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Asian J Neurosurg 2024;19:452–461.

article published online June 24, 2024

DOI [https://doi.org/](https://doi.org/10.1055/s-0043-1775731) [10.1055/s-0043-1775731](https://doi.org/10.1055/s-0043-1775731). ISSN 2248-9614.

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Thieme Medical and Scientific Publishers Pvt. Ltd., A-12, 2nd Floor, Sector 2, Noida-201301 UP, India

Conclusion Patients with traumatic EDH fitting the criteria of initial nonsurgical treatment necessitates 48 hours of close observation and serial CT scans at 6, 12, 24, and 48 hours to confirm the regression or early detect the EDHP. Patients with high GCS, frontal hematomas, and associated fissure fracture are at low risk for EDHP. Increased alertness is mandatory for young age and patients with persistent nausea/vomiting, early CT scan, temporal hematomas, or coagulopathy.

Introduction

Extradural hematoma (EDH), the most serious preventable complication of head injury, is encountered in \sim 2.7 to 4% of head injury patients.^{1,2} Patients with acute EDH greatly vary in their mechanisms of trauma and clinical presentations.³

EDH may attain maximum size within minutes of injury; however, it may gradually progress over the first 24 hours after injury. Rebleeding or continuous oozing is the main cause of progression. Different sources of bleeding in EDH include laceration of the middle meningeal artery, venous oozing, or laceration of dural venous sinus.⁴

Treatment of asymptomatic or mildly symptomatic EDH is a matter of debate among neurosurgeons. With the considerable safety of EDH surgery, many neurosurgeons prefer to go for surgical evacuation in doubtful cases to avoid the risk of significant brain compression and secondary ischemic damage.⁵

Although surgical evacuation is considered the definitive treatment for acute EDH, several recent reports have described successful conservative management. Several reports of conservatively managed EDH suggested that some of these lesions may resolve spontaneously without squeal. Conservative management requires careful patient selection together with close clinical observation and serial follow-up computed tomography (CT) scans. $6,7$

Various clinical and radiographic factors have been found to affect the management strategy for EDH. So, in our study, the main objective was to identify the significant predictors for possible regression versus progression of acute posttraumatic EDH (EDHR and EDHP) initially planned for nonsurgical treatment.

Materials and Methods

Study Design and Patients

Our study is a retrospective comparative study conducted from October 2020 to October 2022. We revised the data of all patients with traumatic brain injury (TBI) who were admitted to our hospital during this period; a total of 195 patients diagnosed with acute posttraumatic EDH were collected; 76 patients required urgent surgical evacuation and were immediately shifted to the operating room, while 119 patients were initially treated conservatively and were put under close observation.

The criteria for initial nonsurgical management for EDH were consistent with the literature^{2,6,7} and included; EDH

volume $\leq 30 \text{ cm}^3$, midline shift (MLS) $\leq 5 \text{ mm}$, maximum hematoma thickness ≤ 10 mm, and no associated neurological deficit. Patients, who were initially treated conservatively and fulfilled our inclusion criteria, were divided into two groups: (EDHR group) patients in whom the EDH started to regress spontaneously and (EDHP group) patients who developed EDHP and subsequent surgical evacuation.

Ethical Approval

This study was approved by the local ethical scientific committee of our institution (Institutional Review Board approval number: 3-2023.NEUS 1–5). Being a retrospective study, patients' consents for participation in the study and for publication were not applicable.

Sample Size Estimation

A previous study showed that the odds ratio of the coagulopathy in predicting conversion to surgery in patients with EDH was 6.122. So, the sample size to study the results of the current study with a significant $p < 0.05$ and power of study of 80% is calculated according to the OpenEpi⁸ calculator. So, at least, 106 patients should be recruited to the study.

Inclusion Criteria

In this study, we included posttraumatic patients of either sex with no age restriction who had acute EDH diagnosed on CT of the brain and were initially planned for conservative treatment.

Exclusion Criteria

We excluded patients with (1) associated other intracranial pathology that required surgical intervention, (2) incomplete data or did not continue for follow-up, (3) postoperative or recurrent EDH, (4) bilateral EDH in CT scan, and (5) history of significant premorbid psychiatric or neurological history or drug abuse.

Data Collection

Demographic, clinical, and radiographic data were collected from patients' medical records of our hospital including data on and during the period of admission then data during the first 3 months after discharge.

All patients were submitted to full medical history, general examination, and full neurological assessment. Evaluation of age; sex; mechanism of trauma including fall from height (FFH), road traffic accident, or assault; clinical presentations (loss of consciousness [LOC], headache,

nausea/vomiting, or posttraumatic amnesia); GCS on and during admission; severity of head injury including mild (GCS 13–15), moderate (GCS 9–12), or severe (GCS 3–8); time interval from trauma to the initial CT scan; and presence of coagulopathy.

