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ORIGINAL RESEARCH

Acute Trauma Factor Associations With Suicidality Across the First 5 Years After Traumatic Brain Injury

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Abstract

Objective: To determine whether severity of head and extracranial injuries (ECI) is associated with suicidal ideation (SI) or suicide attempt (SA) after traumatic brain injury (TBI).

Design: Factors associated with SI and SA were assessed in this inception cohort study using data collected 1, 2, and 5 years post-TBI from the National Trauma Data Bank and Traumatic Brain Injury Model Systems (TBIMS) databases.

Setting: Level I trauma centers, inpatient rehabilitation centers, and the community.

Participants: Participants with TBI from 15 TBIMS Centers with linked National Trauma Data Bank trauma data (N=3575).

Interventions: Not applicable.

Main Outcome Measures: SI was measured via the Patient Health Questionnaire 9 (question 9). SA in the last year was assessed via interview. ECI was measured by the Injury Severity Scale (nonhead) and categorized as none, mild, moderate, or severe.

Results: There were 293 (8.2%) participants who had SI without SA and 109 (3.0%) who had SA at least once in the first 5 years postinjury. Random effects logit modeling showed a higher likelihood of SI when ECI was severe (odds ratio=2.73; 95% confidence interval, 1.55−4.82; *P*=.001). Drug use at time of injury was also associated with SI (odds ratio=1.69; 95% confidence interval, 1.11−2.86; *P*=.015). Severity of ECI was not associated with SA.

Conclusions: Severe ECI carried a nearly 3-fold increase in the odds of SI after TBI, but it was not related to SA. Head injury severity and less severe ECI were not associated with SI or SA. These findings warrant additional work to identify factors associated with severe ECI that make individuals more susceptible to SI after TBI.

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Individuals with traumatic brain injury (TBI) are 3 to 4 times more likely to die as a result of suicide compared with the general population.[1] TBI survivors also have a high rate of suicidal ideation (SI), which is known to carry a 5-fold increase in the risk of suicide attempt (SA), and these risks persist for up to 15 years after TBI.[2,3] Many of the risk factors for suicidality (a broader term used to incorporate both SI and SA) in noninjured populations overlap with chronic sequelae of TBI, including aggression, poor cognitive inhibition, poor problem-solving, reduced ability to benefit from predominantly verbal counseling interventions, impulsive behavior, substance abuse, and psychiatric disorders (eg, major depression).[3-7] Veterans with the polytrauma clinical triad of posttraumatic stress disorder, TBI, and chronic pain have significantly increased odds of suicidality, particularly in the setting of substance abuse.[8] Although chronic pain is common after TBI, its role in both SI and SA seems to be related more to an individual's perception or acceptance of pain, rather than pain severity,[9] which is in turn associated with disability,[10] an additional known risk factor for suicidality.[11,12] Severity of TBI has not been linked consistently to depression or risk of suicidality,[7,13,14] but other injury-related factors (eg, severity of extracranial injury [ECI]) that may lead to both increased distress and greater disability have not been assessed in previous studies.

March et al[15] reported that adults with unintentional major traumatic injuries, indicated by total Injury Severity Scores (ISSs) >12 (combined head and extracranial ISS), have >4 times the risk of SA or completed suicide compared with the general population, even after adjusting for psychiatric conditions (anxiety/mood disorders and substance abuse), physical comorbidities, and other psychosocial factors (income and residence). Similarly, those with life-threatening physical illness (eg, TBI, stroke, myocardial infarction, spinal cord injury [SCI]) have higher rates of SI (11.3%) developing up to 2 years after illness onset, compared with the general population.[16] Ryb et al[12] suggest that higher suicide rates among individuals who are traumatically injured compared with the general population may be caused primarily by a higher prevalence of alcohol abuse in those with traumatic injuries.

The goal of the study was to determine whether severity of head and ECIs were associated with SI or SA after TBI. We hypothesized that (1) severity of head injury would not have a significant association with SI or SA; (2) severity of ECI would be associated with and predictive of SI and SA; and (3) physical disability and substance abuse would be associated with an increased risk of SI and SA.

