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Moderate elevations in international normalized ratio should not lead to delays in neurosurgical intervention in patients with traumatic brain injury

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Abstract

BACKGROUND—The management of severe traumatic brain injury (TBI) frequently involves invasive intracranial monitoring or cranial surgery. In our institution, intracranial procedures are often deferred until an international normalized ratio (INR) of less than 1.4 is achieved. There is no evidence that a moderately elevated INR is associated with increased risk of bleeding in patients undergoing neurosurgical intervention (NI). Thrombelastography (TEG) provides a functional assessment of clotting and has been shown to better predict clinically relevant coagulopathy compared with INR. We hypothesized that in patients with TBI, an elevated INR would result in increased time to NI and would not be associated with coagulation abnormalities based on TEG.

METHODS—A secondary analysis of prospectively collected data was performed in trauma patients with intracranial hemorrhage that underwent NI (defined as cranial surgery or intracranial pressure monitoring) within 24 hours of arrival. Time from admission to NI was recorded. TEG and routine coagulation assays were obtained at admission. Patients were considered hypocoagulable based on INR if their admission INR was greater than 1.4 (high INR). Manufacturer-specified values were used to determine hypocoagulability for each TEG variable.

RESULTS—Sixty-one patients (median head Abbreviated Injury Scale [AIS] score, 5) met entry criteria, of whom 16% had high INR. Demographic, physiologic, and injury scoring data were similar between groups. The median time to NI was longer in patients with high INR (358 minutes vs. 184 minutes, $p = 0.027$). High-INR patients were transfused more plasma than patients with an

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AUTHORSHIP

S.E.R., R.R.B., A.J.R., and M.A.S. contributed to the study concept and design. T.C.L., K.A.F., S.J.U., and S.E.R. acquired data. S.E.R., R.R.B., T.C.L., and K.A.F. analyzed and interpreted the data. S.E.R., R.R.B., and T.C.L. drafted the manuscript. S.E.R., R.R.B., T.C.L., K.A.F., A.J.R., and M.A.S. critically revised the manuscript for important intellectual content. S.E.R. obtained funding.

INR of 1.4 or less (2 U vs. 0 U, $p = 0.01$). There was no association between an elevated INR and hypocoagulability based on TEG.

CONCLUSION—TBI patients with an admission INR of greater than 1.4 had a longer time to NI. The use of plasma transfusion to decrease the INR may have contributed to this delay. A moderately elevated INR was not associated with coagulation abnormalities based on TEG. Routine plasma transfusion to correct a moderately elevated INR before NI should be reexamined.

LEVEL OF EVIDENCE—Diagnostic study, level III.

Keywords

Traumatic brain injury; thrombelastography; time to intervention; coagulopathy

Severe traumatic brain injury (TBI) causes significant morbidity and mortality in the United States and worldwide.[1,2] Surgical intervention is recommended for traumatic subdural,[3] epidural,[4] and intraparenchymal hematomas[5] that meet specific anatomic and physiologic criteria. Invasive intracranial pressure (ICP) monitoring is also commonly used in patients with severe TBI, especially those managed nonoperatively. Specific recommendations for the use of ICP monitoring have been outlined in national guidelines published in the United States[6] and the United Kingdom,[7] although its ongoing use has been questioned.[8,9] Compliance with Brain Trauma Foundation guidelines is often inconsistent for a variety of reasons.[10,11]

Neurosurgical intervention in TBI patients with an elevated international normalized ratio (INR) is often deferred until the INR has been corrected.[11] A variety of cutoff values are used, and no standard value has been universally adopted. Neurosurgical textbooks have recommended that patients undergoing invasive procedures have an INR less than an arbitrary number, often 1.4.[12,13] Plasma transfusions are often given to achieve this, leading to potential delays in neurosurgical intervention, both at our institution and at other centers.[14] This approach has been based largely on dogma and has only been directly examined in one study.[15]

A moderate elevation in INR is known to be associated with only a modest deficiency in clotting factors that may be clinically insignificant.[16] There is a lack of evidence to support the clinical benefit of prophylactic plasma transfusion to correct the INR in neurosurgical patients[17] and patients undergoing a variety of other invasive procedures.[16] Furthermore, plasma transfusion may not even be effective at lowering the INR in patients with mild elevations.[18]

