Early Progression of Traumatic Cerebral Contusions: Characterization and Risk Factors

Carole L. White, RN, PhD, Stephen Griffith, MD, and Jean-Louis Caron, MD

Background: Traumatic intracerebral contusions carry a high rate of early progression and are associated with morbidity and mortality. Our objectives were to better characterize the prevalence of progression of traumatic contusions, risk factors, and the association with outcome.

Methods: Participants were 46 patients with traumatic intracerebral contusion who underwent a repeat computed tomography (CT) scan within 24 hours of injury. Hemorrhage volume on the CT scan was quantified using the ABC/2 technique. Univariate and multivariate statistics were used to define growth (percentage increase and absolute volume increase), to examine the relationship between the risk factors of interest and hemorrhage expansion, and with neurologic function and discharge destination.

Results: Sixty-five percent of the patients experienced progression in the size of the lesion in the initial 24 hours postinjury. The international normalized ratio was significantly higher in the group that demonstrated progression. Deterioration on the Glasgow Coma Score was associated with a threefold risk of hemorrhage expansion being found on the CT as defined by percentage increase (odds ratio [OR] = 3.43; 95% confidence interval [CI]: 0.90 to 13.10) and similarly when defined as absolute increase in volume (OR = 3.32; 95% CI: 0.96 to 11.41). Controlling for injury severity, there was an association between hemorrhage growth and death with those displaying progression more likely to die during hospitalization (OR = 1.08; 95% CI: 0.97 to 1.20).

Conclusion: A high proportion of intracerebral contusions evolve in size very early in the postinjury period and are associated with negative outcomes. There is still not a proven therapy for limiting the expansion although the association of an elevated international normalized ratio with expansion suggests that coagulation abnormalities must be actively corrected.

Key Words: Traumatic brain injury, Intracerebral hematoma, Progressive hemorrhagic injury, Outcomes, Coagulopathy.

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physiological cascades. A better understanding of the acute course and the risk factors specifically for intracerebral contusion expansion is needed to better define the therapeutic window and to develop effective treatments, particularly for small lesions that are not managed surgically. Thus, the objectives of this study were (i) to estimate the proportion of patients with a traumatic hemorrhagic cerebral contusion who undergo intracranial surgery before the time of the second scan; (ii) no CT evidence of other intracranial hemorrhages; (iii) initial CT scan demonstrated a hemorrhagic cerebral contusion; (iv) did not undergo intracranial surgery before the time of the second scan; (v) were not irretrievably injured as defined by an AIS score of 6; and (vi) managed according to an adult protocol. The sample was restricted to those who did not undergo intracranial surgery before the second scan to better characterize the course and outcomes of small intracerebral contusions. Infants and young children were excluded as there is evidence that their clinical course and prognosis differ from adults. Permission to review the medical records was provided by the Institutional Review Board.

### PATIENTS AND METHODS

#### Study Sample

The sample was selected from patients included in a prospective trauma registry at a Level I Trauma Center in Texas. The records of all patients admitted to the Shock-Trauma Unit during 12 consecutive months (December 2004 to December 2005) with Abbreviated Injury Scale (AIS) score for head & neck >3 and CD9 codes for TBI were reviewed. The AIS (head & neck) >3 was chosen to ensure that all patients with an associated diagnosis of cerebral contusion would be captured. Patients were eligible if they met the following criteria: (i) the initial CT scan demonstrated a hemorrhagic cerebral contusion; (ii) no CT evidence of other intracranial hemorrhages; (iii) underwent a repeat CT within 24 hours of injury; (iv) did not undergo intracranial surgery before the time of the second scan; (v) were not irretrievably injured as defined by an AIS score of 6; and (vi) managed according to an adult protocol. The sample was restricted to those who did not undergo intracranial surgery before the second scan to better characterize the course and outcomes of small intracerebral contusions. Infants and young children were excluded as there is evidence that their clinical course and prognosis differ from adults. Permission to review the medical records was provided by the Institutional Review Board.