Methods

Participants and measures

Participants were recruited as part of the 20-site Traumatic Brain Injury Model Systems (TBIMS) National Database longitudinal study, the longest standing longitudinal database of TBI outcomes (currently with >25 years of data collected). This database has the following inclusion criteria: ≥16 years old and evidence of a moderate to severe TBI (defined as any one of the following: Glasgow Coma Scale score <13, loss of consciousness >30min, posttraumatic amnesia >24h, and/or trauma-related intracranial abnormality). Informed consent was provided by either participant or proxy as necessary, and all procedures were approved by each center's institutional review board. Information on data collection has been described previously[17] and can be found online (https://www.tbindsc.org/). Acute care data obtained from the National Traumatic Data Base, the largest aggregation of trauma registry data (https://www.ntdbdatacenter.com/), were linked to the TBIMS National Database (data from 15 TBIMS Centers) through a probabilistic linkage based on common data elements across the TBIMS and National Trauma Data Bank databases using methods described previously.[18] Demographic, premorbid, clinical, mood, suicidality, functional impairment, and other outcome data collected acutely (National Trauma Data Bank and TBIMS National Database) and at 1, 2, and 5 years postinjury (TBIMS National Database) were used for analyses. The specific measures collected and their sources (TBIMS National Database vs National Trauma Data Bank) are summarized in table 1.

For the primary variables of interest, question 9 on the Patient Health Questionnaire-9 was used as a measurement of SI because this item is a direct scale question regarding how frequently respondents have had thoughts of hurting themselves or that they would be better off dead. This specific item captures suicidal thoughts and has been found to be associated with increased risk for SA.[19] Those endorsing a score ≥1 were categorized as positive for SI. SA in the last year was assessed via interview conducted as part of regular follow-up evaluation per the TBIMS protocol. Head injury severity was measured with the maximum Abbreviated Injury Scale head score. Injuries are ranked on the Abbreviated Injury Scale from 1 to 6, with increasing severity. The ISS is derived from the sum of the square of the Abbreviated Injury Scale in the 3 most severely injured body regions.[20,21] ECI was measured by the ISS (3 most severely injured body regions, excluding the head) and categorized as none (0), mild (1−8), moderate (9−15), or severe (>15).

Data analysis

We conducted an initial analysis comparing baseline demographic and injury characteristics of participants who reported no SI at all follow-up time points with participants with SI, but no SA, at any follow-up time point (1, 2, and/or 5y postinjury) and with those participants reporting SA at any follow-up time point. We used random effects models to identify predictors and associated factors of SI and SA across the first 5 years post-TBI, including both head and ECI severity, motor and cognitive disability, and substance abuse measures. This analytical approach controls for intrasubject correlation associated with repeated measures, while allowing for inclusion of multiple time points for covariates and outcomes. Coefficients can be exponentiated, allowing calculation of odds ratios for individual covariates across time. We first developed base models that included relevant demographic, acute trauma, and follow-up variables. We then added head and ECI severity to the base models, allowing us to conduct a likelihood ratio test to determine if the addition of these acute injury characteristics significantly improved model fit. Analysis was conducted with Stata version 13.[a] The *P* values ≤.05 were considered statistically significant.

List of abbreviations:

ECI	**extracranial injury**
ISS	**Injury Severity Score**
SA	**suicide attempt**
SCI	**spinal cord injury**
SI	**suicidal ideation**
TBI	**traumatic brain injury**
TBIMS	**Traumatic Brain Injury Model Systems**

