HHS Public Access

Author manuscript

Neurocrit Care. Author manuscript; available in PMC 2017 February 01.

Published in final edited form as:

Neurocrit Care. 2017 February ; 26(1): 80–86. doi:10.1007/s12028-016-0303-5.

Derivation of a Predictive Score for Hemorrhagic Progression of Cerebral Contusions in Moderate and Severe Traumatic Brain Injury

Randall Z. Allison[1], **Kazuma Nakagawa**[2,3], **Michael Hayashi**[4,5], **Daniel J. Donovan**[2,4], and **Matthew A. Koenig**[2,3]

[1]Division of Neurosurgery, Department of Surgery, University of Texas Medical Branch, Galveston, TX, USA

[2]Department of Surgery, Neuroscience Institute, The Queen's Medical Center, 1301 Punchbowl Street- QET5, Honolulu,HI 96813, USA

[3]Department of Medicine, John A. Burns School of Medicine,University of Hawaii, Honolulu, HI, USA

[4]Department of Surgery, John A. Burns School of Medicine,University of Hawaii, Honolulu, HI, USA

[5]Department of Surgery, The Queen's Medical Center,Honolulu, HI, USA

Abstract

Backgrounds—After traumatic brain injury (TBI), hemorrhagic progression of contusions (HPCs) occurs frequently. However, there is no established predictive score to identify high-risk patients for HPC.

Methods—Consecutive patients who were hospitalized (2008–2013) with non-penetrating moderate or severe TBI were studied. The primary outcome was HPC, defined by both a relative increase in contusion volume by ≥30 % and an absolute increase by ≥10 mL on serial imaging. Logistic regression models were created to identify independent risk factors for HPC. The HPC Score was then derived based on the final model.

Results—Among a total of 286 eligible patients, 61 (21 %) patients developed HPC. On univariate analyses, HPC was associated with older age, higher initial blood pressure, antiplatelet medications, anticoagulants, subarachnoid hemorrhage (SAH) subdural hematoma (SDH), skull fracture, frontal contusion, larger contusion volume, and shorter interval from injury to initial CT. In the final model, SAH (OR 6.33, 95 % CI, 1.80–22.23), SDH (OR 3.46, 95 % CI, 1.39–8.63), and skull fracture (OR 2.67, 95 % CI, 1.28–5.58) were associated with HPC. Based on these factors, the HPC Score was derived (SAH = 2 points, SDH = 1 point, and skull fracture = 1 point). This score had an area under the receiver operating curve of 0.77. Patients with a score of 0–2 had a 4.0 % incidence of HPC, while patients with a score of 3–4 had a 34.6 % incidence of HPC.

Correspondence to: Matthew A. Koenig.

Compliance with Ethical Standards: Conflict of interest The authors report no conflicts of interest related to this manuscript.

Conclusions—A simple HPC Score was developed for early risk stratification of HPC in patients with moderate or severe TBI.

Keywords

Cerebral contusion; Traumatic brain injury; Triage; Critical care; Resource allocation

Introduction

In blunt traumatic brain injury (TBI), hemorrhagic progression of contusions (HPCs) refers to enlargement of the hemorrhagic component of cerebral contusions after initial imaging [1]. Commonly called contusion "blossoming," HPC can result in neurological worsening from mass effect, cerebral edema, and parenchymal tissue necrosis [2, 3]. Prior case series have reported an incidence of HPC in TBI patients that ranges from 18 to 51 % [4–10]. Early identification of patients at high risk for HPC could lead to interventions that reduce the morbidity and mortality of TBI. Conversely, for patients who are at low risk for HPC, neurological monitoring in the intensive care unit (ICU) with frequent serial neurological examinations and repeat imaging studies substantially adds to the acuity and cost of care for TBI patients [11].

Prior reports have identified that radiographic factors such as the presence of a subdural hematoma (SDH) [4, 6, 12], subarachnoid hemorrhage (SAH) [4, 5, 7], and larger contusion volume [5, 6, 10] are associated with higher risk for HPC, as well as clinical factors such as older age [4, 13], hyperglycemia [14], antiplatelet medication use [15], low platelet count [16], and timing of the initial CT scan [4, 7]. One prior group attempted to develop a predictive score using clinical parameters [14]; but the lowest risk patient group still had a high incidence of HPC (10.3 %) in this study, and the algorithm was complex.

