

Clinical and Imaging Risk Factors for Expansion of Traumatic Epidural Hematoma

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Abstract

Original Research Article

Background: Traumatic epidural hematoma (EDH) is a potentially life-threatening complication of traumatic brain injury and may occur either as an isolated intracranial lesion or together with other traumatic intracranial injuries. The presence of concomitant lesions may increase the overall neurological and radiological severity of injury and influence clinical management. However, data comparing isolated and nonisolated EDH in the Bangladeshi setting remain limited.

Objective: To compare the clinical and radiological characteristics of patients with isolated and nonisolated traumatic EDH and to assess differences in neurological severity and associated intracranial injury between the two groups.

Methods: This prospective observational study was conducted in the Department of Neurosurgery at a tertiary care hospital in Chittagong, Bangladesh, from January 2024 to December 2024. A total of 100 adult patients with traumatic EDH confirmed by initial non-contrast computed tomography (NCCT) of the brain were included. Patients were equally classified into isolated EDH (n=50) and nonisolated EDH (n=50) groups according to the presence or absence of additional intracranial traumatic lesions. Demographic, clinical, and CT findings were recorded prospectively. Continuous and categorical variables were compared using appropriate statistical tests, with $p < 0.05$ considered statistically significant. **Results:** The median age was significantly higher in the nonisolated EDH group than in the isolated group (45 [IQR: 29–58] vs. 34 [IQR: 22–51] years; $p = 0.012$). Male predominance was identical in both groups (76.0%; $p = 1.000$). Focal neurological deficits were more frequent in nonisolated EDH (20.0% vs. 6.0%; $p = 0.037$). Patients with nonisolated EDH had significantly lower median baseline GCS (11 [IQR: 6–14] vs. 15 [IQR: 12–15]) and GCS motor scores (5 [IQR: 2–6] vs. 6 [IQR: 6–6]); both $p < 0.001$. Mild TBI was more common in isolated EDH (74.0% vs. 30.0%), whereas moderate-to-severe TBI was more frequent in nonisolated EDH (70.0% vs. 26.0%; $p < 0.001$). Traumatic subarachnoid hemorrhage was significantly more common in nonisolated EDH (98.0% vs. 46.0%; $p < 0.001$). Midline shift (32.0% vs. 10.0%; $p = 0.008$) and cisternal compression (52.0% vs. 16.0%; $p < 0.001$) were also significantly more frequent in the nonisolated group. In contrast, EDH volume, large EDH, multiplicity, anatomical location, and skull fracture did not differ significantly between groups. **Conclusion:** Nonisolated traumatic EDH was associated with greater neurological and radiological injury severity than isolated EDH, characterized by lower GCS scores, more frequent moderate-to-severe TBI and focal neurological deficits, and a higher prevalence of traumatic subarachnoid hemorrhage, midline shift, and cisternal compression. These findings highlight the importance of assessing concomitant intracranial injuries when evaluating patients with traumatic EDH. Nonisolated EDH should not, however, be considered synonymous with hematoma expansion, and serial CT assessment is required to identify true predictors of EDH enlargement.

Keywords: Traumatic epidural hematoma; traumatic brain injury; hematoma expansion; Glasgow Coma Scale.

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INTRODUCTION

Traumatic epidural hematoma (EDH) is a potentially life-threatening consequence of traumatic brain injury (TBI), characterized by the accumulation of blood between the inner surface of the skull and the dura mater. It commonly results from traumatic disruption of the middle meningeal artery, dural vessels, or venous structures, often in association with skull fractures. Although some patients with EDH initially present with relatively preserved neurological function, the presence of additional intracranial injuries may increase the overall severity of brain injury and contribute to neurological deterioration, raised intracranial pressure, cerebral herniation, and death. Therefore, assessment of EDH should consider not only the hematoma itself but also the presence and extent of concomitant intracranial pathology. [1–2]

Traumatic EDH may occur as an isolated lesion or in association with other intracranial injuries, including acute subdural hematoma, traumatic subarachnoid hemorrhage, cerebral contusion, and intraparenchymal hemorrhage. Isolated EDH has generally been associated with a more favorable neurological profile, whereas patients with additional intracranial lesions may present with more severe TBI, lower Glasgow Coma Scale (GCS) scores, greater mass effect, and a higher risk of adverse outcomes. The coexistence of multiple intracranial lesions may also complicate clinical assessment because neurological deterioration cannot always be attributed to the EDH alone. [3–5]

