Factors affecting the prognosis of traumatic acute subdural hematomas

Magdy A. Elhawary, Usama M. El Shokhaiby, Wafik E. Ali, Abdallah M. Gafar, Ahmed A. Abd El Rahman

Background Trauma is the most frequent cause of young adult (under 45 years of age) fatalities worldwide, and traumatic brain injury accounts for up to 50% of these deaths. The incidence of traumatic brain injuries is increasing globally, largely due to an increase in motor vehicle use in low-income and middle-income countries.

Purpose The aim of study was to investigate the factors that modulate the prognosis of patients with traumatic acute subdural hematoma.

Patients and methods This study was conducted on 30 patients with acute traumatic subdural hematoma managed in the Department of Neurosurgery, ER, and Surgical ICU, Al-Azhar University Hospitals. All the data collected were statistically analyzed and the results were compared with the international results. Our recommendations were based on that comparison.

Results Mortality rate was 53.3% (16 patients) in our study. The mean and SD of age in our study was 46.3±20.6 years. The range of age was 64 years (21–85 years). There was no statistical difference between sexes as regards age (*P*=0.393). The entire study population was subjected to logistic regression analysis, severity of injury, presence of a secondary injury especially hypoxia or both hypoxia and hypotension. Polytrauma, status of conscious level at admission, anemia, thickness of hematoma, and necessity for endotracheal intubation were enrolled. The hypoxia and lower Glasgow coma scale at admission were found to be significant predictors of mortality.

Conclusion Traumatic acute subdural hematoma still has a high mortality rate despite all developments in neurosurgical interventions. Initially low Glasgow coma scale and hypoxia are important parameters that correlate with the mortality rate.
Sci J Al-Azhar Med Fac, Girls 2019 3:246–251

The Scientific Journal of Al-Azhar Medical Faculty, Girls 2019 3:246–251

Keywords: acute subdural hematoma, Glasgow coma scale, hypotension, hypoxia

Department of Neurosurgery, Al-Azhar University Hospitals, Cairo, Egypt

Correspondence to Dr Ahmed Ali Abd El Rahman, Resident of Neurosurgery Department Al Azhar University, Tel: 002-010-93194656; e-mail: doctorahmed20222@gmail.com

Received 1 January 2019 **Accepted** 10 February 2019

Introduction

Traumatic acute subdural hematoma (ASDH) is one of the most fatal conditions in traumatic head injury. It harbors higher mortality among all traumatic intracranial lesions (40–90%) in many large-scale series [1–4]. Recent researches focused on the quality of patient's life after traumatic brain injuries [5]. Prediction of outcome may facilitate developing diagnostic measures or treatment strategies in order to lower mortality rates. Several predictors such as age and preadmission Glasgow coma scale (GCS) [6–9] were identified previously; other factors were not evaluated yet. The aim of this study was to investigate the factors that modulate the prognosis of patients with traumatic ASDH.

Patients and methods

This was a cohort, uncontrolled observational study held in the Department of Neurosurgery, Al-Azhar University Hospitals from August 2017 to January 2018. The question of the study was: do age, hematoma thickness, comorbidities (e.g. coagulation disorders, hypoxia, hypotension, ..., etc.) and coincidental cranial injuries (axial hematoma and basal fractures) and noncranial injuries affect the outcome in patients with ASDH. This study was conducted on 30 patients with acute traumatic subdural hematoma managed in the department of neurosurgery, ER, and surgical ICU. All the data collected were statistically analyzed and the results were compared with the international results. Our recommendations were based on that comparison.

Inclusion criteria

(1) All age groups.
(2) Both sexes.
(3) Traumatic cases.

Exclusion criteria

(1) Previous chronic subdural hematoma.
(2) Postoperative ASDH.
(3) Spontaneous cases.

Data processing

(1) Full history taking, general and neurological examination, GCS and full laboratory tests.
(2) Radiology (computed tomography of the brain without contrast). Hematoma thickness, midline shift, associated brain edema, and other cranial pathologies.
(3) Operative findings (if present).
(4) Presence of secondary insults.
(5) Glasgow Outcome Scale (GOS).