A simple predictive score for HPC could provide a useful tool to improve resource allocation, especially in settings where ICU beds or specialty services may be limited. In this retrospective cohort study, we identified common clinical and radiographic factors associated with HPC in patients admitted for moderate or severe TBI. These factors were then used to derive a predictive scoring model that could be easily calculated to stratify risk for HPC. We hypothesized that an accurate predictive score for HPC could be derived from early markers of injury severity.

Methods

The Queen's Medical Center (QMC) Institutional Review Board approved this research project with waiver of informed consent. A prospectively collected trauma database from QMC, the only American College of Surgeons-verified trauma center in Hawaii, was queried to identify consecutive patients admitted for non-penetrating moderate or severe TBI from January 1, 2008, to December 31, 2013. Severe TBI was defined as blunt head trauma with initial Glasgow Coma Scale (GCS) of 3–8, and moderate TBI was defined as initial GCS of 9–12 [17].

The standard practice at QMC is to admit patients with severe TBI to a dedicated Neurosciences ICU staffed by specialized neurocritical care nurses, acute care nurse practitioners, and board-certified neurointensivists. Moderate TBI patients are triaged to either the Neurosciences ICU or a neurological specialty acute medical-surgical ward by the admitting trauma surgeon and neurosurgeon based on the anticipated clinical course of the patient. Repeat brain CT within 6–12 h after the initial scan is a standard institutional practice unless there are reasons not to perform repeat imaging, e.g., rapid neurological improvement or injuries that are perceived to be nonsurvivable. A portable bedside CT scanner (Ceretom) is available in the ICU and is often used for repeat imaging in the ICU patients.

Inclusion and Exclusion Criteria

The trauma database was queried for all TBI patients admitted during the study period. Consecutive patients with moderate or severe blunt TBI and repeat brain CT within 24 h were included in the analysis. The presence of a cerebral contusion on the initial brain CT was not required for inclusion because development of contusions may occur after the initial CT, and we believed that an accurate predictive score would need to account for delayed contusions. Patients with penetrating brain injury and those who did not undergo repeat brain imaging were excluded. TBI patients who met the initial GCS criteria due to intoxication but had a rapid subsequent neurological improvement to GCS > 12 were excluded from the study.

Outcome Variable

The primary outcome variable for the study was HPC. HPC was defined as both a relative increase in volume of the lesion by ≥30 % compared to the initial CT and an absolute increase in the total contusion volume by ≥10 mL on repeat CT. The ≥30 % threshold was based on prior reports [6] and was chosen to set a relatively high threshold for HPC due to potential differences in technique between the initial and follow-up CT scan. Enlargement of lesion volume by ≥10 mL was based on prior reports [4] and was chosen to identify HPC that had the potential to be clinically significant. The first two brain CT scans were retrospectively evaluated by a board-certified neurointensivist for identification of HPC. Parenchymal hematoma volume was estimated using the ABC/2 method, which has been utilized in prior studies of spontaneous and traumatic hematoma volume [2, 3, 6, 18, 19]. Heterogeneous lesions, defined as mixed high and low density, were measured using the entire length of the hyperdense portion of the lesion. In the case of multiple contusions, the volumes of all contusions were summed together in an effort to account for mass effect from multiple lesions. Punctate contusions were estimated at 1 mL each.

The secondary outcome measure was HPC associated with neurological decline, which was defined by the following criteria: unplanned craniotomy or ICP monitor placement, unplanned endotracheal intubation, unplanned ICU transfer during the first 24 h of hospitalization, or sustained decline in GCS by ≥1 point for ≥24 h that was not attributable to sedation. The GCS criterion was adopted for high sensitivity identification of patients with neurological decline while avoiding inclusion of patients with fluctuations of GCS due to sedative medications and circadian patterns. Additional secondary outcome measures

were hospital length of stay, ICU length of stay, mortality, and discharge modified Rankin Scale (mRS) score.