Table 1 Description of measures

Measures	Construct	Summary of Measure
NTDB measures		
AIS/ISS	Head injury severity Nonhead injury severity	➢ An anatomic trauma severity scale that quantifies overall severity of injury across multiple body regions.[22] ➢ Injuries are ranked on a scale of 1–6, with 1 being minor and 6 being an unsurvivable injury. ➢ ISS scores are derived from the AIS, calculated as the sum of the square of the 3 most severely injured body regions.[23] ➢ The most severe head injury was taken as maximum AIS head score. ➢ Extracranial ISS was calculated as the sum of the square of the 3 most severely injured extracranial body regions.
GCS	Head injury severity	➢ Most commonly used tool of acute neurologic injury severity. ➢ Focuses on verbal response, eye opening, and motor response.[24] ➢ Scores range from 3–15, with 3 being most severe and 15 being most mild.
Infectious complications	Infectious burden	➢ Coded based on presence of deep wound infection, urinary tract infection, or pneumonia during acute hospital stay.
Alcohol blood level	Alcohol use at time of injury	➢ Blood alcohol level was measured through blood draw at the acute facility and is expressed as a concentration of grams per deciliter.
Drug use at time of injury	Drug use at time of injury	➢ Drug use at time of injury was determined with blood draw at the acute facility.
Mechanism of injury	Intent	➢ Mechanism of injury caused by accident, assault, or self-inflicted.
TBIMS measures		
Years of education	Education	➢ Education level at the time of injury. ➢ Coded binary with high school education or greater or less than high school level education.
Race	Race	➢ Race was categorized as white, black, Asian, Native American, and other.
Payor source	Health care resources	➢ Payor was dichotomized as Medicare and Medicaid vs all others.
FIM	Motor disability Cognitive disability	➢ The FIM is the most widely accepted and used measure of functional recovery during inpatient rehabilitation and after community integration.[25] ➢ An 18-item ordinal scale assessing level of physical, motor, and cognitive disability. ➢ The cognitive and motor subscores at follow-up were used in this study.
PHQ-9	Depressive symptoms	➢ A 9-item ordinal screening questionnaire based on the *Diagnostic and Statistical Manual of Mental Disorders, 4th edition* criteria for major depressive disorder. ➢ Has been validated for use in populations with TBI.[26] ➢ The PHQ-8 scoring (questions 1–8 only) was used in this study as a measure of depressive symptoms to predict suicidality.
Time to follow commands (d)	Injury severity	➢ Refers to the date after injury that a patient is able to follow motor commands. ➢ The individual must either follow simple motor commands accurately at least 2 out of 2 times within a 24-hour period or have a score of 6 on the motor component of the Glasgow Coma Scale.
Substance abuse at time of follow-up	Substance abuse at follow-up	➢ Participant is asked the following question at the time of follow-up: During the last 12mo, did you use any illicit or nonprescription drugs?
Preinjury SA	Preinjury SA	➢ Participant is asked the following question at the time of enrollment into the study: Have you ever attempted suicide?
Outcome measures		
PHQ-9, question 9	SI	➢ PHQ-9 question 9 assesses SI and asks, over the last 2wk, how often have they been bothered by thoughts that you would be better off dead or of hurting yourself in some way? ➢ Use of single items from large depressive symptom inventories has been validated for assessing SI.[27] ➢ SI was defined as a score of ≥1 and no SA in the last year.
SA in the last year	SA	➢ This was determined in follow-up interview with either the participant or proxy. The question specified SA in the previous year.

Abbreviations: AIS, Abbreviated Injury Scale; GCS, Glasgow Coma Scale; NTDB, National Trauma Data Bank; PHQ-8, Patient Health Questionnaire-8; PHQ-9, Patient Health Questionnaire-9.

Results

Of the 3575 participants in the final linked dataset, 293 (8.2%) endorsed suicidal thoughts in the 2 weeks prior to scheduled follow-up interviews but did not attempt suicide in the previous year, and 109 (3.0%) attempted suicide at some point during the year prior to the follow-up interview. Baseline and follow-up characteristics are shown in table 2. Those with SI only, and those

