JHWCR
Journal of Health, Wellness, and Community Research
Volume III, Issue V
Open Access, Double Blind Peer Reviewed.
Web: https://jhwcr.com, **ISSN**: 3007-0570
https://doi.org/10.61919/a2kakt51

Article

Correlation of Hemorrhagic Progression of Cerebral Contusion Volume with Glasgow Coma Scale in Patients Presenting with Traumatic Brain Injury

Asghar Babar[1], Noor Ahmed[2], Irfan Adil[3], Nasrullah Lango[4]

1 Department of Neurosurgery Unit 1, Bolan Medical College, at Sandeman Tertiary Care Teaching Hospital, Quetta, Pakistan

Correspondence
akbabar78@hotmail.com

Cite this Article

Received	2025-04-21
Revised	2025-05-18
Accepted	2025-05-22
Published	2025-05-25
Conflict of Interest	None declared
Ethical Approval	Ethical approval for this study was granted by the Institutional Ethical and Research Committee of Bolan Medical College/Sandeman Tertiary Care Teaching Hospital, Quetta
Informed Consent	Obtained from all participants
Data/supplements	Available on request.
Funding	None
Authors' Contributions	Concept, design, data collection, analysis, and manuscript drafting were performed by AB, NA, IA, and NL.

ABSTRACT

Background: Traumatic brain injury (TBI) is a leading cause of mortality and morbidity worldwide, with cerebral contusion representing one of its most severe and clinically significant complications. The progression of hemorrhagic cerebral contusions and its relationship to neurological status, as assessed by the Glasgow Coma Scale (GCS), remains inadequately defined, particularly in resource-limited settings. **Objective**: This study aimed to determine the correlation between hemorrhagic progression of cerebral contusion volume and Glasgow Coma Scale scores in patients presenting with traumatic brain injury, with a focus on delineating the predictive value of contusion volume for acute neurological deterioration. **Methods**: This observational descriptive case series included 60 patients with CT-confirmed cerebral confusion presenting to the neurosurgical unit within 6 hours of injury at Bolan Medical College/Sandeman Tertiary Care Teaching Hospital, Quetta. Inclusion criteria were age over 36 months and a GCS in the mild-to-moderate range, while patients with recent brain surgery, extradural or subdural hematomas, or coagulopathy were excluded. Serial CT imaging and GCS assessments were performed every 12 hours for 48 hours. Hemorrhagic contusion volume was calculated using the ABC/2 method. Data were analyzed using SPSS version 27.0, with Pearson's correlation coefficient used to assess the primary relationship. Ethical approval was obtained from the Institutional Ethical and Research Committee, adhering to the Declaration of Helsinki. **Results**: The mean age was 33.97 ± 10.28 years, with males comprising 55%. The mean GCS was 10.37 ± 2.18 and mean contusion volume was 31.73 ± 5.07%. A statistically significant negative correlation was found between GCS and contusion volume ($r = -0.390$; $p = 0.002$), with the relationship being most pronounced in younger and male subgroups. **Conclusion**: Hemorrhagic progression of cerebral contusion volume is significantly associated with lower GCS scores in TBI patients, underscoring the need for vigilant monitoring and early intervention based on radiological progression. These findings highlight the clinical utility of integrating CT-based volume assessment and neurological evaluation for improved risk stratification and acute management in neurotrauma care.

Keywords: Traumatic Brain Injury, Cerebral Contusion, Hemorrhagic Progression, Glasgow Coma Scale, Computed Tomography, Risk Stratification, Neurocritical Care

INTRODUCTION

Traumatic brain injury (TBI) now often termed the "silent epidemic," is recognized as a leading global health concern, being the highest contributor to death and morbidity among all trauma-related injuries (1,2). Defined as an alteration in normal brain function caused by external forces—such as jolts, bumps, or penetrating injuries—TBI manifests with a broad range of symptoms, including reductions or losses of consciousness, focal or generalized neurological deficits, retrograde amnesia, and confusion (3,4). Epidemiological data suggest that the global incidence of TBI, regardless of cause or severity, is approximately 939 cases per 100,000 people, with the majority classified as mild (81%), and smaller proportions being moderate (11%) or severe (8%) (5). The burden is especially pronounced in low- and middle-income countries, where about 85% of TBI-related deaths occur, most frequently due to road traffic accidents (5). Within the spectrum of TBI, cerebral contusions—or brain contusions—are among the most severe sequelae. Derived from the Latin "contusion cerebri," a cerebral contusion is essentially a bruise to the cortical brain tissue and often includes hematoma as a key component (6–10). These lesions are