Factors Associated with HPC

Clinical data were obtained from the prospectively collected trauma database supplemented with retrospective review of the electronic medical record (Epic). Initial GCS was defined as the highest recorded value in the emergency department (ED) obtained by the attending trauma surgeon, neurosurgeon, and/or neurointensivist prior to sedation. Decline of GCS during the first 24 h was defined as a reduction in GCS by ≥1 point that was sustained over 24 h and not attributable to sedation. Initial motor examination and pupil size and reactivity were determined by the attending trauma surgeon, neurosurgeon, or neurointensivist on initial examination in the ED.

Initial brain CT was defined as the first imaging study available in the QMC computerized radiology system. For patients presenting directly to QMC, the initial CT was obtained in the QMC ED. For patients transferred to QMC from another facility, the initial CT was reviewed from the referring hospital if it was electronically transmitted or scanned into the QMC computerized radiology system. All CT scans were evaluated for Marshall CT classification [20]; Rotterdam CT score [21]; presence, displacement, and location of skull fractures; presence, location, and maximal thickness of SAH, epidural hematoma, and SDH; presence, location, and volume of cerebral contusions; extent of midline shift; and presence of intraventricular hemorrhage and hydrocephalus. Examples of HPC in two patients are shown in Fig. 1.

Statistical Analysis

Data were analyzed using commercially available statistical software (SPSS 22.0, Chicago, IL). Patient characteristics were summarized using descriptive statistics appropriate to variable type. Those with HPC were compared to those without HPC using the Chi-square test for categorical data, two-tailed t test for normally distributed, continuous variables, and the Mann–Whitney U test for nonparametric data. Data were reported as mean ± SD except as noted. Variables that could have a biologically plausible association with HPC, could act as potential confounders, or had been reported to be associated with HPC in prior studies were included in the analyses.

A multivariable model using logistic regression was then constructed to identify independent risk factors for HPC. Variables with $p \leq 0.1$ in the univariate testing were entered in the model. Odds ratio (OR) and 95 % confidence interval (CI) were calculated from the beta coefficients and their standard errors. Levels of $p < 0.05$ were considered statistically significant. After performing the initial regression model, a predictive score was derived using a weighted system by dividing the beta coefficients by a common denominator to obtain an integer score proportional to the magnitude of the beta coefficient. The performance of the score was assessed by calculating the area under the receiver operating characteristic (ROC) curve.

Results

Based on the GCS criteria, we identified 419 patients with moderate or severe TBI due to blunt head trauma. Of these patients, we excluded 133 patients who did not have repeat imaging within 24 h of the injury. Among the excluded patients, 19 were children (median age 9 years, range 0–16 years) for whom repeat imaging was not recommended in order to minimize radiation exposure. Among the 114 adult patients who were excluded, 53 died from traumatic injuries prior to performance of a repeat head CT or had injuries that were believed to be nonsurvivable by the treating physician so repeat imaging was not obtained; 36 had no apparent intracranial lesion or minor lesions on the initial head CT, and the treating physician believed that a repeat head CT was unnecessary; 12 had advance directives or early family request for comfort measures only; 7 had improvement in neurological examination, and the treating physician believed that a repeat head CT was unnecessary; and 6 had initial imaging at an outside hospital that was not available for retrospective review.

A total of 286 patients met the inclusion criteria and were included in the final analyses. Of these patients, 146 had moderate TBI and 140 had severe TBI. Characteristics of the study population are shown in Table 1. The median initial composite GCS was 9 (interquartile range (IQR) 6–12). The mean initial contusion volume was 6.2 ± 11.6 mL among patients who had contusions on the initial brain CT. The mean interval from injury to initial brain CT scan was 2.4 ± 1.6 h, and the mean interval between first and second brain CT scan was 9.4 ± 4.8 h. Twenty-eight patients (9.8 %) underwent a craniotomy or decompressive craniectomy prior to the second brain CT, but no patients had evacuation of cerebral contusions. Of the 286 patients, 193(67 %) were initially admitted to the Neurosciences ICU, 36 (12.6 %) were admitted to other ICUs, and 57 (19.9 %) were admitted to the medical-surgical ward. For the total study population, the mean ICU length of stay was 7.6 ± 9.5 days and mean hospital length of stay was 10.9 ± 14.4 days. In-hospital mortality occurred in 59 patients (21 %), and the discharge mRS was ≤2 in 102 patients (36 %).

