 (36.4) | 30 (76.9) | |

GCS: Glasgow coma scale; CT: computed tomography; EDH: epidural hematoma; SDH: subdural hematoma; tSAH: traumatic subarachnoid hemorrhage; IVH: intraventricular hemorrhage; MLS: midline shift; NS: not significant.

Data are expressed as n (%) or mean ± standard deviation.

that intracranial blood vessels may develop re-bleeding and formation of hematomas after relieving tamponade effect on the contralateral side.

In analyzing risk factors for the progression of EAH, the frequent development of original contusions was observed, in agreement with previously published studies.⁴ Furthermore, Chang et al.¹⁷ and Oretel et al.¹⁸ reported that progressive EAH patients often have a high-volume original hematoma and pupillary abnormalities. The current study reported a novel result by quantifying the initial volume of the original hematoma and found that a specific initial volume was associated with a higher probability of growth. We observed that the original EAH with an initial volume of ≥10 mL was much more likely to develop a progressive EAH at the original FTP site ($p = 0.008$). Hypothetical explanation of this result is that a hematoma of approximately 10 mL has sufficient space inside the cranial cavity for expansion, especially when high intracranial pressure (ICP) is decreased.

Alike original hematoma volume, the degree of midline shift was another risk factor for progressive EAH in patients after CDS. Considering the relation between initial hematoma volume and

midline shift, it is possible that hematomas of more than 10 mL at the original impact site can generate a sufficient mass effect to resist contralateral compression. Consequently, consisted with previous research, the midline shift in the cases group was not obvious.^{15,19}

In most reports of progressive EAH after acute CDS, an overlying skull fracture was demonstrated at the site of original fracture.^{4,6,9} In the current study, we found that patients in the progression group had a higher incidence of fracture at the original impact site (81.8 vs 43.6, $p=0.025$). Although univariate analysis showed that the original fracture was associated with progressive EAH, multivariate logistic model revealed that it was not statistically significant. We do not know if a greater number of cases or a different classification method would affect this result. Further research is needed to verify this explanation.

Due to the high cost, ICP monitor was implanted only in a few patients. Therefore, ICP monitoring was not assessed in our research. But if possible, implanting a continuous ICP monitor during the first surgery will help identify progression of EAH in a timely manner.^{20,21}

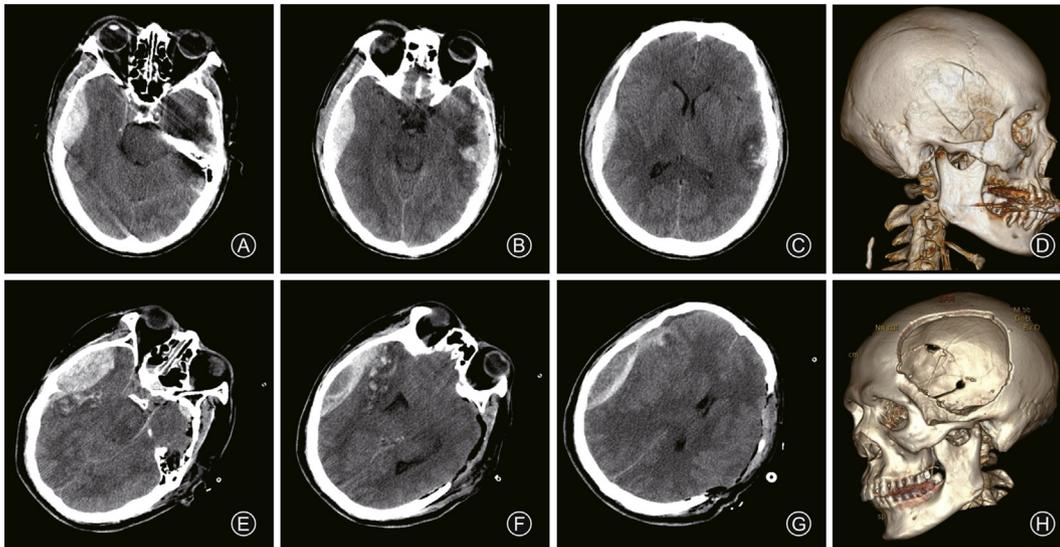


Fig. 1. A 55-year-old man with progressive epidural hematoma following traffic accident (Case 6). (A–C) Initial computed tomography (CT) demonstrated a small epidural hematoma in the right temporal lobe, subdural hematoma and contusion in the left temporal lobe. The midline shifted to the right slightly. The left pupillary reactivity was dull. (D) Comminuted fracture of the original right temporal bone. (E–G) Intraoperative brain swelling after contralateral decompressive surgery, prompting an immediate CT scan, which revealed a progressive epidural hematoma in the original right temporal site with basal cistern compression and a midline shift to the left. (H) Patient underwent emergent evacuation of the original hematoma, and the bone flap was restored.