All patients were submitted for CT scan on admission to detect the side, location, volume, and maximum thickness of the EDH; measure the degree of MLS; and detect associated fracture or other intracranial injuries. EDH volume was calculated in three dimensions. The width was measured as the transverse diameter, the length as the anteroposterior diameter, and the depth as the superoinferior diameter. Approximated volume was computed by multiplying the three dimensions using the equation: volume = $ABC/2^9$

Management

First, resuscitation efforts were performed including ABC (assessment and stabilization of Airway patency, Breathing, and Circulation). A thorough trauma evaluation was done and severity of TBI was assessed using GCS. During admission, close observation and repeated neurological examinations were done. CT of the brain was done upon presentation then routinely repeated after 6, 12, 24, and 48 hours. However, CT was immediately performed whenever neurological deterioration occurred.

Dehydrating measures, cerebroprotective agents, and anticonvulsive drugs were given in certain cases that had concomitant brain injury, edema, convulsion, or threatening to coma.

While under observation, urgent craniotomy and EDH evacuation were performed if the patient developed signs of localized brain compression or herniation that was confirmed by progression of EDH in CT scan.

Outcome Measures

Outcome measures were: (1) percentage of patients treated conservatively and showed spontaneous regression of their EDH; (2) percentage of patients initially treated conservatively then developed progression of their EDH and subsequent surgical evacuation; (3) timing for complete spontaneous

Table 1 The Extended Glasgow Outcome Scale score¹⁰

EDH resolution; (4) timing for EDHP and delayed surgical evacuation; (5) the Extended Glasgow Outcome Scale (GOSE) score¹⁰ is shown in **-Table 1.** GOSE score was measured at discharge from our department, whether the discharge destination was home or another medical facility. Patients who had moderate disability or good recovery (GOSE score from 5 to 8) were included together in the good outcome group. Patients who were severely disabled, vegetative, or died (GOSE score from 1 to 4) were included together in the poor outcome group.

Statistical Analysis

To tabulate and statistically analyze the results, SPSS V.22 (IBM Corporation, Armonk, New York, United States), and Microsoft Excel 2010 (Microsoft Corporation, One Microsoft Way Redmond, Washington, United States) were used. The descriptive statistics included mean (x), median, and standard deviation. The count data were expressed as the rate and analyzed using the chi-square test. Standard Student's t -test (t) , for independent samples was used for comparing the means between the two groups in various factors of the study. A p-value \leq 0.05 was considered statistically significant.

Results

A total of 119 head trauma patients were diagnosed with acute EDH and were initially planned for conservative treatment; 13 patients were excluded (4 patients had incomplete data, 1 patient had bilateral EDH, and 8 patients did not complete for follow-up). So, in our study, we included 106 patients; 74 patients (69.8%) showed spontaneous regression of their EDH, while 32 patients (30.2%) developed EDHP and were shifted for surgical evacuation.

Demographic and Clinical Data of the Entire Sample

The mean age in the entire sample was 20.37 ± 12.712 years, ranging from 2 to 53 years, the distribution of age between the two groups is demonstrated in ►Fig. 1. The majority of cases were male (65.1%). GCS on admission ranged from 5 to

Fig. 1 Age distribution in the whole sample. EDHP, extradural hematoma progression; EDHR, extradural hematoma regression.

15 with the mean GCS of 12.83 ± 2.113 . The majority of cases (67.9%) had a mild TBI (GCS 13–15). FFH was the most frequent mechanism of injury (48.1%). The most frequent clinical presentations included headache (56.6%) followed by LOC (48.1%). Coagulation abnormalities "high international normalized ratio" (INR) was identified in (6.6%). ►Table 2 gives the detailed demographic and clinical data in each

group and their association with either regression or progression of EDH.

Radiographic Data of the Entire Sample

The mean time interval between trauma and initial CT was 10.33 ± 7.815 hours. CT showed right-sided EDH in 56.6% of cases. The most common locations for EDH were frontal,

Table 2 Comparison of demographic and clinical data of patients in the two groups

Abbreviations: EDHP, extradural hematoma progression; EDHR, extradural hematoma regression; FFH, fall from height; GCS, Glasgow coma scale; INR, international normalized ratio; LOC, loss of consciousness; RTA, road traffic accident; SD, standard deviation. Statistically significant.