### TABLE 1. Summary of Results of Studies Examining Risk Factors for Intracranial Hemorrhage Progression

<table>
<thead>
<tr>
<th>Authors</th>
<th>Sample Description</th>
<th>Timing of CT Scans</th>
<th>Hemorrhage Progression*</th>
<th>Risk Factors (Unadjusted) for Hemorrhage Progression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bullock et al., 19904</td>
<td>N = 59 with postcraniotomy recurrent hemorrhage in sample of 850 TBI patients</td>
<td>Data not given</td>
<td>7%</td>
<td>Alcohol intake at time of injury; cerebral atrophy; coagulopathy</td>
</tr>
<tr>
<td>Stein et al., 199212</td>
<td>N = 253 TBI with serial CTs (review of medical records)</td>
<td>Scan 2 within 72 h of admission</td>
<td>48.6% (new or progressive lesions)</td>
<td>Presence of coagulopathy</td>
</tr>
<tr>
<td>Servadei et al., 199511</td>
<td>N = 37 comatose TBI (retrospective review of medical records)</td>
<td>Scan 2 within 12 h of injury</td>
<td>60%</td>
<td>Increased ICP; clinical deterioration; intracerebral and extradural hematomas more likely to progress</td>
</tr>
<tr>
<td>Givner et al., 20027</td>
<td>N = 104 pediatric TBI (review of the medical records)</td>
<td>Scan 2 about 27 h after scan 1</td>
<td>48%</td>
<td>Lower GCS (10.4 vs. 13.2); more likely to be intubated (46% vs. 17%)</td>
</tr>
<tr>
<td>Oertel et al., 20029</td>
<td>N = 142 TBI with intracranial hemorrhage (prospective cohort)</td>
<td>Scan 1, mean of 2 h after injury; scan 2, mean of 7 h after scan 1</td>
<td>51% (ICH)</td>
<td>Older age; earlier initial CT scan; longer PT; episode of hypotension</td>
</tr>
<tr>
<td>Sanus et al., 200410</td>
<td>N = 98 TBI of all severities (review of medical records)</td>
<td>Scan 2 within 48 h of injury</td>
<td>48% Progression of ICH—40.4%</td>
<td>Older age; increase in ICP; type of injury (intracerebral hemorrhage progressed more than other types of hemorrhage); worse ISS; coagulopathy; earlier initial CT scan</td>
</tr>
<tr>
<td>Chieregato et al., 20055</td>
<td>N = 141 TBI with traumatic subarachnoid hemorrhage (prospective cohort)</td>
<td>Scan 1, mean of 1.5 h from injury; scan 2, within 12 h of injury</td>
<td>38%</td>
<td>Initial size of hemorrhage</td>
</tr>
<tr>
<td>Beaumont and Gennarelli, 20063</td>
<td>N = 21 with TBI (review of medical records)</td>
<td>Interval between the two scans approximately 12 h</td>
<td>50%</td>
<td>Absence of perilesional edema on initial scan</td>
</tr>
<tr>
<td>Chang et al., 20065</td>
<td>N = 113 head trauma patients with intraparenchymal contusions (retrospective review of prospective registry)</td>
<td>Scan 1, mean of 2 h from injury; scan 2, mean of 17 h after scan 1</td>
<td>38% (ICH)</td>
<td>Effacement of sulci; effacement of cisterns; concurrent presence of SAH and/or SDH; larger initial size</td>
</tr>
</tbody>
</table>

ICH, intracerebral hemorrhage; ICP, intracranial pressure; SAH, subarachnoid hemorrhage; SDH, subdural hematoma.

* Refers to progression of intracranial progression and includes all subtypes of hemorrhage unless ICH directly stated. Please note that the definition of progression varies greatly across studies.

† Predictors related to injury progression in general and not specific to hemorrhage progression.
Procedures

Records were reviewed to retrieve data on the mechanism of injury, GCS recorded in the ER and at the time of each CT scan, ISS as recorded by the trauma team at time of admission, presence of systemic hypertension or hypotension, presence of antplatelet/anticoagulants at time of injury, platelet count, partial thromboplastin time (PTT), and international normalized ratio (INR). Systemic hypertension was defined as at least one episode of systolic blood pressure (SBP) >180 mm Hg in the period between admission and the second CT scan, and hypotension was defined as at least one episode of SBP <90 mm Hg in this same time period. Where multiple laboratory values were available, the initial result was recorded. Time of CT scanning relative to time of injury and the time between initial and the second scan was also recorded.