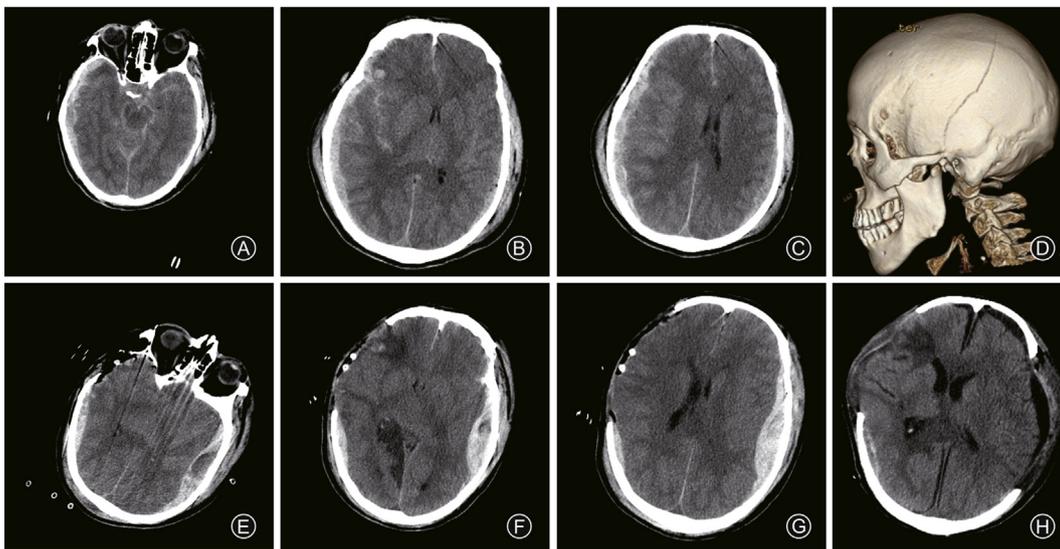


Fig. 2. A 56-year-old man with progressive epidural hematoma after fall (Case 10). (A–C) Initial computed tomography (CT) image demonstrated a subarachnoid hematoma in the left temporal lobe. (D) Original linear fracture extending from the parietal bone to the skull base. (E–G) Postoperative routine CT at 13 h after decompression revealed a progressive epidural hematoma at the original left temporo-parietal site with the absence of basal cistern and a midline shift to the right. (H) Patient underwent emergent evacuation of the original hematoma and decompressive craniectomy.

To detect progressive EAH, it should be noted that half of the patients (6/11) developed intraoperative brain swelling after CDS. We recommend a CT scan immediately after decompression, which helps to detect and treat the hematoma early. As for the method of exploratory burr holes,⁵ we prefer CT scan as the utility of blind exploration is doubtful. In the case of brain hernia, we can even do exploratory burr-hole on the original site straight after CDS, if intraoperative CT has not been done. We emphasize the importance of guarded durotomy as an effective measure to reduce the incidence of progressive EAH. In addition, multiple fenestration of the dura helps reduce brain shift.^{22,23}

Huang and colleagues³ believe that newly developed or expanded remote EDHs are most likely to occur within 24 h after

injury or even earlier due to the decompressive effects. Therefore, we set the time interval from injury as <24 h and ≥ 24 h. In our study, a total of 27 (54%) patients underwent emergency surgery within 24 h after TBI. However, early surgery (<24 h) failed to be a predictor of progression.

As original EAH progression is a dynamic process, 5 of our patients with progressive original EAH were recognized by postoperative routine examination or neurological worsening including pupil dilation, conscious deterioration, or high intracranial pressure within 1–4 days after surgery. Previous studies have reported that the mean interval between injury and hematoma enlargement is 36 h after the initial CT scan.²⁴ Furthermore, one of our patient with no a warning signs was found having a progressive EDH by routine

examination at 96 h after decompression. Therefore, we supported that timely follow-up postoperative CT was surveilled to detect this dangerous complication.

Consistent with previous reports,^{2,3} our results showed that progressive EAH following decompressive surgery did not influence outcomes of TBI patients. After six months of follow-up, 45.5% (5/11) patients with progressive EAH had a favorable outcome. A possible reason may be that it is the severity of TBI determines prognosis rather than the progressive EAH.⁶ Another reason is that surgical timing affects the prognosis of patients with progressive EAH leading to brain structural compression. As long as the mass effect does not progress to the stage of permanent brain dysfunction, nerve recovery will not be compromised. We believe that although EAH progression may be fatal after CDS, timely CT scans and rapid hematoma clearance can effectively restore neurological function.

Our research has some limitations. This is a retrospective study in a single-center and only 11 patients with progressive EAH are sampled, which limits a more detailed statistical analysis of risk factors for EAH progression after CDS. The unequal distribution of the two groups affects the power of our statistical analysis. A prospective multicenter study is necessary to analyze this lethal but preventable complication.

In conclusion, the incidence of progressive EAH at the original FTP site after the treatment of CDS was 22.0% in our study. Hematoma volume ≥ 10 mL at the original impact location and the absence of an apparent midline shift (< 5 mm) are significant risk factors for progression of EAH. Patients with fractures at the original impact site had a higher incidence of this complication; however, multivariate logistic model showed it was not a significant predictor. Although progressive EAH after CDS is devastating, timely CT scans and evacuation treatment are crucial and efficient.

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Ethical Statement

This study has been approved by the local ethical committee. Patient privacy is particularly note and data use is limited to this study.

Declaration of Competing Interest

There are no conflicts of interest.

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