TEG was first introduced in 1948 and is a rapid point-of-care test that measures the viscoelastic properties of whole blood from clot initiation to lysis and has been used in a wide variety of clinical fields.[19] TEG may provide a better assessment of coagulation in injured patients compared with routine coagulation assays such as INR.[20] Although TEG has replaced INR for initial evaluation of coagulation at a few trauma centers, it has not yet become widely available for clinical use.[21–23]

We hypothesized that in patients undergoing neurosurgical intervention (NI) after TBI, an elevated INR would result in increased time to NI and would not be associated with coagulation abnormalities based on TEG.

PATIENTS AND METHODS

The study was approved by the institutional review board at Oregon Health and Science University. A retrospective analysis of prospectively collected data was performed. Adult patients (>15 years) admitted from March 2010 to January 2013 with a traumatic intracranial hemorrhage (ICH) from a blunt mechanism that subsequently underwent NI were included in the study. Patients that received at least 1 U of red blood cells within 6 hours of arrival for non–central nervous system injuries or that received recombinant factor VIIa were excluded. Additional exclusion criteria included pregnancy, warfarin or clopidogrel use, and the presence of a known coagulation disorder.

Patient characteristics including demographics, admission physiologic and laboratory values, Abbreviated Injury Scale (AIS) scores and Injury Severity Score (ISS) were recorded. NI was defined as the performance of a craniotomy or craniectomy or the placement of an invasive ICP monitoring device (generally an intraparenchymal monitor or ventriculostomy) within 24 hours of admission. The specific interventions and time to intervention were recorded.

TEG was performed by dedicated research personnel with standardized training. The TEG 5000 machine (TEG, Hemoscope, Niles, IL) underwent quality control measures every other day as per manufacturer guidelines. Fresh whole-blood specimens in kaolin-activated cups were used. Standard TEG measurements, including *R* value, *K* value, maximum amplitude (MA), α angle, and LY30 were obtained. Manufacturer reference ranges were used to define hypocoagulability (Table 1). TEG was generally performed as soon as possible after arrival and before NI although this was not feasible in all cases, either because of clinical circumstances or the timely availability of a research coordinator. For analyses involving TEG parameters, only those patients that had a TEG performed within 6 hours of arrival and before NI were included.

Patients were defined as hypocoagulable by INR (high INR) if the admission INR was greater than 1.4. This value was selected because neurosurgeons at our institution typically require an INR of 1.4 or less before performing an NI. Patients were defined as hypocoagulable by TEG if one or more TEG variables met the following manufacturer-specified parameters: *R* greater than 9 minutes, *K* greater than 3 minutes, α angle less than 59 degrees, MA less than 55 mm, and LY30 greater than 8 %. The coagulation index (CI), a calculated value that takes into account the relative contributions of all TEG variables, was also determined. Patients with CI less than −3.0 were considered hypocoagulable.

TBI patients admitted to the trauma intensive care unit at our institution are comanaged by the trauma and neurosurgical services. The decision to transfuse plasma before NI was primarily made by the neurosurgical team. Results of routine coagulation assays were available to treating physicians, but TEG data were not.

Patient data were deidentified and were maintained in Microsoft Excel (Microsoft, Redmond, WA). Statistical analyses were performed using Stata 12 (StataCorp., College Station, TX). Categorical data were analyzed using the χ^2 test, and continuous variables were analyzed using the Mann-Whitney U-test. Median and interquartile ranges (IQRs) were determined for nonparametric data. Significance was defined as $p \leq 0.05$.

RESULTS

The database included 61 patients with traumatic ICH that underwent NI. All patients had a blunt mechanism of injury. Admission demographic, injury scoring, physiologic, and routine laboratory data are shown in Tables 2 and 3. Median head AIS score was 5 (IQR, 4–5). The specific types of ICHs are listed in Table 4. Ten patients (16%) had an INR greater than 1.4 (high INR) at admission and 51 (84%) had an INR of 1.4 or less (low INR). Median INR in the high-INR group was 1.7 (1.5–1.7), and the highest INR in the group was 2.06.