HPC occurred in 61 patients (21 %) and was associated with neurological decline in 32 (52 %) of these patients. Of the 32 patients with neurological decline due to HPC, 1 required unplanned ICU transfer from the medical-surgical ward, 3 required unplanned endotracheal intubation, and 4 required unplanned craniotomy or decompressive craniectomy. In univariate analyses (Table 2), HPC was associated with older age, higher initial systolic blood pressure (SBP), use of antiplatelet medications, use of anticoagulants, larger contusion volume, shorter interval from injury to initial brain CT, receipt of mannitol, no packed red blood cell (pRBC) transfusion, and presence of SAH, SDH, skull fracture, and frontal lobe contusion on the initial brain CT.

A multivariable regression model was then constructed (Table 3). In the full model, SAH (OR 6.33, 95 % CI, 1.80–22.23), SDH (OR 3.46, 95 % CI, 1.39–8.63), skull fracture (OR 2.67, 95 % CI, 1.28–5.58), and pRBC transfusion (OR 0.31, 95 % CI, 0.10–0.93) were independently associated with HPC. Note that pRBC transfusion was negatively associated with HPC, while the other factors were positively associated with HPC.

Using the regression model, a predictive score was derived based on presence or absence of SAH, SDH, and skull fracture. We did not include pRBC transfusion in the final predictive score. The predictive score was constructed for HPC based on the odds ratio of factors in the full multivariable regression model (Table 3). This HPC Score has a possible range of 0–4 calculated by adding 2 points for presence of SAH, 1 point for presence of SDH, and 1 point for presence of a skull fracture. The area under the ROC curve of the HPC Score was 0.77. None of the patients with an HPC Score of 0 ($n = 38$) had HPC. Patients with an HPC Score of 0–2 had a 4.0 % (5/124) incidence of HPC, while patients with an HPC Score of 3–4 had a 34.6 % (56/162) incidence of HPC (Table 4).

Discussion

In a cohort of 286 patients with moderate or severe blunt TBI, we identified factors associated with HPC and derived a simple scoring model called the HPC Score to stratify risk for HPC. This HPC Score has a possible range of 0–4 calculated by adding 2 points for presence of SAH, 1 point for presence of SDH, and 1 point for presence of a skull fracture. Scores of 0–4 had an observed HPC incidence of 0, 3.7, 7.7, 28.9, and 39.5 %, respectively.

As in previously published reports, we identified presence of SAH [4, 5, 7], skull fracture [7], and SDH [3, 6, 12] as major risk factors for HPC. SAH, skull fracture, and SDH may be markers of the overall severity of head trauma, or they may represent injury to the cortical veins, bridging veins, and venous sinuses that could contribute to venous congestion of the brain and increased risk of hematoma expansion [1]. The reason for the independence of these three risk factors for HPC is not known, but they may represent a cumulative injury to the brain such that patients with skull fracture and SDH in addition to SAH experienced greater brain trauma than patients with SAH alone.

Older age, anticoagulant use, antiplatelet medication use, and frontal contusion location have also been associated with HPC in other studies [4, 12, 13, 15, 22–24] and were identified as risk factors in our univariate analyses; however, they were not independent risk factors for HPC in the multivariable model and were not included in the HPC Score. The lack of an independent association between HPC and anticoagulant or antiplatelet medications may be due to several factors, including incomplete or inaccurate ascertainment of medication use and the small number of patients on these medications. This finding does not necessarily imply that TBI patients on anticoagulant or antiplatelet medications are at low risk for HPC but rather that these patients overwhelmingly had SAH or SDH on the initial brain CT. These radiographic findings, rather than clinical factors like age or medication use, were the strongest predictors of HPC and were, therefore, used to construct the HPC Score.

