Original Article

Radiological Parameters to Predict Hemorrhagic Progression of Traumatic Contusional Brain Injury

Lal Rehman, Ali Afzal, Hafiza Fatima Aziz, Sana Akbar, Asad Abbas, Raza Rizvi

Department of Neurosurgery, Jinnah Postgraduate Medical Centre, Karachi, Pakistan

Abstract

Introduction: Traumatic intracerebral contusion is a frequent factor culminating in death and disability, and its progression relates to unfavorable outcome. We evaluated the radiological factors associated with hemorrhagic progression of contusions (HPC). **Materials and Methods:** Two hundred and forty-six patients were enrolled in this prospective cohort over a period of 1 year. Contusion volume was quantified using the "ABC/2" technique, whereas progression was considered as >30% increase in the initial volume. Univariate and multivariate statistics were used to examine the correlation between the risk factors of interest and HPC. **Results:** HPC was seen in 110 (44.7%) patients. Binary logistic regression showed in the final adjusted model that multiplicity (relative risk [RR]: 2.24, 95% confidence limit [CL]: 1.00–5.48), bilateral lesions (RR: 2.99, 95% CL: 1.08–8.25), initial volume of contusion (RR: 4.96, 95% CL: 1.87–13.13), frontal location (RR: 1.42, 95% CL: 1.08–3.56), and presence of concomitant intracranial hematoma (extradural-RR: 3.90, 95% CL: 1.51–10.01, subdural-RR: 2.91, 95% CL: 1.26–6.69, and subarachnoid-RR: 2.27, 95% CL: 1.01–5.80) were significantly associated with HPC. The overall mortality was 18.7% and was almost equal among patients with and without HPC. Mortality was significantly associated with Glasgow Coma Scale on admission (adjusted RR: 12.386, 95% CL: 4.789–32.035) and presence of comorbid conditions (adjusted RR: 0.313, 95% CL: 0.114–0.860). **Conclusion:** Initial computed tomography scan is a good predictor of high-risk group for HPC.

Keywords: *Brain contusion, intracerebral hematoma, intraparenchymal hematoma, progressive hemorrhagic injury, traumatic brain injury*

Introduction

Traumatic intraparenchymal hematomas (tIPH) is a common sequel of primary traumatic brain injury (TBI) occurring in up to 35% of severe TBI.[1] Primary TBI continues damaging brain via several secondary injuries, with "hemorrhagic progression" being one of these devastating entities.[2] Progression of tIPH has been evaluated in several studies with an incidence of around 16%–63%.[3-5] Contusion is a subtype of tIPH with ill-defined areas of mixed density attenuation as salt-and-pepper appearance on computed tomography (CT) scans.[6] It tends to progress more as compared to other "solid appearing" tIPH.[7]

Contusion is a frequent factor culminating in death and disability of TBI victims, and its progression relates to unfavorable outcomes.[8-10] Progression of contusion, first evident in 1979, is being termed differently throughout the literature, but to avoid this ambiguity, Kurland *et al*. adopted the term "hemorrhagic progression of contusion" (HPC).[11,12] HPC has been found to be associated with several clinical (age, injury severity, Glasgow Coma Scale [GCS] score, blood alcohol level anticoagulation/antiplatelet use, platelet count, international normalized ratio, and platelet transfusion) and radiological (initial volume, location, ventriculostomy status, skull fracture, concomitant

Access this article online

Quick Response Code:

Website: www.ruralneuropractice.com

DOI: 10.4103/jnrp.jnrp_335_18

Address for correspondence: Dr. Asad Abbas, Department of Neurosurgery, Jinnah Post Graduate Medical Center, Rafiqui Shaheed Road, Karachi 75510, Pakistan. E-mail: asad.abbas@live.com

How to cite this article: Rehman L, Afzal A, Aziz HF, Akbar S, Abbas A, Rizvi R. Radiological parameters to predict hemorrhagic progression of traumatic contusional brain injury. J Neurosci Rural Pract 2019;10:212-7.