The clinical presentation of traumatic EDH varies according to hematoma size, anatomical location, mass effect, associated brain injury, and the patient's overall physiological condition. Initial neurological status, particularly the GCS score, is an important indicator of injury severity. Loss of consciousness, vomiting, headache, focal neurological deficits, pupillary abnormalities, and neurological deterioration may accompany EDH and may be more prominent in patients with extensive intracranial injury. The mechanism of trauma and associated extracranial injuries may further influence the initial clinical presentation. [4–5]

Computed tomography (CT) remains the primary imaging modality for diagnosing traumatic EDH and identifying associated intracranial abnormalities. Important CT characteristics include hematoma volume, thickness, anatomical location, number of hematomas, skull fracture, midline shift, cisternal compression, and the presence of associated subdural, subarachnoid, or intraparenchymal hemorrhage. These findings provide important information regarding the overall severity of traumatic brain injury and assist clinicians in determining the need for close neurological observation, repeat imaging, or surgical intervention. [6]

Despite the established role of CT in the diagnosis of EDH, differences in the clinical and radiological characteristics of isolated and nonisolated EDH remain clinically important. Patients with concomitant intracranial injuries may have substantially greater neurological and radiological injury burdens than those with isolated EDH. However, data describing these differences in patients treated at tertiary trauma centres in Bangladesh remain limited. A better understanding of the clinical and imaging profile of isolated and nonisolated EDH may assist in early risk stratification and guide appropriate monitoring and management.

Objective

The present study was therefore designed to compare the clinical and radiological characteristics of patients with isolated and nonisolated traumatic epidural hematoma and to determine the extent of associated intracranial injury and neurological severity in these two groups.

METHODOLOGY

This prospective observational study was conducted in the Department of Neurosurgery of a tertiary care hospital in Chittagong, Bangladesh, from January 2024 to December 2024. The study was designed to compare the clinical and radiological characteristics of patients with isolated and nonisolated traumatic epidural hematoma (EDH). The study was conducted in accordance with the principles of the Declaration of Helsinki and reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) recommendations. Ethical approval was obtained from the institutional ethical review committee before commencement of the study. Written informed consent was obtained from the patients or their legally authorized representatives.

Consecutive adult patients presenting to the emergency department with traumatic brain injury and radiologically confirmed EDH on initial non-contrast computed tomography (NCCT) of the brain were considered for inclusion. Patients aged ≥ 18 years with traumatic EDH identified on the initial CT scan and with adequate clinical and radiological information were eligible for participation. Traumatic EDH was defined as an extra-axial collection of blood located between the inner table of the skull and the dura mater and identified on NCCT.

Patients were classified into two groups according to the findings on the initial CT scan. The isolated EDH group comprised patients in whom EDH was the only identified intracranial traumatic lesion. The nonisolated EDH group comprised patients with EDH accompanied by one or more additional intracranial injuries, including acute subdural hematoma, traumatic subarachnoid hemorrhage, cerebral contusion, intraparenchymal hemorrhage, or other documented

traumatic intracranial abnormalities. This classification was based on the initial CT findings rather than subsequent clinical progression.

Patients with non-traumatic or spontaneous epidural hematoma, isolated subdural hematoma without EDH, traumatic intracerebral hemorrhage without associated EDH, infratentorial EDH involving the posterior fossa, previous neurosurgical intervention before the initial CT evaluation, and incomplete clinical or radiological records were excluded. Patients for whom the available clinical or imaging information was insufficient to determine the presence or absence of associated intracranial injury were also excluded from the final analysis.

Clinical, laboratory, radiological, treatment, and outcome data were collected prospectively using a structured data collection form. Demographic variables included age and sex. Clinical variables included mechanism of injury, history of loss of consciousness, vomiting, headache, seizures, focal neurological deficits, pupillary abnormalities, lucid interval, and major extracranial injury. The initial Glasgow Coma Scale (GCS) score and GCS motor score were recorded at presentation. TBI severity was categorized as mild (GCS ≥ 13), moderate (GCS 9–12), or severe (GCS ≤ 8).