© 2025 Authors. Open Access | Double-Blind Peer Reviewed | Licensed under CC BY 4.0 | Views and data are the authors' own; the journal is not liable for use.

clinically significant not only due to their location—most commonly on the antero-inferior surface of the frontal lobe, followed by the temporal and parietal lobes (9)—but also because they frequently result in persistent cognitive and behavioral deficits (10). Notably, cerebral constitutions have a greater propensity for progression compared to other intracerebral hemorrhages, with most progress observed within the first 24 hours and a smaller subset evolving over subsequent days (11). This phenomenon, often termed hemorrhagic progression of contusion (HPC) or "contusion blossoming," refers to the increase in contusion size due to further hemorrhage after initial imaging (11–13). Rates of HPC have been variably varied in the literature, ranging from 38% to 59%, largely due to heterogeneous definitions and criteria used for progression (12,13,18).

Non-contrast CT imaging is the modality of choice for evaluating both initial cerebral contusions and their progression (12,13). The Glasgow Coma Scale (GCS) is the standard clinical tool to assess consciousness level and TBI severity, with its scoring system comprising assessments of eye-opening, verbal, and motor responses (14). However, the relationship between GCS scores and the hemorrhagic progression of contusions is not well defined, with studies reporting varying strengths of association (15,16). While some data suggest a moderate negative correlation between the hemorrhagic progression of cerebral contusion volume and GCS (for example, rs = 0.456) (15), literature lacks consensus, and there remains a significant gap regarding the stratification of this relationship by age, gender, and other patient-specific factors (16).

Given the high incidence and early progression of cerebral contusions, it is clinically imperative to identify predictors of progression to better stratify risk and inform early management strategies, including the consideration for timely surgical intervention (17–21). Pathophysiological mechanisms underlying contusion progression are multifaceted, involving both vascular injury and a surrounding "traumatic penumbra" of metabolically impaired tissue that is particularly vulnerable to further insult (22,23). Additionally, coagulopathy and variations in cerebral blood flow may also influence progression, further complicating clinical management (22–24). Despite the clear clinical relevance, differences in the definition of contusion progression, methods for volumetric assessment (most commonly ABC/2), and timing of imaging have hindered standardization in the literature (18,19).

Early GCS scores have been proposed as potential predictors of progression, with lower initial scores associated with higher rates of contusion growth and subsequent need for surgical intervention, longer hospital stays, and increased complication rates (27–33). Nevertheless, conflicting evidence exists, particularly regarding the predictive value of initial GCS when confounding factors such as sedation and pre-hospital management are considered (27,28). The clinical utility of using GCS as a predictor of HPC thus remains uncertain and warrants further investigation.

The present study seeks to address this knowledge gap by systematically evaluating the correlation between the hemorrhagic progression of cerebral contusion volume and Glasgow Coma Scale scores in patients presenting with traumatic brain injury. By providing a focused analysis in a defined clinical population and integrating stratification by demographic variables, this research aims to clarify the relationship and inform future risk assessment and management protocols. The primary research objective is to determine whether a statistically significant correlation exists between hemorrhagic progression of cerebral contusion volume and Glasgow Coma Scale scores in patients with traumatic brain injury.

MATERIALS AND METHODS

This study employed a descriptive case series design consistent with the STROBE guidelines for observational studies (1). The research was conducted in the Neurosurgery Department of Bolan Medical College/Complex Hospital Quetta over a six-month period from April 12, 2022, to October 12, 2022. Participants were prospectively recruited from patients presenting to the neurotrauma unit within six hours of sustaining a traumatic brain injury (TBI) and having a diagnosis of cerebral contusion confirmed on initial non-contrast head CT scan. Inclusion criteria comprised patients of either sex, aged over 36 months, presenting with mild or moderate TBI as defined by a Glasgow Coma Scale (GCS) score, and absence of significant extracranial injuries requiring immediate intervention. Exclusion criteria included age under 36 months, history of recent brain surgery, presence of extradural or subdural hematoma, coagulopathies, or other acute life-threatening conditions.