The GOS categorizes the outcomes of patients after trauma or surgery:

(1) Death
(2) Persistent vegetative state: minimal responsiveness.
(3) Severe disability: conscious but disabled; dependent on others for daily support.
(4) Moderate disability: disabled but independent; can work in sheltered setting.
(5) Good recovery: resumption of normal life despite minor deficits.

Statistical analysis

Data were analyzed using IBM SPSS Advanced Statistics, version 21 (SPSS Inc., Chicago, Illinois, USA). Numerical data were expressed as mean and SD or median and range as appropriate. Pearson's χ^2 tests were used to compare the incidence of postoperative complications.

Qualitative data were expressed as frequency and percentage. χ^2 test was used to examine the relation between qualitative variables. For not normally distributed quantitative data, comparison between two groups was done using the Mann–Whitney test (nonparametric *t* test). Comparison between quantitative repeated measures was done using analysis of variance with repeated measures. Wilcoxon signed-rank test (nonparametric paired *t* test) was used to compare two consecutive measures of numerical variables. A *P* value less than 0.05 was considered significant.

Indeed, we used the logistic regression analysis to identify covariates that were independently associated with death. We included in the model those covariates that were found to be significant in the correlation analysis

Results

Demographic distribution

The mean and SD of age in our study was 46.3±20.6 years. The range of age was 64 years (21–85 years). There was no statistical difference between sexes as regards age (P=0.393). The distribution of male. In our study, there were 19 (63.3%) men while women were 11 (36.7%).

Mode of trauma

Eighty-three percent (25 patients) of cases were injured due to road traffic accidents while in only five (16.7%) cases the cause of trauma was isolated head injury. In 23 cases, 18 (60%) patients were drivers or passengers while seven (23.3%) were pedestrians.

Polytrauma versus isolated head trauma

Forty percent of our cases have no comorbid fractures or hematoma. Table 1 shows that the majority of cases have other types of trauma.

In our study, there were many comorbid fractures and injuries as illustrated in Table 1. Lung contusion was the most common coincidental injury seen in our study (six, 17.4%). Table 2 shows in detail the cranial, spinal, and extracranial lesions. It has been found that lumbar spine fractures are more prevalent than cervical trauma. Brain contusion was found to be the more prevalent cranial lesion (11.7%) than any other lesions.

Secondary insults

In 17 (56.7%) patients, hypoxia was recorded. Hypotension was recorded in 11 (36.7%) cases. In general, a low conscious level status was recorded to be correlated with the presence of one factor at least (P=0.018 for hypoxia, P=0.042 for hypotension and presence of either risk P=0.042) and presence of two factors (P=0.006) (Table 3).

Incidence of surgical cases

In our study, conservative treatment was carried out in 19 (63.3%) patients and surgical treatment in 11 (36.6%) patients (Fig. 1).

Correlation study

All study variables were enrolled in a correlation study versus mortality to identify the mathematical relationship with the important variable (mortality).

Table 1 Frequency of comorbidities

Number of comorbidities in a single patient	Frequency	Percent
0	12	40.0
1	8	26.7
2	5	16.7
3	3	10.0
4	2	6.7
Total	30	100.0

Table 2 List of cranial, spinal, and extracranial lesions

	Item	*n* (%)
Cranial lesions	Brain contusion	4 (11.7)
	Temporal lobe contusion	1 (2.9)
	ICH	2 (5.88)
	Subarachnoid hemorrhage	2 (5.88)
	Skull base fracture	1 (2.9)
	Pneumocephalus	1 (2.9)
Spinal trauma	Cervical spine fracture	1 (2.9)
	Lumbar fracture	2 (5.88)
Extracranial extraspinal lesion	Femoral fracture	2 (5.88)
	Hemothorax	5 (14.66)
	Lung contusion	6 (17.4)
	Pneumothorax	3 (8.7)
	Both lower limb fracture	1 (2.9)
	Humeral fracture	2 (5.88)
	Rib cage fracture	1 (2.9)
	Total	34[a]

ICH, intracranial hemorrhage. [a]t his does not reflect the total number of patients, instead it reflects the total number of injuries.