An advantage of the HPC Score derived by this study is that it entirely relies on a small number of radiographic factors that can easily be obtained from the initial brain CT without detailed knowledge of the patient's medical history or specific neurological examination findings. If it is externally validated, a predictive score with these features could allow early risk stratification for HPC among TBI patients by emergency physicians, trauma surgeons, and radiologists with limited neurological expertise and in trauma patients with unknown

medical history. The HPC Score requires external validation in other cohorts before adoption in clinical practice can be recommended, however.

We observed an apparent protective effect of pRBC transfusion on the incidence of HPC on the multivariable analysis. However, we did not include pRBC transfusion in the HPC Score for several reasons. First, the reason for this association is unclear. As blunt trauma can be heterogeneous, TBI patients commonly experience concomitant trauma to other body systems with potential for blood loss. These injuries may create competing priorities in the initial management of the trauma patient, including the need for immediate hemorrhage control which may delay the time to initial head CT in TBI. The presence of systemic injuries may also distribute traumatic forces away from the head, decreasing the severity of brain trauma. RBC transfusion may also reflect unmeasured biases such as aggressiveness of the initial resuscitative effort. Second, we did not want to imply that TBI patients should undergo packed red blood cell (pRBC) transfusion in order to lower the chance of HPC. Third, the addition of pRBC transfusion did not significantly increase the accuracy of the model.

Limitations of the study include the retrospective design, which could under-identify patients with neurological decline due to reliance on clinical documentation. The focus on radiographic enlargement may also overemphasize changes in lesion size that are not clinically relevant and under-emphasize important clinical deterioration that is unrelated to changes in the hematoma volume, such as per hematoma edema, mass effect, and delayed hydrocephalus. We also excluded 133 (32 %) of the screened patients because they did not undergo repeat head CT within 24 h, which could result in a selection bias excluding patients perceived as low risk for HPC or those with nonsurvivable injuries. Mild TBI patients were not included in the study because repeat brain CT scans are not routinely performed in this group at our institution. This is an important limitation, and the results may not be applicable to patients with mild TBI.

The next steps include external validation of the HPC Score to confirm whether this predictive tool is able to accurately identify patients at high and low risk for HPC. If so, the HPC Score could become an important clinical tool for early HPC risk stratification in patients with TBI. In this paradigm, patients with high risk for HPC could be identified and triaged to a dedicated Neurosciences ICU with more intensive staffing ratios and more frequent neurological examinations, earlier neurosurgical consultation or ICP monitor placement, or clinical trials of antifibrinolytic agents, clotting factor replacement, or intensive blood pressure control. Conversely, early identification of patients at low risk for HPC could allow more efficient allocation of critical care resources.

External validation of the HPC Score should also include patients with mild TBI based on an initial GCS of 13–15. Patients with initially mild TBI can also develop significant HPC, particularly those with bifrontal cerebral contusions [25]. The identification of mild TBI patients at high risk for HPC is particularly important because this group is commonly admitted to lower acuity clinical environments and more rapidly discharged from the hospital.

Acknowledgments

Funding Dr. Nakagawa was partially supported by the National Institute on Minority Health and Health Disparities of the National Institutes of Health (P20MD000173).