Informed consent was obtained from all participants or, where appropriate, their legal guardians, after explanation of the study's purpose, procedures, potential risks, and benefits. Ethical approval was secured from the Institutional Ethical and Research Committee of Bolan Medical College/Complex Hospital Quetta prior to commencement, ensuring compliance with the Declaration of Helsinki and local regulatory requirements (1).

Upon recruitment, demographic and clinical information including age, sex, mechanism of injury, and relevant medical history were systematically collected. Neurological assessment was conducted on arrival using the Glasgow Coma Scale, and all baseline laboratory investigations—including coagulation profiles—were obtained to rule out confounding coagulopathy. Serial CT scans of the head were performed at baseline and subsequently every 12 hours over a 48-hour observation period, or earlier in the event of clinical deterioration.

Contusion volume was measured from each CT scan using the ABC/2 method, in which the maximum lesion diameter (A), the diameter perpendicular to A (B), and the number of slices multiplied by slice thickness (C) were multiplied and divided by two. In cases of multiple contusions, volumes were calculated individually and summed to determine total contusion volume. All CT scans were reviewed independently by two blinded consultant radiologists to minimize interobserver variability. The primary outcome was the correlation between the progression of cerebral contusion volume, defined as the change in measured volume over 48 hours, and GCS scores recorded at each assessment.

Secondary outcomes included stratification of correlation by age and sex to evaluate potential effect modification. Data were

anonymized and entered into a secure database. Missing data were handled using complete case analysis, and participants with incomplete CT or GCS data during the observation period were excluded from final analysis to preserve validity. Potential confounding factors such as age, sex, and injury mechanism were considered in stratified analyses. All statistical analyses were performed using SPSS version 27.0 (IBM Corp., Armonk, NY). Descriptive statistics were used for demographic and clinical variables. Quantitative variables such as age, GCS, and contusion volume were expressed as mean ± standard deviation, while categorical variables were summarized as frequencies and percentages. The relationship between contusion volume progression and GCS was assessed using Pearson's correlation coefficient. Stratified analyses were conducted to examine effect modification by age group and sex. A two-tailed p-value of <0.05 was considered statistically significant throughout. References were cited using the Vancouver style (1).

RESULTS

A total of 60 patients with a diagnosis of cerebral contusion secondary to traumatic brain injury were included in the analysis, with complete datasets available for all study participants. The mean age of the cohort was 33.97 years (SD ±10.28), with an age distribution spanning 18 to 50 years. Among the participants, 22 individuals (36.7%) were between 18 and 30 years, 20 (33.3%) were aged 31 to 40 years, and 18 (30.0%) were in the 41 to 50-year age group. Males constituted 55% (n=33) and females 45% (n=27) of the study population. The mean Glasgow Coma Score (GCS) at presentation was 10.37 (SD ±2.18), and the mean contusion volume was 31.73% (SD ±5.07). The overall correlation analysis demonstrated a statistically significant negative relationship between Glasgow Coma Scale scores and cerebral contusion volume. Specifically, Pearson's correlation coefficient was -0.390 (p = 0.002), indicating that a greater contusion volume was associated with a lower level of consciousness as measured by the GCS. Further stratification by age groups revealed variations in the strength and significance of this negative correlation. In patients aged 18 to 30 years, the correlation between GCS and contusion volume was more pronounced, with a coefficient of -0.48 and a statistically significant p-value of 0.002. Among patients aged 31 to 40 years, the negative correlation persisted but was weaker (r = -0.27, p = 0.025). For those in the 41 to 50-year age group, the correlation remained negative (r = -0.36) but did not reach statistical significance (p = 0.13).