Table 3 Hypoxia and hypotension in correlation with Glasgow coma scale

	n (%)	*P* value	Significance
GCS below 11			
Hypoxia			
No	13 (43.3)	–	
Yes	17 (56.7)	0.018	S
Hypotension			
No	19 (63.3)	–	
Yes	11 (36.7)	0.042	S
Number of risk factor(s)[a]			
No	13 (43.3)	–	
Single risk	6 (20)	0.042	S
Double risk factor	11 (36.7)	0.006	HS

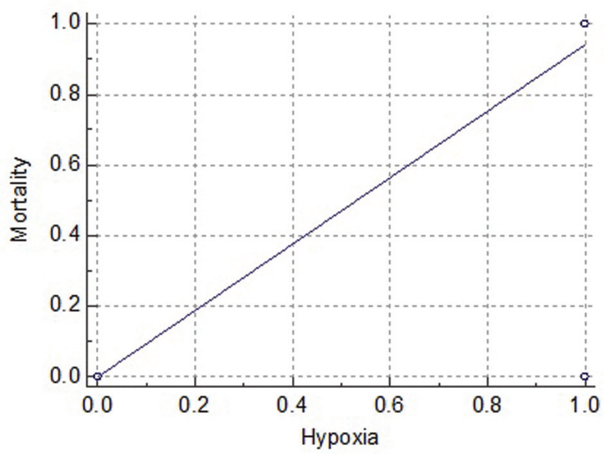
[a]irs factor refers to hypoxia and hypotension. GCS, Glasgow coma scale; HS, highly significant; S, significant.

It has been found that severity of injury, presence of a secondary injury especially hypoxia or both hypoxia and hypotension, polytrauma, status of conscious level at admission, anemia, thickness of hematoma, and necessity for endotracheal intubation were highly associated with mortality ($P<0.05$). Midline shift was highly correlated with the size of hematoma (<0.05) but no other factors like severity of injury, GCS at admission, presence of other risk factors like hypoxia or hypotension, and presence of contusions were found to be correlated with the size of hematoma or even midline shift ($P>0.05$) (Table 4).

Regression analysis

When the entire study population was subjected to logistic regression analysis, severity of injury, presence of a secondary injury especially hypoxia or both hypoxia and hypotension, polytrauma, status of conscious level at admission, anemia, thickness of hematoma and necessity for endotracheal intubation were enrolled. Hypoxia and low GCS at admission were found to be significant predictors of mortality (Table 5). Figures 2 and 3 show the scattered plot and regression lines of hypoxia, hypotension, and mortality

Figure 1



Pie chart of patients who underwent surgery.

Discussion

ASDH occurred in about 30% of brain injuries due to severe head trauma [10]. The rate of mortality of ASDH was reported to be about 60% until the 1990s. The rate decreased to the level of 20% around 2000 and was as low as 14% within the last 10 years [11]. Although on the other side the rate of functional recovery was about 20–45% in traumatic ASDH [4]. Although the indication for surgery has not been clear in traumatic ASDH, decompressive craniectomy and craniotomy were done for management purposes to induce relaxation of the brain [12].

In our study, all the patients were operated after admission. Hematoma was evacuated with decompressive craniectomy or craniotomy, depending on the clinical condition, hematoma thickness, parenchymal edema of the patient, and other factors. In patients with lower GCS severe edema as additional pathology, decompressive craniectomy was the better choice of management. The aim of decompressive craniectomy was to provide enough space for brain relaxation and to prevent herniation [10].