Overall, the median time to NI was 231 minutes (96–363 min). In the high-INR group, the median time to NI was 358 minutes (285–478 minutes), and in the low-INR group, it was 184 minutes (87–343 minutes, $p = 0.027$). NIs performed are shown in Table 5. Patients in the high-INR group showed a trend toward being more likely to receive a monitoring device as the sole NI ($p = 0.091$).

Overall mortality was 21%. Mortality was 50% in the high-INR group and 16% in the low-INR group ($p = 0.015$). Of the 13 patients that died, 9 had support withdrawn because of devastating brain injury, 3 progressed to brain death, and 1 died of refractory shock. Patients with a high INR were more likely than patients with a low INR to receive a plasma transfusion (70% vs. 24%, $p = 0.004$). The median number of units of plasma transfused was also higher in the high-INR group (2 U [0–6 U] vs. 0 U [0–1 U], $p = 0.006$). There were no significant differences between the high-INR and low-INR groups in the likelihood or number of red blood cell or platelet transfusions.

TEG was performed in 46 patients within 6 hours of admission and before NI. The mean (SD) time to initial TEG was 68 (60) minutes after admission. TEG values are shown in Table 6. Eighteen patients (39%) had one or more TEG variables on the hypocoagulable side of the reference range. There was no association between a high INR and any individual abnormal TEG parameter, analyzed either as continuous or dichotomous variables. An abnormally low CI (CI < −3.0) was also not correlated with a high INR ($p = 0.169$).

DISCUSSION

A significant proportion of patients with TBI present with an elevated INR, partial thromboplastin time, or low platelet count.[24] In these patients, NI is often postponed until plasma transfusions are given in an attempt to decrease the INR below an arbitrary number, often 1.4.[12,13] This paradigm exists in large part because of expert opinion and established practice patterns. It has rarely been formally studied. In 2004, Davis et al.[15] published a retrospective review of patients that had an ICP monitor placed and stratified patients by admission INR. An INR of 1.3 to 1.6 was defined as “borderline,” while an INR of 1.7 or greater was defined as “elevated.” Among the six patients with a borderline INR who

received plasma transfusion, only one had a decrease in INR to the normal range. Similarly, among 13 patients with an elevated INR who underwent plasma transfusion, 1 corrected to a normal INR, 5 corrected to a borderline range, and 7 remained uncorrected. Only 1 of the 42 patients with INR between 1.3 and 1.6 and 1 of the 12 patients with an INR of 1.7 or greater had a hemorrhagic complication after ICP monitor placement. Both of the hemorrhages were 2 mm or less in size, and neither was clinically significant. The authors noted that plasma transfusions led to an increase in time to ICP monitor placement and concluded that correction of an INR in the range of 1.3 to 1.6 is not necessary. They did not speculate on the safety of placing ICP monitors in patients with an INR of 1.7 or greater. In our study, we found that the time interval between admission and NI in patients with an INR greater than 1.4 was almost twice as long as patients with an INR less than 1.4. While we cannot conclusively determine the causes of this time difference, all but one patient in the high-INR group received plasma before NI, whereas only one patient in the low-INR group received plasma before NI. Based on the general consensus of the neurosurgeons at our institution and the routine adherence to the practice guideline recommending an INR of less than 1.4 before NI, it is likely that patients in the high-INR group had a longer time to NI to receive plasma transfusions, Furthermore, based on the existing neurosurgical literature, it is likely that this practice at our hospital is reflective of many centers across the country.