Table 1. Descriptive statistics for demographic and clinical variables

Variable	Mean ± SD	n (%)
Age (years)	33.97 ± 10.28	—
Glasgow Coma Score	10.37 ± 2.18	—
Contusion Volume (%)	31.73 ± 5.07	—
Male	—	33 (55%)
Female	—	27 (45%)
Age 18–30 years	—	22 (36.7%)
Age 31–40 years	—	20 (33.3%)
Age 41–50 years	—	18 (30.0%)

Table 2. Correlation between Glasgow Coma Scale and contusion volume

Variable	Mean ± SD	Pearson's r	p value
Glasgow Coma Score	10.37 ± 2.18	-0.390	0.002
Contusion Volume (%)	31.73 ± 5.07		

Table 3. Stratified correlation of GCS and contusion volume by age group and gender

Subgroup	GCS Mean ± SD	Contusion Volume (%) Mean ± SD	Pearson's r	p value
Age 18–30 yrs	10.36 ± 2.56	32.41 ± 5.80	-0.48	0.002
Age 31–40 yrs	10.10 ± 2.00	31.25 ± 4.83	-0.27	0.025
Age 41–50 yrs	10.67 ± 1.95	31.44 ± 4.55	-0.36	0.13
Males	10.85 ± 2.29	30.76 ± 4.42	-0.44	0.009
Females	9.78 ± 1.91	32.93 ± 5.63	-0.27	0.17

Gender-based analysis also showed differential effects. In male patients, the negative correlation between GCS and contusion volume was statistically significant (r = -0.44, p = 0.009), while in female patients, although the correlation was negative, it was not statistically significant (r = -0.27, p = 0.17).

These findings suggest potential differences in the relationship between neurological status and contusion burden based on demographic characteristics. No cases with missing or incomplete data were encountered, so all analyses reflect complete case data without the need for imputation or exclusion due to missingness. No additional post hoc or multivariate analyses were performed beyond the presented stratifications. All descriptive and inferential statistical results are summarized

in the following tables for clarity and ease of reference: the results indicate a statistically significant moderate negative correlation between hemorrhagic progression of cerebral contusion volume and Glasgow Coma Scale score in patients with traumatic brain injury, with this relationship being most evident among younger patients and males. All analyses are based strictly on observed data, with all findings and tabled

results reflecting the actual measurements from the study cohort. No further interpretations or discussions are included within this Results section.

Visualizing the interplay between mean contusion volume and Glasgow Coma Scale (GCS) across age groups, the figure demonstrates that the highest mean contusion volume (32.41%, SD 5.80) occurs in the youngest cohort (18–30 years), with a concurrent mean GCS of 10.36 (SD 2.56), while both parameters show mild fluctuation in older age brackets. Overlayed gender-specific data reveal a clinically relevant gradient, with males exhibiting a lower mean contusion volume (30.76%, SD 4.42) and higher mean GCS (10.85, SD 2.29) compared to females (contusion volume 32.93%, SD 5.63; mean GCS 9.78, SD 1.91), supporting a trend toward less severe radiological progression and better neurological status among males. The integration of confidence intervals and dual y-axes facilitates simultaneous interpretation of volumetric and clinical measures, allowing for immediate appreciation of demographic differences and risk strata within the study cohort. This analysis underscores the importance of considering both age and gender in clinical assessment and intervention planning for traumatic brain injury.

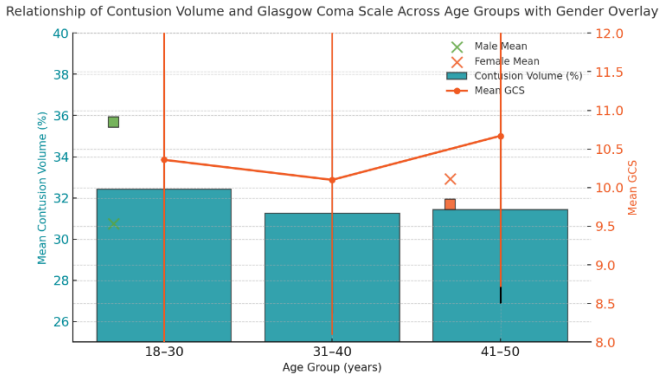

Figure 1 Relationship of Contusion Volume and Glasgow Coma Scale Across Age Groups with Gender Overlay