In our study, the mean and SD of age was 46±20 years. The range of age was 64 years (21–85 years), 63.3% of our cases were men. Studies from the United States of America and European countries determined that

Table 4 Correlation study of variables versus mortality

	Mortality
Age	
Pearson's correlation	–0.105
Significance (two-tailed)	0.582
Sex	
Pearson's correlation	–0.120
Significance (two-tailed)	0.527
Injury severity	
Pearson's correlation	0.767
Significance (two-tailed)	**0.000**
Risk factor	
Pearson's correlation	0.904
Significance (two-tailed)	**0.000**
Hypoxia	
Pearson's correlation	0.935
Significance (two-tailed)	**0.000**
Hypotension	
Pearson's correlation	0.712
Significance (two-tailed)	**0.000**
Single risk	
Pearson's correlation	0.301
Significance (two-tailed)	0.106
Double risk	
Pearson's correlation	0.712
Significance (two-tailed)	**0.000**
Mode of trauma	
Pearson's correlation	0.170
Significance (two-tailed)	0.370
Number of comorbidities	
Pearson's correlation	0.449
Significance (two-tailed)	**0.013**
GCS at admission	
Pearson's correlation	–0.735
Significance (two-tailed)	**0.000**
Time to diagnose	
Pearson's correlation	–0.213
Significance (two-tailed)	0.257
INR	
Pearson's correlation	–0.202
Significance (two-tailed)	0.284
Hg	
Pearson's correlation	–0.498
Significance (two-tailed)	**0.006**
Hematoma thickness	
Pearson's correlation	0.444
Significance (two-tailed)	**0.014**
Midline shift	
Pearson's correlation	0.357
Significance (two-tailed)	0.053
RTA	
Pearson's correlation	–0.218
Significance (two-tailed)	0.247
GCS below 8	
Pearson's correlation	0.668
Significance (two-tailed)	**0.000**
Necessity for ET	
Pearson's correlation	0.668
Significance (two-tailed)	**0.000**
Surgery	
Pearson's correlation	0.157
Significance (two-tailed)	0.407

GCS, Glasgow coma scale; INR, International normalized ration; RTA, road traffic accident. The bold values are the results means that factor is significant.

Table 5 Predictors of mortality of acute subdural hematoma by multivariate logistic regression analysis

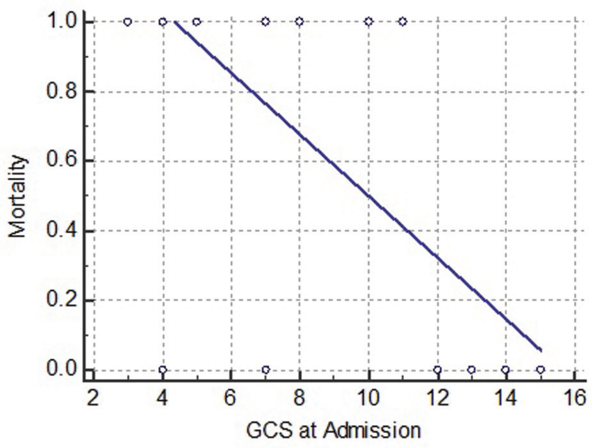
Models	Odd ratio	*P* value
Hypoxia	0.801	0.000
GCS at admission	–0.201	0.026

GCS, Glasgow coma scale.

traumatic ASDH has an important role on the mortality under the age of 45 years [13]. Ryan *et al*. [12] reported that 63% of traumatic ASDH patients were men and the mean age of the study population was 58 years. Yanagawa and Sakamoto [14] reported that 67% of the patients of traumatic ASDH were men and the mean age was 43 years. Shen *et al*. [15] reported that most of the affected patients were men and the patient population had a mean age of 36 years. Okten *et al*. [16] reported that traumatic ASDH patient population had a mean age of 39 years and they noticed that 76% of the patients were men . Our patient population's mean age and male predominance were comparable to other studies. It is clear in our study that an increased percentage of driving and occupational accidents, as well as a higher rate of incidence among adults, are the factors that formed the mean age of our population. Men are exposed more to trauma since they are spending a lot of time out of their houses and are more commonly included in risky jobs such as working in high places (building sites, high-voltage transmission lines, . . . , etc.) and driving vehicles compared with women.