Most of our patients with high INR had only moderate elevations (median INR, 1.68), and no patient had an INR of greater than 2.1. With contemporary reagents and laboratory equipment, an INR of 1.7 is associated with clotting factor concentrations that are approximately 30% of the normal, which is generally considered to be adequate for hemostasis.[16,25] Furthermore, it has been noted that plasma transfusions intended for correction of a moderately elevated INR (1.3–1.7) usually do not lead to a significant decrease in INR.[15,16,18,26] In one study, patients transfused plasma for an INR in the range of 1.1 to 1.8 corrected to the normal range less than 1% of the time, and only 15% had a correction halfway to the normal range.[26]

In recent years, the practice of correcting a moderately elevated INR before surgical or other procedural interventions has been increasingly reexamined. In 2005, Segal and Dzik[27] performed a comprehensive review of the literature examining the impact of abnormal coagulation test results on periprocedural bleeding. Twenty-five studies examining a variety of procedures, such as bronchoscopy, liver biopsy, renal biopsy, femoral angiography, and central vein cannulation, met criteria to be included in the analysis. None demonstrated a significantly increased risk of complications in patients with a moderately elevated INR. The authors concluded that an elevated INR is not predictive of periprocedural bleeding.

Determining the risk of increased bleeding in patients with a mild-to-moderate coagulopathy undergoing a neurosurgical procedure is difficult. Virtually all reports describe a series of elective procedures that are rarely performed in the presence of known coagulopathy. A risk of postoperative hemorrhage of 0.8% was reported by Kalfas and Little[28] in a series of 4,992 elective intracranial operations. However, the risk of bleeding secondary to coagulopathy after a neurosurgical procedure for trauma is much more difficult to quantify in part because the evolution of existing hemorrhage and the development of new bleeding foci are common after injury, even in patients with normal coagulation parameters.

INR was originally developed to assess adequacy of dosing of vitamin K antagonists and examines only a small portion of a complex system of coagulation pathways.[19] It was not designed to evaluate coagulopathy in acutely bleeding patients or to predict patients that are at increased risk for bleeding after procedures.[25–27] In contrast, TEG provides more detailed information related to overall coagulation and provides information on the kinetics of clot initiation and growth, the maximum clot strength, and the fibrinolytic breakdown of clot.[29] TEG has been shown to be a better predictor of clinically significant bleeding in trauma patients and patients undergoing elective surgery compared with routine coagulation assays.[30,31] However, there are limited data regarding the use of TEG in patients with TBI, and no studies have directly demonstrated an association between abnormal TEG values and increased intracranial bleeding.

In our study, TEG was performed before NI in 46 of 61 patients. Thirty-nine percent of the patients had one or more TEG variables on the hypocoagulable side of the reference range; however, we found no association between a high INR and any individual abnormal TEG parameter. Since we did not examine bleeding complications in this study, we cannot speculate whether TEG is superior to INR for assessing coagulation status before NI. However, unpublished data from our laboratory suggest that the MA may be superior to routine coagulation assays for the prediction of progression of ICH. Furthermore, since TEG is a point-of-care test that can be rapidly performed, results may be available for clinical decision making much faster than for INR. While we are hopeful that these data can be used by the neurosurgical community as a basis for reevaluating existing clinical practice guidelines related to the correction of coagulation abnormalities, they are preliminary in nature, and additional study is needed in patients with TBI to determine if TEG is superior to routine coagulation assays for the prediction of bleeding complications, including in patients undergoing NI.

Our study is subject to the usual limitations of a retrospective analysis of prospectively collected data. In addition, not all patients were included in the TEG analysis since not all TEG studies were obtained in less than 6 hours or before NI. Moreover, we did not attempt to determine the clinical reasoning behind the timing of NI in each case. Accordingly, we could not quantify the degree to which NI was delayed in individual high-INR patients for plasma transfusions to occur. Finally, we did not examine complications potentially related to plasma transfusion.

CONCLUSION

Patients with TBI and an admission INR of greater than 1.4 who underwent NI had a longer time to intervention. The practice of preintervention transfusion of plasma to decrease the INR may have contributed to this delay. In this population, a moderately elevated INR was not associated with coagulation abnormalities as determined by TEG. The practice of routine plasma transfusion to correct a moderately elevated INR before NI should be reexamined.

Acknowledgments

DISCLOSURE

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TABLE 1

TEG Parameters Consistent With Hypocoagulability

Variable	Patients, n (%)*
R > 9 min	5 (11)
K > 3 min	7 (15)
α angle < 59°	13 (28)
MA < 55 mm	9 (20)
LY30 > 8%	1 (2)
Any abnormality	18 (39)

*Some patients had more than one abnormal TEG parameter.