References

1. 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. [PubMed: 21988198]
2. Narayan RK, Maas AI, Servadei F, Skolnick BE, Tillinger MN, Marshall LF. Progression of traumatic intracerebral hemorrhage: a prospective observational study. J Neurotrauma. 2008; 25:629–39. [PubMed: 18491950]
3. Iaccarino C, Schiavi P, Picetti E, et al. Patients with brain contusions: predictors of outcome and relationship between radiological and clinical evolution. J Neurosurg. 2014; 120:908–18. [PubMed: 24506250]
4. Oertel M, Kelly DF, McArthur D, et al. Progressive hemorrhage after head trauma: predictors and consequences of the evolving injury. J Neurosurg. 2002; 96:109–16. [PubMed: 11794591]
5. Chieregato A, Fainardi E, Morselli-Labate AM, et al. Factors associated with neurological outcome and lesion progression in traumatic subarachnoid hemorrhage patients. Neurosurg. 2005; 56:671–80.
6. 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. [PubMed: 19575576]
7. Tong WS, Zheng P, Xu JF, et al. Early CT signs of progressive hemorrhagic injury following acute traumatic brain injury. Neuroradiol. 2011; 53:305–9.
8. Kaups KL, Davis JW, Parks SN. Routinely repeated computed tomography after blunt head trauma: does it benefit patients? J Trauma. 2004; 56:475–81. [PubMed: 15128116]
9. Sanus G, Zihni N, Tanriverdi T, Alver I, Aydin S, Uzan M. Evolving traumatic brain lesions: predictors and results of ninety-eight head-injured patients. Neurosurg Q. 2004; 14:97–104.
10. Chang EF, Meeker M, Holland MC. Acute traumatic intraparenchymal hemorrhage: risk factors for progression in the early post-injury period. Neurosurg. 2006; 58:647–56.
11. Reed SD, Bough DK, Meyer K, Jarvik JG. Inpatient costs, length of stay, and mortality for cerebrovascular events in community hospitals. Neurology. 2001; 57:305–14. [PubMed: 11468317]
12. Stein SC, Young GS, Talucci RC, Greenbaum BH, Ross SE. Delayed brain injury after head trauma: significance of coagulopathy. Neurosurg. 1992; 30:160–5.
13. Givner A, Gurney J, O'Connor D, Kassarjian A, Lamorte WW, Moulton S. Reimaging in pediatric neurotrauma: factors associated with progression of intracranial injury. J Pediatr Surg. 2002; 37:381–5. [PubMed: 11877652]
14. Yuan F, Ding J, Chen H, 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. [PubMed: 22568757]
15. Engström M, Romner B, Schalén W, Reinstrup P. Thrombocytopenia predicts progressive hemorrhage after head trauma. J Neurotrauma. 2005; 22:291–6. [PubMed: 15716634]
16. Schnüriger B, Inaba K, Abdelsayed GA, et al. The impact of platelets on the progression of traumatic intracranial hemorrhage. J Trauma. 2010; 68:881–5. [PubMed: 20386283]
17. Brain Trauma Foundation. Guidelines for the management of severe traumatic brain injury, 3rd edition. J Neurotrauma. 2007; 24(Suppl 1):S1–2.
18. Kothari RU, Brott T, Broderick JP, et al. The ABCs of measuring intracerebral hemorrhage volumes. Stroke. 1996; 27:1304–5. [PubMed: 8711791]
19. White CL, Griffith S, Caron JL. Early progression of traumatic cerebral contusions: characterization and risk factors. J Trauma. 2009; 67:508–15. [PubMed: 19741392]
20. Marshall LF, Marshall SB, Klauber MR, et al. A new classification of head injury based on computerized tomography. J Neurosurg. 1991; 75:S14–20.

21. Maas AI, Hukkelhoven CW, Marshall LF, Steyerberg EW. Prediction of outcome in traumatic brain injury with computed tomographic characteristics: a comparison between the computed tomographic classification and combinations of computed tomographic predictors. Neurosurgery. 2005; 57:1173–82. [PubMed: 16331165]

22. Stein SC, Smith DH. Coagulopathy in traumatic brain injury. Neurocrit Care. 2004; 1:479–88. [PubMed: 16174954]

23. Fabbri A, Servadei F, Marchesini G, Stein SC, Vandelli A. Predicting intracranial lesions by antiplatelet agents in subjects with mild head injury. J Neurol Neurosurg Psychiatry. 2010; 81:1275–9. [PubMed: 20643657]

24. Hukkelhoven CW, Steyerberg EW, Rampen AJ, et al. Patient age and outcome following severe traumatic brain injury: an analysis of 5600 patients. J Neurosurg. 2003; 99:666–73. [PubMed: 14567601]

25. Peterson EC, Chesnut RM. Talk and die revisited: bifrontal contusions and late deterioration. J Trauma. 2011; 71:1588–92. [PubMed: 22182868]