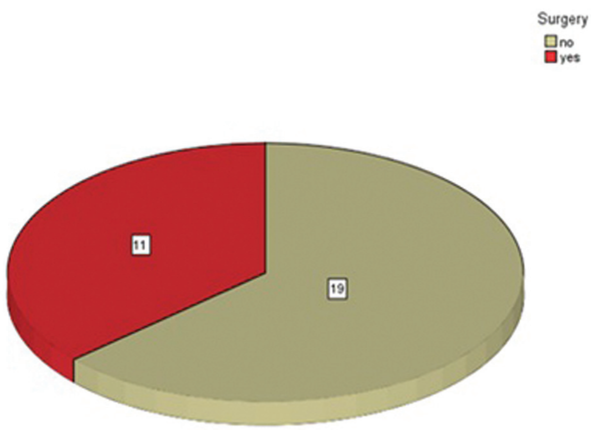
Ryan *et al*. [12], noted that the most common cause of traumatic ASDH was falling from height (FFH), followed by motor traffic accidents. Leitgeb *et al*. [7] correspondingly reported that falls and motor traffic accidents were the most common principles of traumatic ASDH. Yanagawa and Sakamoto [14] reported that ASDH always resulted from motor traffic accidents. It was also reported that falling from a height was the most common cause of ASDH, followed by motor traffic accidents. In other studies, motor traffic accidents were followed by falling from height and thought to be the most common causes of ASDH [14]. In another study from Turkey, Kalayci *et al*. [10], reported similar results for the causes of ASDH. Comparing our study to

Figure 2



Scattered plot and regression line of hypoxia in multivariate analysis.

Figure 3



Scattered plot and regression line of lower GCS at admission in multivariate analysis. GCS, Glasgow coma scale.

other studies, we found that road traffic accident (RTA) was the most common cause of ASDH. The cause of this discrepancy may be the geographical location of the medical centers included in our study as it is close to suburban areas.

In many studies reported that midline shift due to ASDH is related to low GCS and mortality [17]. Chen *et al.* [3], noted that the mean diameter of acute subdural hemorrhage was 11 mm and the measurement is parallel to the degree of midline shift. In another study performed on 21 patients by Yanagawa and Sakamoto [14], a mean GCS of 4.8 was reported and the size of ASDH was 13 mm. Son *et al.* [18] noted that a mean hematoma size of 6 mm and the size was parallel to the amount of shift. Yanaka *et al.* [5], reported that the size of hemorrhage and the amount of shift was large in patients who died compared with those who did not. We thought that the increased amount of hematoma is a direct parameter of injured parenchyma but it also results in secondary losses by inducing more edema due to an increase in the space that it occupies. Thus, we promote that the size of ASDH is proportional to the clinical course.

GCS is a clinical method depending on three components (motor, verbal, eye), each of them determine separate areas of the brain. Therefore, it has been reported that it is the most important factor that directly reflects brain damage, reflects clinical status, and gives information on prognosis during the follow-up [19]. Son and colleagues noted a mean admission GCS score of 13. It was reported in another study that most of the patients had a GCS of more than 13, followed by patients with a GCS lesser than 6 and surgeries were most commonly performed on patients with a low GCS [18]. Most of the patients with ASDH had a GCS lower than 8 in general [17,20]. Also patients with additional pathologies had a lower GCS [21]. Ryan and colleagues reported that GCS increased after follow-up and surgery. It was also reported that the patients who survived generally improved later [12].

The mortality rate of traumatic ASDH is ranging from 40 to 60% [4,6,22]. Different factors, including age, have been noted to determine mortality in traumatic and nontraumatic ASDH patients [11,21]. Busl and Prabhakaran [23], reported a mortality rate of 12% in patients with ASDH and found that the rate was lower in traumatic ASDH patients. Ryan *et al.* [12], reported a mortality rate of 14%. In the study of Leitgeb *et al.* [7], the mortality rate was found to be 47%, additional trauma and mortality were found to be increased as the GCS was lowered. Previous studies have reported that the lower GCS was strongly correlated to mortality [10,21,24]. Shen *et al.* [15] reported that the mortality rate was higher in patients with a lower GCS. Leitgeb *et al.* [7] demonstrated a higher mortality rate in patients with other traumatic brain lesions. Kalayci *et al.* [10] noted a lower mortality rate in patients operated earlier.