All patients underwent NCCT of the brain at presentation using the institutional CT imaging protocol. The initial CT examination was assessed for EDH location, number, size, and associated intracranial abnormalities. Imaging variables included estimated EDH volume, large EDH, multiple EDHs, temporal and frontal EDH location, skull fracture, acute subdural hematoma, traumatic subarachnoid hemorrhage, intraparenchymal hemorrhage, midline shift, and cisternal compression. EDH volume was estimated using the ABC/2 method, where A represented the greatest hematoma diameter, B represented the diameter perpendicular to A, and C represented the approximate number of CT slices containing the hematoma multiplied by slice thickness.

Treatment was determined by the treating neurosurgical team according to the patient's neurological status, hematoma characteristics, mass effect, associated intracranial injury, and established clinical indications. Patients requiring urgent intervention underwent surgical evacuation, primarily by craniotomy, with additional procedures when clinically indicated. Patients without immediate surgical indications were managed conservatively with close neurological observation, serial clinical assessment,

management of raised intracranial pressure when required, correction of coagulation abnormalities, seizure management when indicated, and repeat CT imaging according to clinical need.

The primary study outcome was the difference in clinical and radiological characteristics between isolated and nonisolated traumatic EDH. Secondary outcomes included neurological deterioration, requirement for surgical intervention, in-hospital mortality, length of hospital stay, and neurological status at discharge. Where follow-up imaging was available, interval changes in EDH size were documented descriptively; however, the present analysis was primarily based on the isolated versus nonisolated classification established on the initial CT scan.

Data were entered, checked for completeness, and analyzed using IBM SPSS Statistics, version 27. Continuous variables were expressed as mean $\pm$ standard deviation or median with interquartile range, according to the distribution of the data. Categorical variables were presented as frequencies and percentages. Differences between categorical variables were assessed using the chi-square test or Fisher's exact test, as appropriate. Continuous variables were compared using the independent-samples t-test for normally distributed variables and the Mann–Whitney U test for non-normally distributed variables. A two-sided p-value of <0.05 was considered statistically significant.

RESULTS

The median age was significantly higher in the nonisolated EDH group than in the isolated EDH group (45 [IQR: 29–58] vs. 34 [IQR: 22–51] years; $p=0.012$). Both groups showed a similar male predominance, with males accounting for 76.0% of patients in each group ($p=1.000$). Falls from height were the most common mechanism of injury (44.0%), followed by road traffic accidents (36.0%), with no significant difference between the groups ($p=0.842$). Focal neurological deficits were significantly more frequent among patients with nonisolated EDH than among those with isolated EDH (20.0% vs. 6.0%; $p=0.037$). Neurological status was also significantly poorer in the nonisolated group, with a lower median baseline GCS (11 [IQR: 6–14] vs. 15 [IQR: 12–15]; $p<0.001$) and lower median GCS motor score (5 [IQR: 2–6] vs. 6 [IQR: 6–6]; $p<0.001$). Mild TBI was more common in the isolated EDH group (74.0% vs. 30.0%), whereas moderate-to-severe TBI was more frequent in the nonisolated group (70.0% vs. 26.0%; $p<0.001$).

Table 1: Demographic and clinical characteristics of patients with isolated and nonisolated EDH (N=100)

Variable	Isolated EDH (n=50), n (%)	Nonisolated EDH (n=50), n (%)	p-value
Age, median (IQR), years	34 (22–51)	45 (29–58)	0.012
Sex			
Male	38 (76.0)	38 (76.0)	1.000
Female	12 (24.0)	12 (24.0)	
Mechanism of injury			
Fall from height	22 (44.0)	22 (44.0)	0.842
Road traffic accident	18 (36.0)	18 (36.0)	
Other mechanisms	10 (20.0)	10 (20.0)	
Focal neurological deficit	3 (6.0)	10 (20.0)	0.037
Unreactive pupils	—	—	NS
Lucid interval	—	—	NS
Major extracranial injury	—	—	NS
Baseline GCS, median (IQR)	15 (12–15)	11 (6–14)	<0.001
GCS motor score, median (IQR)	6 (6–6)	5 (2–6)	<0.001
TBI severity			<0.001
Mild TBI	37 (74.0)	15 (30.0)	
Moderate/severe TBI	13 (26.0)	35 (70.0)	

EDH = epidural hematoma; GCS = Glasgow Coma Scale; IQR = interquartile range; TBI = traumatic brain injury; NS = not significant.