CT scans were performed on scanners with a 512 × 512 matrix with 5-mm slices. Repeat CT scans were obtained with the same standard imaging protocol. Clot size was measured using online digital ruler (Synapse PACS). Hemorrhage volume was calculated using the ABC/2 technique.\(^{17}\) This method demonstrates excellent interrater and intrarater reliability (intraclass correlation of 0.99 for both) and has been found to be highly correlated with the measurements made by computer-assisted plainmetric image analysis.\(^{17}\)

Contusion growth was defined as an increase of at least 33% from the initial volume as measured by image analysis on the second CT compared with the baseline CT scan. We used the conservative number of 33% for the same reasons described by Brott et al.\(^{18}\) A 33% change in the volume of a sphere in diameter is clearly visible to the naked eye of a physician viewing serial CT scans of patients with intraparenchymal hemorrhages. Second, from their preliminary measurement of serial CT scans, Brott et al.\(^{18}\) found that some patients, particularly those with small hemorrhages, had up to a third less volume of hemorrhage on follow-up CT. This was felt to be measurement error (i.e., different positioning) rather than a true decrease in hemorrhage volume. Therefore, a 33% definition of growth was more likely to represent true hemorrhage growth and not variability in measurement.

Data Analysis

To examine the relationship between potential risk factors and hemorrhagic contusion growth (as described earlier), univariate comparisons were performed using independent \(t\) tests for continuous variables and Fischer’s exact test for dichotomous variables. All tests were two sided, and a \(p\) value of \(<0.05\) was used for significance. A small increase in the volume of a small hemorrhage translates to a much higher percentage change compared with a larger one. Therefore, in addition to examining the percentage increase, absolute change in volume size was also examined. This provided a more complete characterization of the change between the initial and repeat CT scan. Finally, the relationship between the patient’s neurologic condition as measured by the GCS and hemorrhage growth was examined. Patients were grouped by change (no change or improvement versus deterioration) in GCS measured at the time of the two CT scans, and Fischer’s exact test was used to examine for significant differences between the groups stratified by progression. Those that scored 3 (the floor of the scale) at both time points were classified with the group that deteriorated. Regression analysis was used to look at the impact of increasing volume on the neurologic condition of the patient as measured by the GCS.

RESULTS

Of 660 TBI patients with AIS (head & neck) >3, 46 fulfilled all inclusion criteria and are included in the analyses reported here. The main reason for exclusion was the absence of an intracerebral contusion or multiple intracranial lesions on the initial CT scan. The average age of the sample was 38 years with a range from 11 years to 78 years, and the majority was male (82%). The mean GCS in the ER was 9 with 41% classified as severe (GCS ≤8), 9% moderate (GCS 9–13), and 50% mild (GCS 14–15). The mean ISS was 21 with a range from 9 to 43. The most common mechanism of injury was a motor vehicle crash followed by falls, which explains the relatively high ISS since most sustained multiple injuries. The interval from injury to first CT was, on average, 3 hours (SD 3.9 hours) with the follow-up scan occurring at a mean of 12 hours (SD 6.5 hours) after injury. As expected most contusions were cortical with the frontal and temporal lobes being equally affected.

Comparison between first and second CT scans demonstrated progression in absolute size of the contusion in 30 of the total sample (65%), 8 were unchanged (17%), and 8 (17%) reduced in size. Almost 15% of the sample experienced expansion in hemorrhage volume of >10 mL (Fig. 1). Defining progression by an increase in volume of at least 33% over baseline, two-thirds of the group progressed and for almost 50% of the sample, there was >100% increase in size of hemorrhage from the initial scan (Fig. 2). Figure 3 shows a representative case of intracerebral hemorrhagic progression on serial CT scans.

The sample was stratified by contusion growth, and the group with at least 33% growth was compared with those with <33% growth (including those with no growth or decrease in size). As can be seen in Table 2, timing of the CT scans relative to the injury was not significantly different.
between the groups. The average initial contusion size was not different by group. There was, however, a moderate correlation ($r = 0.44; p = 0.002$) between initial size and the absolute volume of growth indicating that, on average, larger baseline hemorrhages were associated with a larger volume of expansion. For the group that experienced progression of their hemorrhage, the mean volume increased from 2.6 mL (SD 4.5) at the time of the initial scan to 9.1 mL (SD 12.7) on repeat scan.