We noted that the higher mortality rate in motor traffic accidents may be due to the severity of the trauma. As expected, patients with additional traumatic lesions have a higher mortality rate depending on the increased amount of affected brain tissue. In our study, the mortality increased with the preadmission low GCS and presence of hypoxia. This was possibly because of decreased physiological reserve of the brain, decreased brain plasticity, and increased rates of comorbidities in that patient population. An increased amount of hematoma will lead to more brain tissue to become affected and the GCS to be lowered. In our study, it has been reported that International normalized ration (INR) was markedly and statistically significantly higher in those with GOS 1 and 2 other than GOS 4 and 5. Indeed, raised INR was significantly associated with extracranial injuries than those with only intracranial injuries, the difference was statistically significant (P=0.045).

In a large series of patients with ASDH, Bershad and colleagues found that only APACHE III scores and coagulopathy were independently associated with increased hospital deaths. The time until correction of coagulopathy using FFP and/or vitamin K was prolonged [21].

In our study, hemoglobin level was much lower in polytraumatized patients than those with isolated head injury. In contrast, Genét and colleagues found that the hemoglobin level was not different between those two groups.

In our study conservative treatment was carried out in 19 (63.3%) patients and surgical treatment in 11 (36.6%) patients, Yanaka *et al.* [5], reported conservative treatment in 93 (54.7%) patients and surgical treatment in 77 (45.3%) patients. In another study Ryan *et al.* [12] reported that of the 1427 patients, 248 (17%) underwent surgical intervention for subdural evacuation and 1179 (83%) had no surgical evacuation.

Limitations

The limitations of our study were the small sample size and long-term follow-up study was not conducted in methodology.

Conclusion

Traumatic ASDH still has a high mortality rate despite all developments in neurosurgical interventions. Initially low GCS and hypoxia are important parameters that correlate with the mortality rate.

Acknowledgements

This study was supported by the Neurosurgery Department, Al-Azhar University Hospitals.

Financial support and sponsorship

Nil.

Conflicts of interest

There was no conflict of interest.

References

1 Alagoz F, Yildirim AE, Sahinoglu M, Korkmaz M, Secer M, Celik H, *et al.* Traumatic acute subdural hematomas: analysis of outcomes and predictive factors at a single center. *Turk Neurosurg* 2017; **27**:187–191.

2 Braun J, Borovich B, Guilburd JN, Zaaroor M, Feinsod M, Grushkiewicz I. Acute subdural hematoma mimicking epidural hematoma on CT. *Am J Neuroradiol* 1987; **8**:171–173

3 Chen S-H., Chen Y, Fang W-K, Huang D-W, Huang K-C, Tseng S-H. Comparison of craniotomy and decompressive craniectomy in severely head-injured patients with acute subdural hematoma. *J Trauma* 2011; **71**:1632–1636.

4 D'Amato L, Piazza O, Alliata L, Sabia G, Zito G, Frassanito L, *et al.* Prognosis of isolated acute post-traumatic subdural haematoma. *J Neurosurg Sci* 2007; **51**:107–111.

5 Yanaka K, Kamezaki T, Yamada T, Takano S, Meguro K, Nose T. Acute subdural hematoma – prediction of outcome with a linear discriminant function. *Neurol Med Chir (Tokyo)* 1993; **33**:552–558.

6 Kim KH. Predictors for functional recovery and mortality of surgically treated traumatic acute subdural hematomas in 256 patients. *J Korean Neurosurg Soc* 2009; **45**:143–150.

7 Leitgeb J, Mauritz W, Brazinova A, Janciak I, Majdan M, Wilbacher I, Rusnak M. Outcome after severe brain trauma due to acute subdural hematoma. *J Neurosurg* 2012; **117**:324–333.