TABLE 2

Admission Demographics and Injury Scoring Data for Patients Undergoing NI for TBI

Variable	All Patients	High INR	Low INR	*p*
Age, y	48 (30–59)	59 (21–70)	48 (30–57)	0.483
Sex, male, %	79	80	78	0.912
%EtOH positive	40	20	44	0.157
%prehospital ASA	16	10	18	0.550
ISS	30 (25–38)	35 (26–50)	29 (25–38)	0.165
Head AIS score	5 (4–5)	5 (4–5)	5 (4–5)	0.945
Face AIS score	1 (0Y2)	2 (0–3)	0 (0–2)	0.089
Chest AIS score	0 (0–3)	1.5 (0–4)	0 (0–3)	0.171
Abdomen AIS score	0 (0–0)	0 (0–0)	0 (0–0)	0.474
Extremity AIS score	0 (0–2)	2 (0–3)	0 (0–2)	0.095

TABLE 3

Admission Physiologic and Biochemical Data for Patients Undergoing NI for TBI

Variable	All Patients	High INR	Low INR	*p*
Temperature, °C	36.2 (0.96)	35.9 (1.2)	36.3 (0.9)	0.273
Hear rate, beats/min	99 (29)	116 (27)	95 (29)	0.027
Systolic blood pressure, mm Hg	143 (30)	126 (30)	146 (29)	0.069
Mean arterial pressure, mm Hg	105 (25)	94 (21)	107 (26)	0.166
Sodium, mEq/L	139 (5)	140 (4)	139 (5)	0.523
Glucose, mg/dL	151 (46)	164 (69)	148 (41)	0.717
Creatinine, mg/dL	0.94 (0.28)	1.06 (0.44)	0.92 (0.25)	0.581
pH	7.32 (0.14)	7.30 (0.07)	7.32 (0.15)	0.149
Base deficit, mEq/L	−4.0 (5.3)	−6.1 (2.5)	−3.6 (5.5)	0.063
INR	1.1 (1.1–1.3)	1.7 (1.5–1.7)	1.1 (1.05–1.2)	<0.001
Partial thromboplastin time, s	31.8 (22.0)	55.5 (52.8)	27.7 (4.6)	0.004
Platelets, $10^3/\mu L$	234 (74)	196 (78)	241 (72)	0.070
Fibrinogen, mg/dL	262 (102)	211 (149)	271 (90)	0.070
Glasgow Coma Scale (GCS) score	8 (4–14)	9 (6–13)	8 (4–14)	0.976

TABLE 4

Types of ICH in Patients Undergoing NI

Hemorrhage Type	High INR, n = 10	Low INR, n = 51
Subarachnoid, n (%)	7 (70)	40 (78)
Intraparenchymal, n (%)	6 (60)	39 (76)
Subdural, n (%)	7 (70)	30 (59)
Epidural, n (%)	1 (10)	9 (18)

TABLE 5

NIs Performed in Patients Initially Presenting With High Versus low INR

Initial INR	Operation Only*	Monitor Only**	Both
High INR (>1.4)	2	8	0
Low INR (≤1.4)	12	26	13†

*Craniotomy or craniectomy.

**ICP monitor or ventriculostomy placement.

†Performed as separate procedures in five patients; time to the first procedure was used for calculations.

TABLE 6

Initial TEG Values In Patients Undergoing NI for TBI With High Versus Low INR

Variable	All Patients	High INR	Low INR	*p*
R, min	4.9 (3.8 to 6.4)	6.1 (3.8 to 6.6)	4.9 (3.8 to 6.4)	0.643
K, min	1.8 (1.3 to 2.6)	1.8 (1.2 to 3.4)	1.8 (1.3 to 2.5)	0.818
α angle, degree	65 (58 to 72)	61 (52 to 70)	66 (59 to 72)	0.543
MA, mm	63 (60 to 71)	60 (51 to 68)	64 (60 to 71)	0.229
LY30, %	0.2 (0 to 0.7)	0.15 (0 to 0.45)	0.2 (0 to 0.8)	0.876
CI	0.8 (−1.4 to 2.6)	−1.3 (−4.8 to −0.2)	1.1 (−0.8 to 2.6)	0.153