Variables known to influence, directly or indirectly, the coagulation cascade including the potential effect of alcohol,

\begin{table}
\centering
\caption{Patient Characteristics Stratified by Hemorrhage Growth}
\label{table:patient_characteristics}
\begin{tabular}{lcc}
\hline
\textbf{ } & \multicolumn{2}{c}{\textbf{\%33\% Growth (n = 30; 65\%)}} & \multicolumn{2}{c}{\textbf{<33\% Growth (n = 16; 35\%)}} \\
\hline
\textbf{Mean age (SD)} & 40 (18) & 35 (23) \\
\textbf{Mean hours (SD) to CT scan} & & \\
\textbf{Injury to scan 1} & 2.6 (3.2) & 3.8 (5.0) \\
\textbf{Injury to scan 2} & 12.1 (6.6) & 12.2 (6.6) \\
\textbf{Mean (SD) volume of contusion, mL} & & \\
\textbf{Volume at time of scan 1} & 2.6 (4.5) & 2.8 (4.5) \\
\textbf{Volume at time of scan 2} & 9.1 (12.7)* & 2.2 (4.9)* \\
\textbf{Elevated alcohol level on admission, n (\%)} & 15 (50\%) & 4 (25\%) \\
\textbf{Positive history of anti-coagulant use, n (\%)} & 3 (10\%) & 1 (6\%) \\
\textbf{Mean (SD) Hgb, g/dL} & 13.8 (2.6) & 13.3 (1.6) \\
\textbf{Mean (SD) PTT, s} & 29 (5) & 28 (4) \\
\textbf{Mean (SD) Platelets, 10^\text{3}/\mu L} & 221 (84) & 245 (65) \\
\textbf{Mean (SD) INR} & 1.4 (0.3)* & 1.2 (0.2)* \\
\textbf{Hypertension (SBP >180 mm Hg), n (\%)} & 7 (23\%) & 2 (13\%) \\
\textbf{Hypotension (SBP <90 mm Hg), n (\%)} & 7 (23\%) & 2 (13\%) \\
\textbf{Mean (SD) ISS} & 23 (9) & 19 (9) \\
\textbf{Mean (SD) GCS Admission} & 8 (6)* & 12 (5)* \\
\textbf{Time of scan 1} & 9 (5)* & 13 (3)* \\
\textbf{Time of scan 2} & 9 (5)* & 12 (5)* \\
\hline
\end{tabular}
\end{table}

\* Includes those with decrease in size of lesion or no growth

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure2}
\caption{Percentage change in hemorrhage volume between initial and second CT scan.}
\end{figure}

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure3}
\caption{Representative case of intracerebral hemorrhagic progression on serial CT scans.}
\end{figure}
prior use of antiplatelet/anticoagulation agents, clotting factors, and systolic hypertension and hypotension were examined. Although there was a trend for a history of elevated alcohol levels at admission in the group that experienced growth, this was not statistically significant. The incidence of international normalized ratio (INR) >1.3 in this cohort was 28%. The INR was significantly higher in the group that demonstrated growth compared with the group with <33% growth (1.4 compared with 1.2). Figure 4 presents boxplots (stratified by hemorrhage growth) of the INR values, showing their distribution in the two groups. The INR was dichotomized at the median (1.2) to examine the association between INR and hemorrhage progression. There was a trend with hemorrhage progression almost three times more likely in those with an INR above 1.2 compared with 1.2 or less (odds ratio [OR] = 2.87; 95% confidence interval [CI]: 0.80 to 10.40).

The average GCS was significantly lower at each point in time for the group that experienced at least a 33% increase in size of hemorrhage. An examination of the predictive value of the baseline GCS indicated that patients with a normal or near-normal GCS at baseline were significantly less likely to experience progression in the size of the hemorrhage (OR = 0.21; 95% CI: 0.05 to 0.81) compared with those with a GCS of <14. Furthermore, clinical deterioration, as measured by change in the GCS measured at the times of the two scans, was associated with hemorrhage growth on repeat CT scan. For those that showed deterioration on the GCS, there was a threefold risk of hemorrhage expansion being found on the CT as defined by percentage increase (OR = 3.43; 95% CI: 0.90 to 13.10) and similarly when defined as absolute increase in volume (OR = 3.32; 95% CI: 0.96 to 11.41). Regression analysis was used to examine the impact of increasing volume on neurologic function as defined by the GCS. Controlling for baseline GCS, for each cubic centimetre increase in volume difference between Time 1 and Time 2, there was an associated 0.2 point decrease in the GCS (p = 0.05). Finally, an increase in hemorrhage volume was associated with discharge destination. Of the sample, 57% were discharged directly home, 34% to inpatient rehabilitation, and 9% died during their hospital course. Discharge disposition was significantly different between the group with ≥1 mL increase in volume and the group with <1 mL volume increase (p = 0.03). Adjusting for baseline ISS, patients who experienced ≥1 mL increase in volume were 8% more likely to die in hospital compared with those with hemorrhage growth <1 mL (OR = 1.08; 95% CI: 0.97 to 1.20). Of those that died during hospitalization, all had progressive hemorrhagic lesions.