subdural and subarachnoid hematoma, and duration from injury to initial CT scan) parameters.[8,13-15] However, consensus is lacking in literature. As the disease has a short natural course and deterioration occurs within hours of injury, it is crucial to identify the patients at risk of progression.[16]

CT scan is the diagnostic modality of choice for moderate-to-severe TBI. Radiological findings along with clinical assessment help neurosurgeons categorize the patients according to the level of care needed. Here, we intend to evaluate the radiological factors that favor the progression of traumatic contusion. Few studies have been done for this purpose; however, majority of them are either done on small sample size or are retrospective studies with their innate limitations.

Materials and Methods

Prospectively collected data after consecutive sampling of study population during a period of 1 year from January to December 2017 were used to construct the cohort study at the Department of Neurosurgery, Jinnah Post Graduate Medical Center (JPMC), Karachi, Pakistan. The Institutional Review Board at JPMC, Karachi, approved this research project. All patients >14 years of age with a history of blunt TBI within 24 h and initial CT scan showing contusion as a primary lesion were included in the study. All patients with polytrauma, with concomitant other intracerebral hematoma as a primary lesion who underwent evacuation and those who expired before repeat CT scan were excluded from the study.

All CT scans were evaluated by two independent senior on-call neurosurgeons and a consultant radiologist for contusion and all associated intracranial injuries. Volume of contusion is calculated by *ABC*/2 technique (where *A* = maximum diameter in cm, *B* = diameter at 90° to maximum diameter in cm, and *C* = total number of 1-cm axial slices).[17] Volume of multiple contusion was calculated separately by *ABC*/2 method and then adding them together to get the total volume of contusion.[9] Contusions were divided on the basis of initial volume into small (<20 ml) or large (>20 ml), as lesions >20 ml with signs of mass effect are indicated to be managed operatively.[18] HPC is defined as >30% increase from the initial volume on repeat CT scan.[9,14] The primary outcome of the study was designated as HPC that is hypothesized to be associated with radiological parameters such as multiplicity, location, laterality, initial volume contusion, associated intracranial injuries, and presenting GCS.

Statistical analysis

Data were analyzed using SPSS version 20 for IBM Corp. Released 2011. IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp. MS Windows. Means and standard deviations were calculated for continuous variables, whereas frequencies and percentages were calculated for categorical variables. Chi-square test was employed to observe any statistically significant difference in the distribution of independent variables according to progression. Crude and adjusted relative risks of progression were calculated for independent variables. $P < 0.05$ was considered statistically significant.

Table 1: Characteristics of the study population

Characteristics	Frequency (%)
Gender	
Male	212 (86.1)
Female	34 (13.8)
Mode of injury	
RTA	212 (86.2)
Fall	32 (13)
Assault	2 (0.8)
Number of contusions	
Single	138 (56.1)
Multiple	108 (43.9)
Initial volume of contusion (ml)	
<20	200 (81.3)
>20	46 (18.7)
Progression	
No	136 (55.3)
Yes	110 (44.7)
Location of contusion	
Frontal	92 (37.4)
Temporal	52 (21.1)
Parietal	38 (15.4)
Occipital	4 (1.6)
More than one	60 (24.4)
Laterality	
Unilateral	208 (84.6)
Bilateral	38 (15.4)
Associated intracranial injury	
EDH	48 (19.5)
SAH	52 (21.1)
SDH	48 (19.5)
Fracture	64 (26.0)
GCS score on admission	
>9	164 (66.7)
<8	82 (33.3)
Comorbidities	
Yes	42 (17.1)
No	204 (82.9)
Mortality	
Yes	46 (18.7)
No	200 (81.3)
Length of stay (mean)	4.61±3.44