Table 2 Descriptive characteristics

Characteristic	No SI or SA (n=3173)	SI (No Attempt) (n=293)	P*	SA (n=109)	P†
Race (white)	68.1	67.2	.768	72.5	.332
Sex (male)	73.6	74.4	.754	66.1	.082
Age	24 (39–56)[‡]	24 (36–48)[‡]	.001[‡]	20 (38–44)[‡]	<.001[‡]
Education (≥12y)	67.7	64.9	.314	55.1[‡]	.006[‡]
GCS score[§]	8 (3–14)	7 (3–14)	.256	6 (3–14)	.060
Days to follow commands[‖]	2 (0–11)	3 (1–10)	.237	3 (0–12)	.386
Primary payer source (Medicare/Medicaid)	34.5	32.4	.472	43.1	.064
FIM cognitive D/C	24 (19–28)	25 (20–29)	.051	24 (19–28)	.904
FIM motor D/C	68 (56–79)[‡]	70 (60–81)[‡]	.015[‡]	66 (58–74)	.379
Blood alcohol level[¶]	0 (0–0.138)	0 (0–0.122)	.831	0 (0–0.129)	.936
Drug use at time of injury[#]	33.2	41.3[‡]	.012[‡]	46.2[‡]	.017[‡]
Infectious complications	36.6	41.6	.089	32.1	.336
Maximum AIS head score	4 (4–5)	4 (4–5)	.113	3 (3–5)	.909
Extracranial head injury	5 (0–13)[‡]	5 (0–15)[‡]	.030[‡]	5 (1–13)	.554
Assault	7.1	7.6	.615	9.2	.399
Self-inflicted injury	0.8	1.4	.335	2.8[‡]	.034[‡]
Problem substance use					
1y	12.5[‡]	20.8[‡]	<.001[‡]	31.2[‡]	<.001[‡]
2y	14.0[‡]	26.0[‡]	<.001[‡]	32.4[‡]	<.001[‡]
5y	15.8[‡]	22.7[‡]	.013[‡]	24.0[‡]	.059
PHQ-8 total					
1y	3 (0–6)[‡]	11 (6–15)[‡]	<.001[‡]	10 (6–17)[‡]	<.001[‡]
2y	3 (0–6)[‡]	10 (6–15)[‡]	<.001[‡]	11 (4–15)[‡]	<.001[‡]
5y	2 (0–6)[‡]	9 (5–16)[‡]	<.001[‡]	6 (1–10)[‡]	.003[‡]
FIM cognitive					
1y	32 (29–34)[‡]	31 (29–33)[‡]	<.001[‡]	30 (26–32)[‡]	<.001[‡]
2y	33 (30–34)[‡]	32 (29–33)[‡]	<.001[‡]	30 (26–33)[‡]	<.001[‡]
5y	33 (30–35)[‡]	32 (30–34)[‡]	<.001[‡]	31 (28–33)[‡]	<.001[‡]
FIM motor					
1y	89 (82–91)[‡]	88 (81–91)[‡]	.003[‡]	88 (80–91)[‡]	.025[‡]
2y	90 (84–91)	89 (84–91)	.066	86 (79–91)[‡]	.019[‡]
5y	91 (85–91)	90 (86–91)	.635	82 (89–91)[‡]	.033[‡]

NOTE. Values are percentage, median (interquartile range), or as otherwise indicated.
Abbreviations: AIS, Abbreviated Injury Scale; D/C, acute rehabilitation discharge; GCS, Glasgow Coma Scale; PHQ-8, Patient Health Questionnaire-8.
* The *P* value is for comparison of no suicidal endorsement or attempt with suicidal endorsement (no attempt).
† The *P* value is for comparison of no suicidal endorsement or attempt with SA.
‡ Statistical significance of $P<.05$.
§ Sample size differences for baseline characteristics: n=3036, n=284, and n=104, respectively.
‖ Sample size differences for baseline characteristics: n=3081, n=320, and n=108 respectively.
¶ Sample size differences for baseline characteristics: n=2301, n=266, and n=88, respectively.
\# Sample size differences for baseline characteristics: n=2517, n=248, and n=78, respectively.