Parameters	EDHR group $(N = 74)$	EDHP group $(N = 32)$	Test and significance
Time from trauma to CT (mean \pm SD)	11.31 ± 8.485	8.06 ± 5.453	$t = 2.355$, $p = 0.021^*$
Side of EDH (right/left)	41/33	19/13	Chi-square = 0.143, $p = 0.705$
Location of EDH			
F (34.9% of total)	41.9%	18.8%	Chi-square = 5.265, $p = 0.022^*$
P (31.1% of total)	35.1%	21.9%	Chi-square = 1.832, $p = 0.176$
T (21.7% of total)	10.8%	46.9%	Chi-square = 17.101, $p < 0.001$ [*]
$O(8.5\% \text{ of total})$	8.1%	9.3%	Chi-square = 0.046, $p = 0.830$
PF (3.8% of total)	4.1%	3.1%	Chi-square = 0.53, $p = 0.818$
EDH volume (mean \pm SD) cm ³	17.82 ± 4.906	17.13 ± 5.824	$t = 0.636, p = 0.526$
Maximum EDH thickness (mean \pm SD)	5.23 ± 1.997	4.72 ± 1.922	$t = 1.223$, $p = 0.224$
MLS (mean \pm SD)	2.08 ± 1.678	$1.63 + 1.661$	$t = 1.289, p = 0.200$
Associated fissure fracture	47.3%	21.9%	Chi-square = 6.035, $p = 0.014^*$

Table 3 Comparison of the radiographic data of patients in the two groups

Abbreviations: CT, computed tomography; EDH, extradural hematoma; EDHP, extradural hematoma progression; EDHR, extradural hematoma regression; F, frontal; MLS, midline shift; O, occipital; P, parietal; PF, posterior fossa; SD, standard deviation; T, temporal. Statistically significant.

parietal, and temporal (34.9, 31.1, and 21.7%, respectively). EDH volume was $\leq 30 \text{ cm}^3$ in all cases and the mean EDH volume was $17.61 \pm 5.182 \text{ cm}^3$. The mean MLS was 1.94 ± 1.678 mm. Associated fissure fracture was found in 39.6% of cases. ►Table 3 demonstrates the comparison of the radiographic data between the two groups and their association with either regression or progression of EDH.

EDHR versus EDHP

Patients of the EDHR group showed a degree of resolution of their EDH after 2 weeks (in the routine follow-up CT of the brain). Complete EDH resolution ranged from 30 to 90 days with the mean time of 59.85 ± 13.363 days. ►Fig. 2 illustrates CT scans of a patient from EDHR group.

Fig. 2 Spontaneous regression of a right frontal EDH. (A-D) Male patient 34 years old belongs to EDHR group, had history of fall from height, GCS on admission was 15/15. (A) Initial CT of the brain 11 hours after trauma showing right frontal EDH; (B) follow-up CT after 24 hours with no increase in hematoma size; (C) follow-up CT after 2 weeks with start of EDH regression; (D) follow-up CT after 45 days showing complete resolution of the extradural hematoma. CT, computed tomography; EDH, extradural hematoma; EDHR, extradural hematoma regression; GCS, Glasgow coma scale.

Fig. 3 Progression of left parietal EDH. (A, B) Male patient 10 years old belongs to EDHP group, had history of RTA, GCS on admission was 15/15, was complaining for vomiting. (A) Initial CT of the brain 2 hours after trauma showing left parietal small EDH; (B) follow-up CT after 12 hours, showing increase in hematoma size and the patient was shifted for surgical evacuation. CT, computed tomography; EDH, extradural hematoma; EDHP, extradural hematoma progression; GCS, Glasgow coma scale.

EDHP was detected on routine follow-up CT, except for seven patients (21.9%) who developed neurological deterioration and CT was repeated urgently. The mean EDH volume after progression was 35.78 ± 5.405 cm³. The time interval from the initial CT to EDHP ranged from 6 to 30 hours with the mean time of 14.53 ± 5.43 hours. \blacktriangleright Figs. 3 and 4 illustrate CT scans of two patients from EDHP group.

Recovery Outcome on Discharge

The majority of cases (92.5%) had good recovery outcome $(GOSE = 5-8)$ at discharge, while 8 cases (7.5%) had poor discharge outcome $(GOSE = 1-4)$ including only one death (in EDHP group). \blacktriangleright Table 4 shows the distribution of the GOSE scores in the two groups.

Discussion

Conventionally, the accepted management for EDH is urgent craniotomy and hematoma evacuation.³ However, with the routine use of CT in TBI, conservative management of EDH in selected patients has been an accepted management strategy.^{7,11,12}

In our study, only (30.2%) of patients developed EDHP that subsequently required surgical evacuation, while the majority (69.8%) showed spontaneous EDHR and had a successful conservative treatment. We analyzed the different demographic, clinical, and radiographic factors to identify their significant correlation with spontaneous regression versus progression of the EDH.