RTA: Road traffic accident, EDH: Extradural hematoma, SAH: Subarachnoid hemorrhage, SDH: Subdural hematoma, GCS: Glasgow Coma Score

Results

Among 246 patients, 212 (86%) were males, with a mean age of 40.38 ± 19.58 years, whereas the remaining were females with a mean age of 34.29 ± 15.38 years. Road traffic accident (RTA) was the most common mode of injury occurring in 212 patients (86.2%), followed by fall in 32 patients (13%). A total of 108 (44%) patients suffered from multiple contusions, whereas the most common region involved was frontal in 92 patients (37.4%) [Table 1]. Detailed distribution of regional involvement is shown in Figure 1.

The GCS score on admission was ≥9 in 66.7% (n = 164) of the patients. Majority of the patients, that is, 81.3% (n = 200) had volume of contusion <20 ml, while the remaining 18.7% (n = 46) had volume >20 ml. HPC was seen in 110 (44.7%) patients. Binary logistic regression in the final adjusted model showed that the risk of HPC was 1.5 times more in patients with frontal contusion. Similarly, patients with bilateral contusion were at three times greater risk of progression compared to those who had unilateral contusion. Multiple contusions were three times more prone to develop progression as compared to single contusion. Volume of contusion >20 ml was associated with five times greater risk of progression while the presence of concomitant intracranial hematoma significantly increases the risk of progression. Risk of progression of is increased fourfold with extradural hematoma (EDH), whereas subdural hematoma (SDH) and subarachnoid hematoma increase the risk by three times and two times, respectively.

Mean length of stay was 4.6 days, with a difference of 1.4 days in both groups (5.43 in HPC vs. 3.97 with P = 0.02). The overall mortality rate was 18.7% (n = 46) and was almost equal among patients with and without progression (8.94% vs. 9.75%). There was no statistically significant difference in the mortality rate of patients with and without progression (P = 0.37). Tables 2 and 3 show the risk factors associated with hemorrhagic progression and mortality, respectively. Mortality was significantly associated with GCS scores on admission and presence of comorbid conditions. Patients with GCS <8 were at 12 times higher risk of dying compared to those with better GCS scores on admission. Similarly, patients with comorbid conditions were at three times greater risk of death compared to those patients with no comorbid conditions.

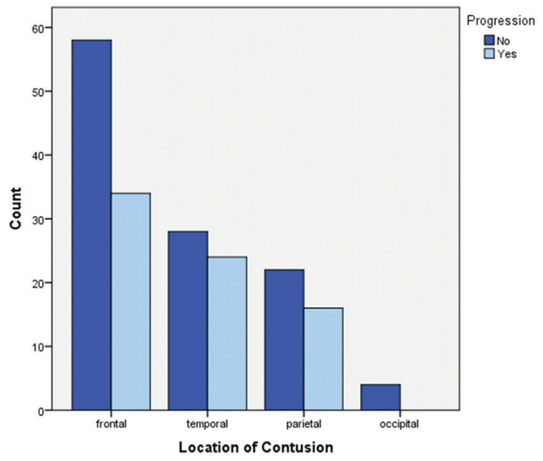

Figure 1: Distribution of location of contusion and its progression on repeat computed tomography scan

Discussion

Profound understanding of the contributing factors that leads to progression could be the major step in the better management of TBI, especially in facilities where cost of management outweighs the unsorted placement of all patients to high-dependency and intensive care units and frequent repeat CT scans. Hematological and biochemical parameters are less practical to use as they are time consuming and due to their cost in resource-limited setups. All patients with suspected TBI undergo CT scan as the first choice of investigation; hence, we assessed the radiological factors in order to highlight the group of patients at risk for HPC.