**DISCUSSION**

This study was undertaken to gain a better understanding of the rate of progression, predictors of hemorrhage expansion, and the clinical effects in a homogeneous sample of those with traumatic intracerebral contusion. Within this sample, 65% experienced early progression (within the first 24 hours postinjury) with 15% showing increases of >10 mL. Our results are consistent with previous findings that hemorrhagic cerebral contusions frequently progress.5,9,10 Progression has been found to be associated with the timing of the initial CT scan and the earlier the scan is performed relative to the injury, the more likely it is that the repeat scan will show progression.9,10 Although the timing of the initial scan in this study was earlier in the group that experienced progression (mean = 2.6 hours) compared with the group that did not experience progression (mean = 3.8 hours), these differences were not statistically significant. The very early timing of the initial scans in this study, earlier than previously reported in the literature, may have contributed to the higher percentage of hemorrhage progression seen in this sample. Furthermore, this sample included only those with intracerebral contusions, which have been observed to have a higher rate of expansion than other intracranial hemorrhages.10 Across studies, there is little consistency in the method of defining progression, and few studies have quantified the size of the lesion along with a clear definition of progression. We used the ABC/2 technique to quantify lesion size and then applied the definition of Brott et al.15 to define hemorrhage progression. Adopting a standard definition for progression of hemorrhage is an important step toward better characterizing the problem and identifying potential interventions. The quantification of lesion size has provided a clearer picture of the relationship among the initial volume, expansion, and clinical deterioration.

The findings from this study underscore important clinical points about traumatic contusions. The first relates to the role of coagulopathy in hemorrhage expansion. The incidence of coagulopathy (defined here as an INR >1.3) was 28%, similar to that found in a recently published meta-analysis of traumatic coagulation disorders after TBI, which reported an overall incidence of 32.7%.19 The frequen-
cies of coagulation disorders varied from 10% to 98% in those studies included in the meta-analysis. This wide variability relates to the differing definitions used to diagnosis a traumatic coagulopathy, the differing study designs, and patient populations. We found that coagulation abnormalities were a risk factor for hemorrhage expansion with a significantly higher INR at admission in the group with ≥33% hemorrhage progression (INR of 1.4) compared with the group with <33% expansion (INR of 1.2). This is consistent with the findings from other studies (see Table 1).4,9,10,12 Furthermore, traumatic coagulopathy has also been associated with a worse outcome.19–23 We are concerned that potential deleterious progression of intraparenchymal bleeding is associated with such a modest elevation in the INR. However, other studies using a similar definition of coagulopathy have also reported an association with hemorrhage expansion and unfavorable outcomes.10,23–26 It therefore seems reasonable to actively correct coagulation abnormalities in patients with INRs at this level to prevent expansion. The results of the phase two clinical trial conducted with rFVIIa demonstrated that it does inhibit expansion of posttraumatic hemorrhagic intracranial lesions.14 However, this study has not gone on to phase three trial to confirm clinical benefit related to the failure of rFVIIa to improve survival or functional outcome after spontaneous intracerebral hemorrhage.

Second, our results demonstrated that even a small increase in volume size is associated with clinical deterioration. We studied a group with intracerebral contusions who, based on the size of the lesion, were not felt to be surgical candidates in the early postinjury phase. Despite their size, these lesions progressed at a high rate with consequent negative clinical outcomes. The increase in volume, whether defined by percentage increase or by absolute volume increase, was associated with a decrease in the GCS. Although not statistically significant, irrespective of the initial size of the hemorrhage and controlling for baseline ISS, those with expansion of at least 1 mL were 1.08 times more likely to die during hospitalization than those with less than a 1 mL volume increase.

Third, by restricting our sample to those that underwent a second CT scan within 24 hours, we have been able to better define the time course of progression and the therapeutic window for intervention. The mean time to second scan in our cohort was 12 ± 6 hours. The second scan in the study by Chang et al.5 was performed, on average, at 16 hours after injury and in the rFVIIa phase 2 trial was performed at 24 ± 3 hours.14 The findings here provide supportive evidence on the very early evolution of the hemorrhagic lesion, earlier than has been previously reported.