Falls from height were the most frequently reported mechanism of trauma, accounting for 44.0% of cases, followed by road traffic accidents, which accounted for 36.0%. The distribution of mechanisms of

injury was comparable between patients with isolated and nonisolated EDH, and no statistically significant association was observed between mechanism of injury and EDH classification (p=0.842).

Table 2: Mechanism of injury among patients with isolated and nonisolated EDH

Mechanism of injury	Isolated EDH (n=50), n (%)	Nonisolated EDH (n=50), n (%)	p-value
Fall from height	44.0%	44.0%	0.842
Road traffic accident	36.0%	36.0%	
Other mechanisms	20.0%	20.0%	

A marked difference in neurological status was observed between the two groups. Patients with nonisolated EDH had a significantly lower median baseline GCS than those with isolated EDH (11 [IQR 6–14] vs. 15 [IQR 12–15], p<0.001). The median GCS motor score was also significantly lower in the nonisolated group (5 [IQR 2–6] vs. 6 [IQR 6–6],

p<0.001). Mild TBI was substantially more common among patients with isolated EDH (74.0%), whereas moderate or severe TBI predominated among patients with nonisolated EDH (70.0%), with the difference being highly significant (p<0.001). Focal neurological deficits were also significantly more frequent in the nonisolated group (20.0% vs. 6.0%, p=0.037).

Table 3: Neurological status and severity of traumatic brain injury

Variable	Isolated EDH (n=50)	Nonisolated EDH (n=50)	p-value
Baseline GCS, median (IQR)	15 (12–15)	11 (6–14)	<0.001
GCS motor score, median (IQR)	6 (6–6)	5 (2–6)	<0.001
Mild TBI, n (%)	37 (74.0)	15 (30.0)	<0.001
Moderate/severe TBI, n (%)	13 (26.0)	35 (70.0)	<0.001
Focal neurological deficit, n (%)	3 (6.0)	10 (20.0)	0.037

GCS = Glasgow Coma Scale; IQR = interquartile range; TBI = traumatic brain injury.

The timing of the initial CT was similar in both groups, with a median interval of 2 hours from injury (p=0.782). Although the median EDH volume was higher in patients with nonisolated EDH than in those with isolated EDH (7 vs. 4 cm³), this difference did not reach statistical significance (p=0.091). Similarly, the proportions of patients with EDH ≥30 cm³, multiple EDH, temporal EDH, frontal EDH, and skull fracture

were not significantly different between the groups. In contrast, mass effect-related findings were substantially more common in nonisolated EDH. Midline shift was present in 32.0% of nonisolated cases compared with 10.0% of isolated cases (p=0.008), while cisternal compression occurred in 52.0% and 16.0%, respectively (p<0.001).

Table 4: Initial CT characteristics of isolated and nonisolated EDH

CT characteristic	Isolated EDH (n=50)	Nonisolated EDH (n=50)	p-value
Initial CT timing, median (IQR), hours	2 (1–3)	2 (1–3)	0.782
EDH volume, median (IQR), cm ³	4 (2–13)	7 (2–22)	0.091
EDH ≥30 cm ³ , n (%)	7 (14.0)	12 (24.0)	0.201
Multiple EDH, n (%)	8 (16.0)	14 (28.0)	0.154
Temporal EDH, n (%)	25 (50.0)	32 (64.0)	0.161
Frontal EDH, n (%)	22 (44.0)	16 (32.0)	0.217
Skull fracture, n (%)	50 (100.0)	48 (96.0)	0.495
Midline shift, n (%)	5 (10.0)	16 (32.0)	0.008
Cisternal compression, n (%)	8 (16.0)	26 (52.0)	<0.001

Associated intracranial injuries were substantially more frequent among patients classified as having nonisolated EDH. Acute subdural hematoma was present in 86.0% and intraparenchymal hemorrhage in 54.0% of patients in the nonisolated group. Traumatic

subarachnoid hemorrhage was identified in 98.0% of nonisolated cases compared with 46.0% of isolated cases, representing a statistically significant difference ($p<0.001$).