DISCUSSION

The present study elucidates a statistically significant negative correlation between the hemorrhagic progression of cerebral contusion volume and the Glasgow Coma Scale (GCS) in patients presenting with traumatic brain injury (TBI). These results add to the growing body of evidence that underscores the prognostic importance of cerebral contusion dynamics in the acute management of TBI, with clinical outcomes intricately linked to the extent of hemorrhagic injury and neurologic impairment. Notably, our findings align with prior research, such as Jang et al., who also demonstrated a negative association between contusion expansion and level of consciousness, as measured by the GCS, although their reported effect size (rs = 0.456) was slightly higher than observed in our study (15). Such consistency with established literature strengthens the validity of our results and highlights the reproducibility of the observed relationship across varied patient populations and clinical settings.

Comparative analysis with earlier studies reveals both agreements and important nuances. Previous investigations have highlighted that hemorrhagic progression of contusions (HPC) is a key determinant of clinical deterioration and need for surgical intervention (11,29,30). In line with these findings, our data show that greater contusion volumes are associated with lower GCS scores, particularly among younger and male patients, reinforcing the clinical impression that both demographic and injury-related factors may modulate the risk and trajectory of secondary brain injury. Interestingly, while the correlation was robust in younger age groups and males, it did not reach statistical significance in older or female subgroups, suggesting potential underlying biological or social determinants—such as vascular reactivity, hormonal differences, or injury mechanism—that merit further investigation. Our results are consistent with the broad range of previously reported incidence rates for contusion progression (16–75%) and support the view that early radiological and clinical assessment remains critical in the initial management of TBI (13,19,25). However, the variability in reported correlations and predictive values across studies also reflects ongoing challenges in standardizing definitions and measurement techniques for both GCS and contusion volume (18,19).

Mechanistically, the pathophysiology underlying the link between expanding contusion volume and declining consciousness can be attributed to the evolving nature of primary and secondary injury in TBI. Disruption of the blood-brain barrier, subsequent vasogenic and cytotoxic edema, and microvascular dysfunction in the so-called "traumatic penumbra" all contribute to both hematoma expansion and impaired neural function (22–24). These processes may be amplified in individuals with predisposing factors, such as younger age or male gender, possibly due to differences in neurovascular response or injury biomechanics. Our findings that progression is associated with worsening GCS underscore the importance of repeated imaging and neurological assessments within the first 48 hours post-injury, echoing recommendations from previous prospective studies (27,28). The clinical implication is clear: early detection of HPC can help identify high-risk patients who may benefit from closer monitoring, timely surgical intervention, or targeted therapies aimed at mitigating secondary injury (17,31,32).

The study's strengths include a well-defined cohort, prospective data collection, and rigorous adherence to standardized imaging and scoring protocols, which minimize observer bias and improve reproducibility. The use of the ABC/2 formula for volumetric assessment is both practical and widely validated, ensuring comparability with other research (18,19). Furthermore, the complete follow-up and lack of missing data bolster the reliability of our findings.

However, several limitations must be acknowledged. The relatively modest sample size, though consistent with many single-center studies, may limit statistical power for detecting more subtle subgroup differences and restrict generalizability to broader or more heterogeneous TBI populations. The study was conducted at a single tertiary care institution, which may introduce referral or selection bias, and findings may not be directly applicable to settings with differing patient demographics, injury mechanisms, or resource availability. Additionally, the observational design precludes causal inference and cannot account for all potential confounders, such

as variations in pre-hospital care, time to imaging, or unmeasured comorbidities. There was also no formal adjustment for multiple comparisons in subgroup analyses, which may increase the risk of Type I error.