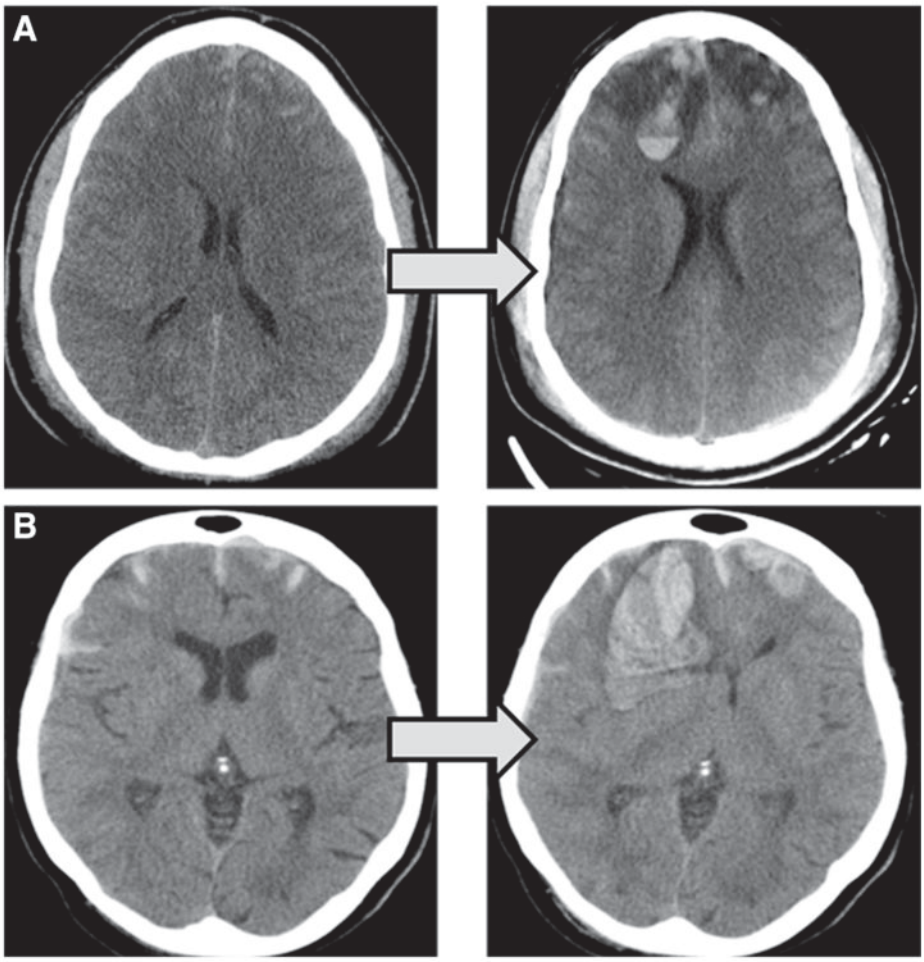

Fig. 1. Examples of hemorrhagic progression of contusions in patients a, b on serial brain CT

Table 1
Characteristics of the study population (*n* = 286)

Population	286
Age (mean ± SD)	47.6 ± 26.7
Sex (% male)	202 (71 %)
Race	
Native Hawaiian or Pacific Islander	85 (30 %)
Asian (non-Filipino)	71 (25 %)
White	72 (25 %)
Filipino	40 (14 %)
Mechanism of injury (% most common)	
Moped/motorcycle crash	36 (12.6 %)
Assault	43 (15 %)
Fall from height	50 (17.5 %)
Fall from standing	88 (30.7 %)
History	
Hypertension	76 (26.6 %)
Diabetes mellitus	36 (12.6 %)
Antiplatelet medications	39 (13.6 %)
Aspirin	32 (11.2 %)
Clopidogrel	5 (1.7 %)
Aspirin + clopidogrel	2 (0.6 %)
Anticoagulant medications	13 (4.5 %)
Warfarin	11 (3.8 %)
Other anticoagulant	2 (0.7 %)
Methamphetamine use	23 (8 %)
Alcohol use	90 (31 %)
Physical examination	
Initial GCS, median (IQR)	9 (6–12)
Initial pupils non-reactive (%)	44 (15.4 %)
Unilateral non-reactive pupil	22 (7.7 %)
Bilateral non-reactive pupils	22 (7.7 %)
Injury Severity Score (mean ± SD)	24 ± 10
Imaging	
Initial Marshall Scale (median)	2c
Initial Rotterdam CT Score (median)	3
Initial contusion volume (mean ± SD)	6.2 ± 11.6