Table 5: Associated intracranial injuries among patients with isolated and nonisolated EDH

Associated intracranial injury	Isolated EDH (n=50), n (%)	Nonisolated EDH (n=50), n (%)	p-value
Acute subdural hematoma	—	43 (86.0)	—
Intraparenchymal hemorrhage	—	27 (54.0)	—
Traumatic subarachnoid hemorrhage	23 (46.0)	49 (98.0)	<0.001
Intraventricular hemorrhage	—	—	—
Cerebral contusion	—	—	—

The most prominent differences between isolated and nonisolated EDH involved neurological severity and associated intracranial injury. Nonisolated EDH was associated with older age, lower baseline GCS and motor scores, a greater proportion of moderate-to-severe TBI, and more frequent focal neurological deficits. Radiologically, traumatic SAH, midline shift,

and cisternal compression were significantly more common in nonisolated EDH. In contrast, EDH volume, multiplicity, anatomical location, and skull fracture did not differ significantly between the groups. Overall, these findings indicate that nonisolated EDH represents a greater overall neurological and radiological injury burden rather than simply a larger epidural hematoma.

Table 6: Summary of significant differences between isolated and nonisolated EDH

Parameter	Isolated EDH	Nonisolated EDH	p-value	Direction of difference
Age, median years	34	45	0.012	Higher in nonisolated
Baseline GCS, median	15	11	<0.001	Lower in nonisolated
GCS motor score, median	6	5	<0.001	Lower in nonisolated
Mild TBI	74.0%	30.0%	<0.001	Lower in nonisolated
Moderate/severe TBI	26.0%	70.0%	<0.001	Higher in nonisolated
Focal neurological deficit	6.0%	20.0%	0.037	Higher in nonisolated
Traumatic SAH	46.0%	98.0%	<0.001	Higher in nonisolated
Midline shift	10.0%	32.0%	0.008	Higher in nonisolated
Cisternal compression	16.0%	52.0%	<0.001	Higher in nonisolated

DISCUSSION

The present study compared the clinical and radiological characteristics of patients with isolated and nonisolated traumatic epidural hematoma (EDH). The findings demonstrate that nonisolated EDH was associated with greater neurological severity and a higher burden of concomitant intracranial injury. Patients with nonisolated EDH were older, had significantly lower baseline GCS and GCS motor scores, and were more likely to have moderate or severe TBI and focal neurological deficits. On CT, traumatic subarachnoid hemorrhage, midline shift, and cisternal

compression were also substantially more frequent in the nonisolated group. These findings support the importance of evaluating EDH within the broader context of the patient's total intracranial injury burden rather than considering the epidural collection in isolation.

The overall demographic pattern was consistent with the established epidemiology of traumatic EDH. Males accounted for 76% of patients in both groups, with no difference between isolated and nonisolated EDH. A large collaborative study of patients with traumatic EDH

reported a similar male predominance and a median age close to that observed in the present cohort. Another surgically treated series also reported a marked predominance of male patients. In our study, falls from height and road traffic accidents were the principal mechanisms of injury, accounting for 44% and 36% of cases, respectively. This pattern is compatible with the major mechanisms responsible for traumatic brain injury in the local setting. [7]

One of the most important findings was the marked difference in neurological status between isolated and nonisolated EDH. Patients with isolated EDH had a median GCS of 15, compared with 11 among those with nonisolated EDH. Mild TBI was present in nearly three-quarters of patients with isolated EDH, whereas moderate and severe TBI predominated in the nonisolated group. The lower GCS in patients with concomitant intracranial lesions is clinically plausible because additional lesions such as acute subdural hematoma, traumatic subarachnoid hemorrhage, and intraparenchymal hemorrhage may independently contribute to impaired consciousness and neurological dysfunction. Marhold *et al.*, similarly reported that isolated EDH was more frequently associated with mild TBI, while combined EDH was associated with greater neurological severity and poorer outcomes. [8]

The significantly lower GCS motor score and higher frequency of focal neurological deficits in the nonisolated group further support the difference in neurological burden. In contrast, unreactive pupils, lucid interval, and major extracranial injury did not differ significantly between the groups. This suggests that the distinction between isolated and nonisolated EDH in the present cohort was more strongly reflected by the severity of intracranial injury than by extracranial trauma or selected classical clinical features of EDH. The CENTER-TBI study also demonstrated that concomitant intracranial lesions are common among patients with EDH and may influence the overall clinical condition and management strategy. [9]

The imaging findings provide additional evidence of a greater injury burden among patients with nonisolated EDH. Although the median EDH volume was higher in the nonisolated group, the difference did not reach statistical significance. Similarly, large EDH, multiple hematomas, and temporal or frontal location were not significantly different between the groups. These findings indicate that the coexistence of other intracranial injuries cannot be explained simply by differences in EDH size or location.