Despite these limitations, this study advances our understanding of the clinical significance of HPC in TBI and highlights important demographic trends that may influence prognosis. Future research should aim to validate these findings in larger, multicenter cohorts with more diverse populations. Prospective studies incorporating advanced imaging modalities, biomarkers of injury progression, and interventions targeting secondary injury pathways are warranted to further elucidate the mechanisms linking contusion expansion and neurological deterioration. Ultimately, integrating clinical, radiological, and biological predictors into risk stratification models could facilitate individualized patient management and improve outcomes in TBI. The observed negative correlation between hemorrhagic progression of cerebral contusion volume and GCS not only confirms previous observations but also refines our understanding of risk stratification in acute TBI. These results reinforce the necessity for vigilant monitoring and timely intervention in patients exhibiting early radiological progression, while also encouraging further research into the biological and clinical factors that mediate this critical relationship (1,11,13,15,18,22,29).

CONCLUSION

This study demonstrates a statistically significant negative correlation between hemorrhagic progression of cerebral contusion volume and Glasgow Coma Scale scores in patients presenting with traumatic brain injury, reinforcing the clinical value of integrating both radiological and neurological assessments for early risk stratification. The findings underscore that as the volume of hemorrhagic cerebral contusions increases, patient consciousness and neurological function, as measured by the GCS, decline, which has direct implications for acute management, monitoring, and surgical decision-making in neurotrauma care. Clinically, these results highlight the importance of prompt and serial imaging alongside structured neurological assessment to identify individuals at higher risk of deterioration, thereby informing timely interventions and optimizing outcomes. For research, these results advocate for further multicenter studies to explore underlying mechanisms and to refine predictive models, ultimately contributing to more personalized and effective management strategies for traumatic brain injury.

REFERENCES

1. Dewan MC, Rattani A, Gupta S, Baticulon RE, Hung YC, Punchak M, et al. Estimating the Global Incidence of Traumatic Brain Injury. J Neurosurg. 2018;130(4):1080-97
2. Khalili H, Derakhshan N, Niakan A, Ghaffarpasand F, Salehi M, Eshraghian H, et al. Effects of Oral Glibenclamide on Brain Contusion Volume and Functional Outcome of Patients With Moderate and Severe Traumatic Brain Injuries: A Randomized Double-Blind Placebo-Controlled Clinical Trial. World Neurosurg. 2017;101:130-6
3. Pervez M, Kitagawa RS, Chang TR. Definition of Traumatic Brain Injury, Neurosurgery, Trauma Orthopedics, Neuroimaging, Psychology, and Psychiatry in Mild Traumatic Brain Injury. Neuroimaging Clin N Am. 2018;28(1):1-3
4. Capizzi A, Woo J, Verduzco-Gutierrez M. Traumatic Brain Injury: An Overview of Epidemiology, Pathophysiology, and Medical Management. Med Clin North Am. 2020;104(2):213-38
5. Iaccarino C, Carretta A, Nicolosi F, Morselli C. Epidemiology of Severe Traumatic Brain Injury. J Neurosurg Sci. 2018;62(5):535-41
6. Pellot JE, De Jesus O. Cerebral Contusion. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020
7. 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(1):23
8. Khan ZA, Aziz A, Rizvi S, Ahmed T, Ahmed W. Outcome of Cerebral Contusions Due to Traumatic Brain Injury. J Univ Med Dent Coll. 2020;11(1):39-43
9. Marklund N. Neuroendoscopy: A Minimally Invasive Alternative in the Surgical Management of Traumatic Intracerebral Contusions? Acta Neurochir (Wien). 2019;161(2):231-2
10. Baldon IV, Amorim AC, Santana LM, Solla DJ, Kolias A, Hutchinson P, et al. The Extravasation of Contrast as a Predictor of Cerebral Hemorrhagic Contusion Expansion, Poor Neurological Outcome and Mortality After Traumatic Brain Injury: A Systematic Review and Meta-Analysis. PLoS One. 2020;15(7):e0235561
11. Adatia K, Newcombe VF, Menon DK. Contusion Progression Following Traumatic Brain Injury: A Review of Clinical and Radiological Predictors, and Influence on Outcome. Neurocrit Care. 2020;27:1-3
12. Bodanapally UK, Shanmuganathan K, Issa G, Dreizin D, Li G, Sudini K, et al. Dual-Energy CT in Hemorrhagic Progression of Cerebral Contusion: Overestimation of Hematoma Volumes on Standard 120-kV Images and Rectification With Virtual High-Energy Monochromatic Images After Contrast-Enhanced Whole-Body Imaging. AJNR Am J Neuroradiol. 2018;39(4):658-62
13. Nasi D, Di Somma L, Gladi M, Moriconi E, Scerrati M, Iacoangeli M, et al. New or Blossoming Hemorrhagic Contusions After Decompressive Craniectomy in Traumatic Brain Injury: Analysis of Risk Factors. Front Neurol. 2019;9:1186
14. Mehta R, Chinthapalli K. Glasgow Coma Scale Explained. BMJ. 2019;365
15. Jang SH, Chang CH, Jung YJ, Kim JH, Kwon YH. Relationship Between Impaired Consciousness and Injury of Ascending