Lack of standardization of the term "progression" and a uniform cutoff value for initial contusion identified on the first CT scan led to high variability in the incidence of hemorrhagic progression from 16% to 74%, as reported in literature.[4,13,16,19]

However, recent studies using progression as >30% increase in size report incidence between 45% and 65%,[9,10,14,20] which is comparable to our result (44.7%). RTA is the major cause of injury in our study; similarly, Hilmer *et al*. recently reported RTA, especially motor bike accidents, as the major cause of traumatic contusion.[21]

Prior studies associate a statistically significant relation between older age and HPC;[3,13,14] however, our univariate analysis shows a borderline association of age and HPC, but our multivariate analysis failed to predict a positive relation between them. This difference could be due to higher mean age in other studies.[5,13,14] A number of studies show result parallel to our study.[8,9] However, age is a proven predictor of outcome.[10,20]

Oertel *et al*. hypothesized that female gender has a neuroprotective effect of estrogen and progesterone against HPC and showed a significant correlation between male sex and HPC.[22] Here, we relate our findings to them that, on univariate analysis, it was

Table 2: Risk factors associated with hemorrhagic progression among patients with contusion (n=246)

	Progression, *n* (%)		*P**	Crude RR (95% CI)	Adjusted RR (95% CI)**
	No progression	Progression			
Age (years)					
<50	92 (59)	64 (41)	0.08	-	-
≥50	44 (48.9)	46 (51)		1.50 (0.89-2.53)	0.86 (0.40-1.87)
Gender					
Male	112 (52.8)	100 (47.2)	0.039	-	-
Female	24 (70.6)	10 (29.4)		2.14 (0.98-4.70)	0.74 (0.25-2.21)
Location of contusion					
Parietal	22 (57.9)	16 (42.1)	0.023	-	-
Frontal	58 (63.0)	34 (37.0)		1.75 (1.11-2.74)	1.42 (1.08-3.56)
Temporal	28 (53.8)	24 (46.2)		1.46 (0.73-2.91)	0.90 (0.37-2.22)
Occipital	4 (100.0)	0 (0.0)		***	***
Laterality					
Unilateral	126 (60.6)	82 (39.4)	<0.001	-	-
Bilateral	10 (26.3)	28 (73.7)		4.30 (1.98-9.32)	2.99 (1.08-8.25)
Volume of contusion (ml)					
<20	124 (62.0)	76 (38.0)	<0.001	-	-
>20	12 (26.1)	34 (73.9)		4.62 (2.25-9.47)	4.96 (1.87-13.13)
Number of contusions					
Single	80 (58.0)	58 (42.0)	0.007	-	-
Multiple	56 (51.9)	52 (48.1)		2.94 (1.20-4.45)	3.02 (1.35-6.78)
GCS score on admission					
>9	96 (58.5)	68 (41.5)	0.094	-	-
<8	40 (48.8)	42 (51.2)		1.48 (0.87-2.52)	0.671 (0.32-1.40)
EDH					
No	114 (57.6)	84 (42.4)	0.096	-	-
Yes	22 (45.8)	26 (54.2)		1.60 (1.04-2.55)	3.90 (1.51-10.01)
SDH					
No	122 (61.6)	76 (38.4)	<0.001	-	-
Yes	14 (29.2)	34 (70.8)		3.89 (1.96-7.73)	2.91 (1.26-6.69)
SAH					
No	122 (62.9)	72 (37.1)	<0.001	-	-
Yes	14 (26.9)	38 (73.1)		4.59 (2.33-9.06)	2.27 (1.01-5.80)
Fracture					
Yes	38 (59.4)	26 (40.6)	0.269	-	-
No	98 (53.8)	84 (46.2)		1.25 (0.70-2.23)	0.909 (0.41-2.00)
Isolated head injury					
Yes	108 (55.1)	88 (44.9)	0.519	-	-
No	28 (56.0)	22 (44.0)		0.96 (0.51-1.80)	1.422 (0.64-3.16)
Comorbidity					
Yes	20 (47.6)	22 (52.4)	0.177	-	-
No	116 (56.9)	88 (43.1)		0.69 (0.35-1.34)	1.156 (0.47-2.81)
Length of stay					
Mean±SD	3.97±2.68	5.43±4.08	0.02	-	-
Mean difference (CI)		1.45 (0.59-2.3)		-	-