Patient's age ranged from 2 to 53 years, the active age of life where people are more susceptible to trauma. Also, acute EDH is less frequent among elderly people because of strong adhesion between calvarial bone and dura.¹³ Similar result

was documented by Zwayed and Lucke-Wold's 14 study, where patients' age was from 4 to 55 years.

In our study, younger age was a significant predictive factor for EDHP and conversion to surgery ($p = 0.006$), where the patients \leq 20 years old represented 71.9% in the EDHP group and only 41.9% in the EDHR group. This comes in accordance with Basamh et $al⁶$ who concluded in their study that patients of EDHP group who had surgery were significantly younger than the other group ($p < 0.0001$).

In both groups, males were more commonly affected than females and the most common mechanism of traumawas FFH. Male predominance may be due to the fact that males are more involved in outdoor activities. These results come in accordance with most of other studies conducted on EDH.^{6,13,14}

We did not find any significant association between patients' gender and mechanism of trauma with either regression or progression of EDH ($p > 0.05$). The same results were documented in Basamh et al's $⁶$ study, where the</sup> majority of cases (81.6%) were males and FFH was the most common mechanism of injury; however, both factors were not associated with EDHP.

In EDHR group, 81.1% had mild head trauma (GCS 13–15) in comparison to 37.5% in EDHP group. And so, higher GCS on admission was significantly associated with spontaneous EDHR ($p = 0.002$). Zwayed and Lucke-Wold¹⁴ concluded that patients with GCS of 13 or more can be treated nonoperatively, and this is in agreement with our results. Also, Zakaria et al¹⁵ concluded that EDH can be managed nonoperatively provided that the GCS remains the same with symptomatic improvement. On the other hand, there was no significant correlation between GCS and EDHP in Basamh et al's⁶ study.

In our study, there were no significant differences between the two groups regarding headache, posttraumatic

Fig. 4 (A, B) Male patient 9 years old belongs to EDHP group, had history of FFH, GCS on admission was 14/15, had posttraumatic amnesia, headache, and repeated vomiting. (A) Initial CT of the brain 1 hour after trauma showing right temporal small EDH; (B) follow-up CT after 6 hours, showing increase in hematoma size and the patient was shifted for surgical evacuation. CT, computed tomography; EDH, extradural hematoma; EDHR, extradural hematoma progression; GCS, Glasgow coma scale.

Table 4 Distribution of GOSE scores among patients in the two groups

Abbreviations: EDHP, extradural hematoma progression; EDHR, extradural hematoma regression; GOSE, extended Glasgow outcome scale.

amnesia, or LOC, and none of these presentations was significantly correlated with either regression or progression of EDH ($p > 0.05$). Persistent nausea/vomiting was the only clinical symptom with significant difference in EDHP group $(p = 0.046)$. This may be attributed to increased intracranial pressure secondary to EDHP causing irritation and/or compression of the vomiting center. Other studies did not find significant correlation between any clinical presentation and either EDHR or EDHP.

Coagulation abnormality (high INR) was a significant factor for EDHP and conversion to surgery ($p = 0.001$). Similar results were documented in Basamh et al's $⁶$ study where</sup> coagulopathy was a significant factor for conversion to surgery ($p = 0.009$). Also, Ding et al¹⁶ found a significant correlation between higher INR with EDHP. However, other studies reported no association between coagulopathy and $EDHP^{17–19}$

In our study, a short time interval between onset of trauma and initial CT significantly correlated with EDHP ($p = 0.021$). Knuckey et al²⁰ in a small retrospective study reported 7 of 22 patients developed EDHP; initial CT was done < 6 hours from onset of trauma. Ding J, et al¹⁶ in their study also found that, to a lesser extent, shorter time lapse between trauma onset and initial CT was a significant factor in EDHP.

There were no statistically significant differences between the two groups regarding the hematoma side, volume, maximum thickness, or the degree of MLS ($p > 0.05$).

Our results are similar with Basamh et al's⁶ results, where none of the hematoma side, volume, or the degree of MLS was a predictor of progression. Also, Moussa et al²¹ concluded that EDH can be treated conservatively depending on the neurological state of the patient rather than the size of the hematoma.

In our study, 100% of cases had EDH volume $<$ 30 cm³ which is consistent with most of previous studies $.265,7.22$ Bullock et al²³ found the volume of 12 to 38 mL suitable for conservative management. In Moussa et $al²¹$ study, the maximum volume of the hematoma was 15 ml.