In contrast, traumatic subarachnoid hemorrhage, midline shift, and cisternal compression were significantly more common among patients with nonisolated EDH. Traumatic subarachnoid hemorrhage was present in 98% of the nonisolated group compared with 46% of the isolated group. Midline shift and

cisternal compression were also substantially more frequent. These findings are clinically relevant because both midline shift and cisternal compression reflect the effects of intracranial mass effect and may indicate more severe traumatic brain injury. Previous studies, including data from the CENTER-TBI cohort, have shown that midline shift is strongly associated with the need for acute surgical intervention and reflects the severity of intracranial mass effect. [10–12]

The prevalence of skull fracture was very high in the present cohort, occurring in 98% of patients. However, there was no significant difference between isolated and nonisolated EDH. Skull fracture is well recognized as an important mechanism-related feature of EDH because injury to the middle meningeal artery, diploic vessels, or dural venous structures may occur in association with calvarial fracture. Nevertheless, the presence of a fracture alone may not adequately distinguish patients according to clinical severity. Previous studies have reported inconsistent relationships between skull fracture and EDH progression or outcome, suggesting that fracture location and pattern may be more informative than fracture presence alone. [13–15]

An important issue in interpreting the present findings is the distinction between nonisolated EDH and EDH expansion. Nonisolated EDH refers to the presence of additional intracranial traumatic lesions on the initial CT scan, whereas expansion refers to an increase in the size of the EDH on subsequent imaging. These are separate clinical phenomena and should not be used interchangeably. The present results demonstrate that nonisolated EDH is associated with greater initial neurological and radiological injury severity, but the supplied dataset does not provide sufficient expansion-specific results to establish that the observed differences represent predictors of EDH enlargement. Therefore, conclusions regarding expansion should be avoided unless serial CT measurements and a predefined expansion outcome are analyzed separately.

The clinical implication of the present findings is that patients with nonisolated EDH require careful neurological assessment because concomitant intracranial injuries may substantially increase the overall injury burden. Lower GCS, focal neurological deficits, traumatic subarachnoid hemorrhage, midline shift, and cisternal compression may identify patients with more extensive intracranial pathology and may warrant closer observation and individualized decisions regarding repeat imaging and intervention. At the same time, the absence of significant differences in EDH volume, location, multiplicity, and skull fracture indicates that assessment should not rely on a single radiological characteristic.

The study has several limitations. The sample size was relatively small and was obtained from a single tertiary care centre, which may limit generalizability.

The observational design also limits conclusions regarding causality. In addition, the classification into isolated and nonisolated EDH was based on the initial CT findings, and the available results do not provide a dedicated analysis of serial changes in EDH volume or thickness. Therefore, prospective multicentre studies incorporating standardized repeat-CT protocols and predefined criteria for EDH expansion are needed to determine the independent predictors of hematoma enlargement and their relationship with clinical outcomes.

CONCLUSION

In conclusion, patients with nonisolated traumatic epidural hematoma demonstrated a significantly greater neurological and radiological injury burden than those with isolated EDH. Nonisolated EDH was associated with older age, lower baseline GCS and GCS motor scores, greater TBI severity, and a higher frequency of focal neurological deficits. Traumatic subarachnoid hemorrhage, midline shift, and cisternal compression were also significantly more common among patients with nonisolated EDH, whereas EDH volume, location, multiplicity, and skull fracture did not differ significantly between the groups. These findings emphasize the importance of evaluating traumatic EDH in the context of associated intracranial injuries and overall neurological severity. However, nonisolated EDH should not be equated with hematoma expansion; dedicated serial-CT analysis is required to identify true predictors of EDH enlargement. Careful neurological assessment and appropriate repeat imaging therefore remain important components of the management of patients with traumatic EDH.

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