**P* value is calculated through Chi-square test, **Adjusted RRs are calculated after adjusting for all the variables in the model, ***RR cannot be calculated due to 0% in progression group. CI: Confidence interval, RRs: Relative risks, SD: Standard deviation, GCS: Glasgow Coma Scale, EDH: Extradural hematoma, SAH: Subarachnoid hemorrhage, SDH: Subdural hematoma

found that male has a significant risk of HPC, but these results are not significant on final adjusted model. However, relatively fewer females (13.8%) in the study sample could interfere this association. Number of studies shows no significant association.[9,13,23]

Frontal contusions are known to progress over a period of time, and extensive bifrontal contusion show syndrome of delayed deterioration.[24] Allison *et al*. reported that HPC was associated with frontal location of contusion.[14] Our data conclude the same results where frontal contusion

Table 3: Factors associated with mortality in patients with contusion (*n*=246)

	Mortality, *n* (%)		*P**	Crude RR (95% CI)	Adjusted RR (95% CI)**
	Alive	Died			
GCS score on admission					
>9	150 (91.5)	14 (8.5)	<0.001	-	-
<8	50 (61.0)	32 (39.0)		6.85 (3.38-13.87)	12.38 (4.789-32.035)
Management					
Conservative	148 (81.3)	34 (18.7)	0.562	-	-
Surgery	52 (81.2)	12 (18.8)		1.01 (0.48-2.08)	0.424 (0.121-1.478)
Comorbid					
Yes	30 (71.4)	12 (28.6)	0.061	-	-
No	170 (83.3)	34 (16.7)		0.50 (0.23-1.07)	0.313 (0.114-0.860)

**P* value is calculated through Chi-square test, **Adjusted RRs are calculated after adjusting for all the variables in the model, CI: Confidence interval, RRs: Relative risks, GCS: Glasgow Coma Scale

constitutes the majority of contusions and is more likely to progress as compared to other locations. Four patients had occipital contusion, and none of these lesions progress.

Initial volume of contusion is a well-identified independent parameter for progression, positively associated with HPC; larger contusions tend to progress.[8-10] Carnevale *et al*. concluded that initial volume of contusion is the most predictive factor of progression among all the studied variables and the rate of progression correlates linearly with it.[13,23] Some analysts found it to be a prognostically relevant factor.[8] Although smaller lesions tend to progress, this does not have an impact on clinical outcome[25] and unlikely to require surgery.[26] In our patients, initial volume of contusion >20 ml was associated with fivefold increased risk of HPC in the final analysis and is the most important predictive factor among all variables.

Patients with bilateral lesions had three times more risk of developing HPC compared to unilateral lesions. This observation is probably associated with the severity of lesion and additive volume of bilateral contusion as initial volume shows a strong association with progression. This variable is not included in previous literatures of contusion. Multiplicity of location of contusion is independently associated with HPC in the final model, similar to other studies[9] where multiplicity of contusion is regarded as a risk factor as additive volume of multiple contusion is higher.[20]

Lower GCS is significantly associated with HPC,[9,13] clinical deterioration,[16,20] delayed surgical intervention,[7,20] and poor outcome as probability of going home.[10,13] Contrary to this, a number of studies failed to prove this association.[3,7,23] Similarly, we conclude that GCS on admission is not associated with HPC.

Previous literature has highlighted the significance of associated extra-axial intracranial hemorrhages; however, it is not clear that which type of hemorrhage is strongly associated with progression. EDH,[13] SDH,[10,14] subarachnoid hemorrhage (SAH),[9,13,14] and vault fracture[14] are independently associated with HPC in the reported literature. Our study showed similar results that presence of EDH, SDH, and SAH is an independent predictor of progression; however, EDH is more strongly associated as compared to SDH and SAH.