8 Li LM, Kolias AG, Guilfoyle MR, Timofeev I, Corteen EA, Pickard JD, *et al.* Outcome following evacuation of acute subdural haematomas: a comparison of craniotomy with decompressive craniectomy. *Acta Neurochir (Wien)* 2012; **154**:1555–1561.

9 McHugh GS, Engel DC, Butcher I, Steyerberg EW, Lu J, Mushkudiani N, *et al.* Prognostic value of secondary insults in traumatic brain injury: results from the impact study. *J Neurotrauma* 2007; **24**:287–293.

10 Kalayci M, Aktunç E, Gül S, Hanci V, Edebali N, Çagavi F, Açikgöz B. Decompressive craniectomy for acute subdural haematoma: an overview of current prognostic factors and a discussion about some novel prognostic parametres. *J Pak Med Assoc* 2013; **63**:38–49.

11 Rao MG, Singh D, Khandelwal N, Sharma SK. Dating of early subdural haematoma: a correlative clinico-radiological study. *J Clin Diagnostic Res* 2016; **10**:HC01–HC05.

12 Ryan CG, Thompson RE, Temkin NR, Crane PK, Ellenbogen RG, Elmore JG. Acute traumatic subdural hematoma. *J Trauma Acute Care Surg* 2012; **73**:1348–1354.

13 Westermaier T, Eriskat J. Clinical features, treatment, and prognosis of patients with acute subdural hematomas presenting in critical condition. *Neurosurgery* 2007; **61**:482–488.

14 Yanagawa Y, Sakamoto T. Results of single burr hole drainage for acute subdural hematoma with non-reactive pupil. *Turk Neurosurg* 2012; **22**:196–199.

15 Shen J, Pan JW, Fan ZX, Zhou YQ, Chen Z, Zhan RY. Surgery for contralateral acute epidural hematoma following acute subdural hematoma evacuation: five new cases and a short literature review. *Acta Neurochir (Wien)* 2013; **155**:335–341.

16 Okten AI, Gezercan Y, Ergün R. Traumatic subarachnoid hemorrhage: a prospective study of 58 cases. *Ulus Travma Acil Cerrahi Derg* 2006; **12**:107–114.

17 Faleiro RM, Faleiro LCM, Caetano E, Gomide I, Pita C, Coelho G, *et al.* Decompressive craniotomy: Prognostic factors and complications in 89 patients. *Arq Neuropsiquiatr* 2008; **66**:369–373.

18 Son S, Yoo CJ, Lee SG, Kim EY, Park CW, Kim WK. Natural course of initially non-operated cases of acute subdural hematoma: the risk factors of hematoma progression. *J Korean Neurosurg Soc* 2013; **54**:211–219.

19 Patel P. Association of subdural hematoma with increased mortality in lobar intrcerebral hemorrhage. *Arch Neurol* 2011; **66**:79–84.

20 Toussaint CP, Origitano TC. Decompressive craniectomy: review of indication, outcome, and implication. *Neurosurg Q* 2008; **18**:45–53.

21 Genét GF, Johansson PI, Meyer MAS, Sølbeck S, Sørensen AM, Larsen CF, *et al.* Trauma-induced coagulopathy: standard coagulation tests, biomarkers of coagulopathy, and endothelial damage in patients with traumatic brain injury. *J Neurotrauma* 2013; **30**:301–306.

22 Cai Y, Zhong X, Wang Y, Yang J, Zheng H. Acute subdural hematoma with swirl signs: clinical analysis of 15 cases. *Chin J Traumatol* 2010; **13**:253–254.

23 Busl KM, Prabhakaran S. Predictors of mortality in nontraumatic subdural hematoma. *J Neurosurg* 2013; **119**:1296–1301.

24 Inamasu J, Hasegawa M, Hayashi T, Kato Y, Hirose Y. Hemispheric differences in the surgical outcomes of patients with traumatic acute subdural hematoma. *J Negat Results Biomed* 2014; **13**:1–8.