Location of EDH was an important predictive factor in both groups. Regression of EDH was more common in patients with frontal hematomas ($p = 0.022$) while EDHP and conversion to surgery was more evident in patients with temporal hematomas ($p < 0.001$).

Zwayed and Lucke-Wold¹⁴ study of 62 EDH cases treated conservatively showed that the most common locations were the frontal region in 24 cases and parietal region in 17 cases.

Subodh and Hamza²⁴ concluded that EDH in locations other than temporal area can be one of the criteria for conservative management.

A prospective series by Bezircioğlu et al²⁵ on 80 EDH patients treated conservatively concluded that in the 5 patients (6.25%) who developed EDHP, the only significant association was temporal location. Also, Basamh et al's⁶ study showed that 48.0% of EDHP cases were in the temporal region.

In the majority of cases, EDH was the sole finding in the CT scan. The presence of skull fissure fracture was significantly associated with EDHR ($p = 0.014$). These results match the results of Tuncer et al²⁶ who concluded that in patients with skull fractures, clot resorption might be earlier than in others who do not have a skull fracture, partly due to the transfer of the clot into the epicranial space through the fracture. Also, Satyarthee et al¹³ and Moussa et al²¹ found a significant association between the success of conservative treatment and the presence of fissure fracture. Knuckey et al²⁰ in a small retrospective study reported 7 of 22 patients developed EDHP; skull fractures traversing major vascular structures were significant risk factors in EDHP.

EDHP may be a rehemorrhage event or continuous slow bleeding.5,27 In our study, EDHP was detected in the first 24 hours in the majority of cases and less frequently beyond that with the mean time interval from initial CT to EDHP was 14.53 ± 5.43 hours.

Most of other studies had similar results. Ding et al's¹⁶ randomized controlled trial reported that 80% of patients (56 out of 70) complicated with EDHP did so within 24 hours. Basamh et al's⁶ study showed that EDHP occurred from 5 to 30 hours (mean 13.85 hours) after the initial CT.

The majority of cases had good recovery outcome in both groups. EDHP was not associated with either good or poor recovery outcome ($p = 0.639$). This can be attributed to close clinical observation together with serial follow-up CT scans for all patients, and the immediate surgical intervention that was done once EDHP was confirmed. Similar results were documented by Basamh et $al⁶$ where the majority of the sample (87.2%) had a good recovery outcome and they concluded that having progression of the EDH was not associated with better or worse outcome ($p = 0.5730$).

Depending on the results of our study, we made a simple algorithm (►Fig. 5) that demonstrates the criteria of initial

Fig. 5 Our recommended algorithm for the criteria of nonsurgical treatment of posttraumatic EDH and the high-risk criteria for EDH progression with subsequent surgical evacuation. CT, computed tomography; EDH, extradural hematoma; GCS, Glasgow coma scale.

nonsurgical treatment for traumatic EDH and our recommendations to extend these criteria to include patients with high GCS on admission, frontally located hematomas and/or concomitant fissure fracture. Also, we recommended a follow-up time frame of 48 hours for all patients with more attention and increased alertness for those with one or more of the predictors of EDHP.

Limitations

Limitations of our study come from its retrospective nature. Another limitation is that, in our study, some cases of EDH were associated with concomitant injuries on admission. Although these concomitant injuries did not affect either the regression or the progression of EDH, the recovery outcome could be influenced by the severity of the initial injury and not only by the EDH.

Conclusion

Patients with traumatic EDH fitting the criteria of initial nonsurgical treatment necessitates 48 hours of close observation and serial CT scans at 6, 12, 24, and 48 hours to confirm the regression or early detect the EDHP. Patients with high GCS, frontal hematomas, and associated fissure fracture are at low risk for EDHP. Increased alertness is mandatory for young age and patients with persistent nausea/vomiting, early CT scan, temporal hematomas, or coagulopathy.

Note

This article has been read and approved by all the authors. This work was self-funded by the authors. This study was approved by the clinical research committee of the Menoufia University Hospital (IRB approval number: 3-2023.NEUS 1–5) and it followed the tenets of the Declaration of Helsinki.

Availability of Data and Materials

All data and materials included in this work are available.

Ethical Approval Our local ethics committee approved our study.

Authors' Contributions

All authors made a significant contribution to the work reported, whether that was in the conception; study design; execution; and acquisition, analysis, and interpretation of data. All authors took part in drafting, revising, and final approval of the article. All agreed to be accountable for all aspects of the work.

Funding None.

Conflict of Interest None declared.

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