Detrimental outcome of HPC in terms of mortality and morbidity is extensively reported in literature.[8,9,27] However, a number of studies show contradictory results. Iaccarino *et al*. identified neurological deterioration as a better prognostic indicator as compared to progression of hematoma.[20] In our study, length of hospital stay in HPC group was 5.43 days as compared to 3.97 days in non-HPC group ($P = 0.02$), which is similar to the study done by Juratli *et al*.[8] Most of the studies have not evaluated length of stay in relation to HPC.

The overall mortality rate in this study is almost equal among both groups with or without HPC, similar to the study done by Juratli *et al*.[8] Low admission GCS scores and presence of comorbidities (hypertension and diabetes mellitus) are independently associated with higher mortality. Patients with GCS score <8 were at 12 times higher risk of dying compared to those with better GCS scores on admission [Table 3]. Younger age and higher GCS scores are strongly associated with good outcomes in different studies.[10,13,20] However, age is neither related to HPC nor related to mortality in this series. Younger mean age in our study comparative to other studies could justify these findings.[13,20] Type 2 diabetes is associated with progression in one study;[9] however, our study showed association with mortality and not to progression.

A limitation of our study is that we repeat CT scan within 24 h of hospital stay; however, a number of contusions show late progression after 3–4 days.[11] Hence, the result could only be associated with early

HPC. Patients with severe nonsalvageable TBI were not included in the study, which could exclude a proportion of patients with severe TBI.