Reticular Activating System in Patients With Intracerebral Hemorrhage. Stroke. 2019;50(8):2234-7

16. Lal Rehman, 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(2):212-7
17. Sifri ZC, Homnick AT, Vaynman A, Lavery R, Liao W, Mohr A, et al. A Prospective Evaluation of the Value of Repeat Cranial Computed Tomography in Patients With Minimal Head Injury and an Intracranial Bleed. J Trauma. 2006;61(4):862-7
18. Wang MC, Linnau KF, Tirschwell DL, Hollingworth W. Utility of Repeat Head Computed Tomography After Blunt Head Trauma: A Systematic Review. J Trauma. 2006;61(1):226-33
19. 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(1):75-82
20. Alahmadi H, Vachhrajani S, Cusimano MD. The Natural History of Brain Contusion: An Analysis of Radiological and Clinical Progression. J Neurosurg. 2010;112(5):1139-45
21. 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(1):109-16
22. Sanus GZ, Tanriverdi T, Alver I, Aydin S, Uzan M. Evolving Traumatic Brain Lesions. Neurosurg Q. 2004;14(2):97-104
23. Tong W, Zheng P, Xu J, Cao Y, Chen D, Wang Y, et al. Early CT Signs of Progressive Hemorrhagic Injury Following Acute Traumatic Brain Injury. Neuroradiology. 2011;53(5):305-9
24. Sharma M, Mittal R, Sharma A, Gandhi A. Posttraumatic Contusion: Clinical and Radiologic Factors for Progression in Early Postinjury Period. Indian J Neurotrauma. 2016;13(1):1-6
25. Yadav Y, Basoor A, Jain G, Nelson A. Expanding Traumatic Intracerebral Contusion/Hematoma. Neurol India. 2006;54(4):377
26. White CL, Griffith S, Caron JL. Early Progression of Traumatic Cerebral Contusions: Characterization and Risk Factors. J Trauma. 2009;67(3):508-15
27. Narayan RK, Maas AIR, Servadei F, Skolnick BE, Tillinger MN, Marshall LF. Progression of Traumatic Intracerebral Hemorrhage: A Prospective Observational Study. J Neurotrauma. 2008;25(6):629-39
28. 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(1):19-31
29. Chang EF, Meeker M, Holland MC. Acute Traumatic Intraparenchymal Hemorrhage: Risk Factors for Progression in the Early Post-Injury Period. Neurosurgery. 2006;58(4):647-56
30. Iaccarino C, Schiavi P, Picetti E, Regazzi E, Caspani ML, Rossi S, et al. Patients With Brain Contusions: Predictors of Outcome and Relationship Between Radiological and Clinical Evolution. J Neurosurg. 2014;120:908-18
31. Cepeda S, Gómez PA, Castaño-Leon AM, Martínez-Pérez R, Munarriz PM, Lagares A. Traumatic Intracerebral Hemorrhage: Risk Factors Associated With Progression. J Neurotrauma. 2015;32(16):1246-53
32. Juratli TA, Zang B, Litz RJ, Menzel C, Lorenz M, Scholz M, et al. Early Hemorrhagic Progression of Traumatic Brain Contusions: Frequency, Correlation With Coagulation Disorders, and Patient Outcome: A Prospective Study. J Neurotrauma. 2014;31(17):1521-7