SD standard deviations, *GCS* Glasgow Coma Scale, *IQR* interquartile range, *CT* computed tomography

Table 2
Factors associated with hemorrhagic progression of contusion (HPC)

	HPC	No HPC	*p* value
Population	61	225	
Age (years)	56.7 ± 24.7	45.2 ± 26.7	0.002
Initial GCS	9 ± 4	9 ± 4	0.756
Initial SBP (mmHg)	152 ± 35	138 ± 28	0.005
INR	1.3 ± 0.6	1.2 ± 0.3	0.835
Platelet count	223.6 ± 65.5	244.3 ± 91.8	0.104
Glucose (mg/dL)	163 ± 52	166 ± 78	0.796
Time to initial CT (h)	2.0 ± 0.8	2.5 ± 1.5	<0.001
Contusion volume (mL)	10.1 ± 11.2	5.1 ± 11.4	0.036
SDH width (cm)	0.8 ± 0.5	0.6 ± 0.5	0.057
Midline shift (cm)	0.3 ± 0.4	0.2 ± 0.4	0.087
Frontal lobe location	38 (62 %)	77 (34 %)	<0.001
Antiplatelet meds	13 (21 %)	26 (12 %)	0.049
Anticoagulant meds	6 (10 %)	7 (3 %)	0.044
Methamphetamine use	5 (8.2 %)	18 (8.0 %)	0.96
Alcohol use	15 (25 %)	75 (33 %)	0.19
SAH	58 (95 %)	134 (56 %)	<0.001
SDH	54 (88 %)	125 (56 %)	<0.001
Skull fracture	40 (66 %)	94 (42 %)	0.001
Mannitol use	23 (38 %)	35 (16 %)	<0.001
pRBC transfusion	5 (8 %)	43 (19 %)	0.043

Data shown as mean ± SD or count (percentage)

GCS Glasgow Coma Scale, *SBP* systolic blood pressure, *INR* international normalized ratio, *CT* computed tomography, *SDH* subdural hematoma, *SAH* subarachnoid hemorrhage, *pRBC* packed red blood cells

Table 3
Multivariable models for hemorrhagic progression of contusions (HPCs)

	Model 1 OR (95 % CI)	Model 2 OR (95 % CI)	Model 3 OR (95 % CI)	Full model OR (95 % CI)
SAH	8.43 (2.47, 28.87) *	8.00 (2.34, 27.38) *	7.82 (2.27, 26.93) *	6.33 (1.80, 22.23) *
SDH	4.52 (1.92, 10.65) *	3.93 (1.65, 9.40) *	4.06 (1.69, 9.77) *	3.46 (1.39, 8.63) *
Convexity skull fracture	1.77 (0.93, 3.37)	2.08 (1.05, 4.09) *	2.24 (1.12, 4.48) *	2.67 (1.28, 5.58) *
Antiplatelet use		1.75 (0.74, 4.15)	1.60 (0.66, 3.87)	1.44 (0.55, 3.77)
Anticoagulant use		2.42 (0.67, 8.70)	3.42 (0.90, 13.07)	2.85 (0.75, 10.91)
pRBC transfusion			0.30 (0.10, 0.90) *	0.31 (0.10, 0.93) *
Age				1.01 (1.00, 1.03)
Time to initial CT				0.91 (0.82, 1.01)

CI confidence interval, *SAH* subarachnoid hemorrhage, *SDH* subdural hematoma, *pRBC* packed red blood cells, *CT* computed tomography

* $p < 0.05$

Table 4

Incidence of hemorrhagic progression of contusions (HPCs) based on HPC Score of 0–4

			HPC Score				
			0	**1**	**2**	**3**	**4**
HPC	No	Count	38	45	36	54	52
		(%)	100	96.3	92.3	71.1	60.5
	Yes	Count	0	2	3	22	34
		(%)	0	3.7	7.7	28.9	39.5

Scoring criteria were: presence of SAH = 2 points; presence of SDH = 1 point; presence of skull fracture = 1 point