Conclusion

Progression of contusion in TBI is related to multiplicity, bilaterality, volume, and frontal location with concomitant intra- and extra-axial hematomas. The initial CT scan is a good predictor of high-risk group for progression.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Lobato RD, Cordobes F, Rivas JJ, de la Fuente M, Montero A, Barcena A, *et al.* Outcome from severe head injury related to the type of intracranial lesion. A computerized tomography study. J Neurosurg 1983;59:762-74.
2. Clifton GL, Grossman RG, Makela ME, Miner ME, Handel S, Sadhu V, *et al.* Neurological course and correlated computerized tomography findings after severe closed head injury. J Neurosurg 1980;52:611-24.
3. Cepeda S, Gómez PA, Castaño-Leon AM, Martínez-Pérez R, Munarriz PM, Lagares A, *et al.* Traumatic intracerebral hemorrhage: Risk factors associated with progression. J Neurotrauma 2015;32:1246-53.
4. Yadav YR, Basoor A, Jain G, Nelson A. Expanding traumatic intracerebral contusion/hematoma. Neurol India 2006;54:377-81.
5. Yuan F, Ding J, Chen H, Guo Y, Wang G, Gao WW, *et al.* Predicting progressive hemorrhagic injury after traumatic brain injury: Derivation and validation of a risk score based on admission characteristics. J Neurotrauma 2012;29:2137-42.
6. Le TH, Gean AD. Neuroimaging of traumatic brain injury. Mt Sinai J Med 2009;76:145-62.
7. Chang EF, Meeker M, Holland MC. Acute traumatic intraparenchymal hemorrhage: Risk factors for progression in the early post-injury period. Neurosurgery 2006;58:647-56.
8. Juratli TA, Zang B, Litz RJ, Sitoci KH, Aschenbrenner U, Gottschlich B, *et al.* Early hemorrhagic progression of traumatic brain contusions: Frequency, correlation with coagulation disorders, and patient outcome: A prospective study. J Neurotrauma 2014;31:1521-7.
9. Wang K, Zhao DQ, Zhang JJ, Li YJ, Zhang HD, Shen ZF, *et al.* Risk factors of progressive brain contusion and relationship with outcome. Zhejiang Da Xue Xue Bao Yi Xue Ban 2015;44:410-6.
10. Alahmadi H, Vachhrajani S, Cusimano MD. The natural history of brain contusion: An analysis of radiological and clinical progression. J Neurosurg 2010;112:1139-45.
11. Kurland D, Hong C, Aarabi B, Gerzanich V, Simard JM. Hemorrhagic progression of a contusion after traumatic brain injury: A review. J Neurotrauma 2012;29:19-31.
12. Gudeman SK, Kishore PR, Miller JD, Girevendulis AK, Lipper MH, Becker DP, *et al.* The genesis and significance of delayed traumatic intracerebral hematoma. Neurosurgery 1979;5:309-13.
13. Carnevale JA, Segar DJ, Powers AY, Shah M, Doberstein C, Drapcho B, *et al.* Blossoming contusions: Identifying factors contributing to the expansion of traumatic intracerebral hemorrhage. J Neurosurg 2018;129:1305-16.
14. Allison RZ, Nakagawa K, Hayashi M, Donovan DJ, Koenig MA. Derivation of a predictive score for hemorrhagic progression of cerebral contusions in moderate and severe traumatic brain injury. Neurocrit Care 2017;26:80-6.
15. Orito K, Hirohata M, Nakamura Y, Yamamoto M, Takeshige N, Aoki T, *et al.* Predictive value of leakage signs for pure brain contusional hematoma expansion. J Neurotrauma 2018;35:760-6.
16. White CL, Griffith S, Caron JL. Early progression of traumatic cerebral contusions: Characterization and risk factors. J Trauma 2009;67:508-14.
17. Kothari RU, Brott T, Broderick JP, Barsan WG, Sauerbeck LR, Zuccarello M, *et al.* The ABCs of measuring intracerebral hemorrhage volumes. Stroke 1996;27:1304-5.
18. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, *et al.* Surgical management of traumatic parenchymal lesions. Neurosurgery 2006;58:S25-46.
19. Bobinski L. On Evolution of Intracranial Changes after Severe Traumatic Brain Injury and its Impact on Clinical Outcome. Umeå Universitet; 2016.
20. Iaccarino C, Schiavi P, Picetti E, Goldoni M, Cerasti D, Caspani M, *et al.* Patients with brain contusions: Predictors of outcome and relationship between radiological and clinical evolution. J Neurosurg 2014;120:908-18.
21. Hilmer LV, Park KB, Vycheth I, Wirsching M. Cerebral contusion: An investigation of etiology, risk factors, related diagnoses, and the surgical management at a major Government hospital in Cambodia. Asian J Neurosurg 2018;13:23-30.
22. Oertel M, Kelly DF, McArthur D, Boscardin WJ, Glenn TC, Lee JH, *et al.* Progressive hemorrhage after head trauma: Predictors and consequences of the evolving injury. J Neurosurg 2002;96:109-16.
23. Carnevale J, Segar DJ, Drapcho B, Doberstein C, Morrison JF, Asaad W. 326 Cerebral contusions: Catalysts and counteractants. Neurosurgery 2016;63 Suppl 1:195.
24. Peterson EC, Chesnut RM. Talk and die revisited: Bifrontal contusions and late deterioration. J Trauma 2011;71:1588-92.
25. Narayan RK, Maas AI, Servadei F, Skolnick BE, Tillinger MN, Marshall LF, *et al.* Progression of traumatic intracerebral hemorrhage: A prospective observational study. J Neurotrauma 2008;25:629-39.
26. Smith JS, Chang EF, Rosenthal G, Meeker M, von Koch C, Manley GT, *et al.* The role of early follow-up computed tomography imaging in the management of traumatic brain injury patients with intracranial hemorrhage. J Trauma 2007;63:75-82.
27. Allard CB, Scarpelini S, Rhind SG, Baker AJ, Shek PN, Tien H, *et al.* Abnormal coagulation tests are associated with progression of traumatic intracranial hemorrhage. J Trauma 2009;67:959-67.