Finally, those with normal or near-normal baseline GCS were significantly less likely to show progression on repeat CT scan compared with those with baseline GCS < 14. Several studies have been designed to specifically examine serial CT scans in the context of TBI and to better define the indications for follow-up imaging.16,28–31 Unfortunately, previous studies have varied in their conclusions, and there is not a consensus regarding indications for serial scanning. The majority of these studies, however, has concluded that repeat CT may not be indicated in patients with normal neurologic examination or in the absence of clinical indicators. Their conclusion was based on the fact that the repeat scan did not seem to influence patient management.

There are several limitations to this study, which suggest caution in interpreting the results. The sample is small and therefore some findings could be by chance. The sample size also limited multivariable analyses, which are needed to build a model of risk. At the time when this study was conducted, a standard procedure regarding serial scanning was not in place, which could have led to selection bias in this study. Those patients who underwent serial CT scans within 24 hours, which was one criterion for inclusion, may in fact have been those patients where the clinician had good reason to suspect expansion of the lesion.

TBI represents an enormous personal and socioeconomic problem. It is estimated that ~1.4 million Americans suffer a TBI every year, with an estimated 90,000 persons experiencing long-term disabilities.32 The progression of a traumatic hemorrhage is only the beginning of a sequence of biochemical events, which leads to further neuronal damage and clinical deterioration. Secondary injury is responsible for prolonged hospital stay and increased morbidity and mortality.15 What are the practical implications of our research findings for the care provided to those with traumatic intracerebral contusion? To date, there is no recommended treatment for the prevention of lesion expansion. Our results have provided further evidence that these hemorrhagic cerebral contusions evolve rapidly in time and they are associated with a coagulopathy. We thus have more precise information about the optimal time for intervention. Based on the relationship between admission INR and progression, careful monitoring and correction of coagulation parameters is critical to prevent further injury. The findings here highlight the need for clinical studies to better define therapeutic interventions to prevent irreversible neurologic injury.

REFERENCES
The authors present a retrospective analysis of a prospective trauma registry from a single Level I trauma center designed to assess the prevalence, risk factors, and outcome of the progression of traumatic intracerebral contusions in patients with traumatic brain injury (TBI). The study sample was selected from a representative 12-month period in the database, by including all adult trauma patients with an Abbreviated Injury Scale score > 3 for the anatomic region head/neck (n = 660) and the presence of intracerebral contusion on a computed tomography scan. A total of 46 patients with head injuries were included in the final analysis. The authors found that the international normalized ratio (INR) was significantly increased in the group that demonstrated progression of intracerebral contusions, when compared with patients with “stable” hematomas. These data imply a higher risk for secondary deterioration of traumatic intracerebral contusions in the presence of coagulopathy.

Although these findings may not be surprising per se—clearly, a decreased ability to form a clot will lead to an increased risk of bleeding—this study sheds some further light on a timely topic of ongoing research and debate. Although the first description of TBI-induced coagulopathy dates back almost 50 years, increased scientific interest in the field has emerged only recently. Several groups reported a significantly increased mortality in coagulopathic patients with head injuries and an impressive incidence of TBI-associated coagulopathy of about 35%. This study provides further knowledge regarding the selected group of patients with traumatic intracerebral contusions, by characterizing coagulopathy as an independent risk factor of hematoma progression within 24 hours. The incidence of coagulopathy in this article was 28%, as defined by an INR > 1.3. Impressively, coagulopathic TBI patients had a threefold increased likelihood of intracranial hemorrhage progression, when compared with the group with an INR ≤ 1.2.

Based on the high incidence and poor outcome associated with coagulopathy in TBI, modified therapeutic strategies are currently appearing on the horizon. Among these, the “off-label” use of recombinant factor VIIa (rF-VIIa) appears most promising because of the potent hemostatic properties of rFVIIa and the pervasive presence of its ligand, tissue factor, in the brain. A recent dose-escalation trial revealed that rFVIIa inhibits the progression of intracranial hematomas in patients with TBI. The risk-benefit ratio and the questionable prophylactic administration of rFVIIa to noncoagulopathic patients with severe TBI require further investigation in well-designed prospective randomized clinical trials.

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