who attempted suicide, were more likely to have been using drugs at injury (41.3% vs 33.2%, $P=.012$ and 46.2% vs 33.2%, $P=.117$, respectively) and were generally younger (mean, 37 vs 42y; $P<.001$ and mean, 33 vs 42y; $P<.001$, respectively) than those with no SI or SA. Additionally, a larger percentage of ideators and attempters reported problems with alcohol or drugs at follow-up (all years), and these participants also reported higher depressive symptoms at follow-up (all years) compared with nonideators and nonattempters. There were statistically significant differences in FIM of motor-based activities of daily living between those with and without SI and SA at time of discharge and follow-up and in FIM cognitive disability at follow-up. However, these differences did not represent a meaningful effect size (FIM motor differences <17 points and FIM cognitive differences <3 points[28]). Ideators and attempters differed significantly at 1 and 2 years postinjury (20.8% vs 31.2% and 26.0% vs 32.4%, respectively; $P<.001$) with regard to problem substance use.

SI models

The initial multivariate model for SI included demographics, clinical factors at time of injury, and clinical factors at time of follow-up known or suspected to contribute to SI after TBI (table 3). History of a preinjury SA was associated with an 11-fold increase in the probability of postinjury SI (odds ratio=10.93; 95% confidence interval, 2.14–55.78; $P=.004$). Depression severity, as measured by the Patient Health Questionnaire-8 (see table 1) at follow-up, also carried an increased odds of SI (odds ratio: 1.27; 95% confidence interval: 1.21–1.32; $P<.001$), indicating that for a single point increase in the Patient Health

Table 3 Factors associated with SI with no SA in the first 5 years after TBI

	Model 1				Model 2			
Associated Factor	OR	95% CI	*z*	*P*	OR	95% CI	*z*	*P*
Demographics								
Race (white)	1.11	0.70−1.75	0.44	.658	1.11	0.71−1.73	0.46	.646
Sex (male)	1.21	0.75−1.93	0.78	.433	1.16	0.73−1.84	0.64	.522
Age (y)	0.99	0.98−1.00	−1.23	.219	0.99	0.98−1.01	−1.00	.316
Education (≥12y)	1.51	0.96−2.40	1.77	.077	1.53	0.98−2.40	1.87	.061
Primary payer source (Medicare/Medicaid)	1.77	0.76−1.84	0.72	.473	1.19	0.78−1.84	0.79	.430
Clinical factors at time of injury								
Injury year	0.90	0.78−1.04	−1.44	.150	0.92	0.80−1.05	−1.24	.216
GCS score	1.00	0.96−1.05	0.22	.827	1.01	0.97−1.06	0.57	.568
Time to follow commands (d)	1.01	0.99−1.03	0.53	.599	1.01	0.99−1.13	0.68	.500
Infectious complications	0.97	0.62−1.52	−0.11	.909	0.89	0.58−1.34	−0.52	.600
Alcohol blood level	1.00	1.00−1.00	−0.26	.795	1.00	0.99−1.00	−0.28	.783
Drug use at time of injury	1.69*	1.11−2.86*	2.44*	.015*	1.86*	1.22−2.84*	2.89*	.004*
Intent†								
Self-inflicted	0.73	0.12−4.29	−0.35	.725	0.77	0.14−4.32	−0.30	.763
Assault	0.89	0.42−1.89	−0.30	.761	0.97	0.46−2.07	−0.08	.939
Clinical factors at time of follow-up								
Problem substance use (postinjury)‡	0.99	0.83−1.18	−0.10	.917	0.98	0.82−1.16	−0.28	.783
Preinjury SA	10.93*	2.14−55.78*	2.80*	.004*	11.07*	2.29−53.39*	2.99*	.003*
Depression (PHQ-8) (at follow-up)‡	1.27*	1.21−1.32*	11.29*	<.001*	1.27*	1.22−1.32*	11.56*	<.001*
FIM cognitive (at follow-up)‡	0.96	0.90−1.02	−1.36	.175	0.96	0.90−1.01	−1.51	.132
FIM motor (at follow-up)‡	1.00	0.97−1.03	0.06	.955	1.01	0.98−1.04	0.44	.658
Independent variables of interest: head and extracranial injury								
Head injury severity (maximum AIS head score; 1−6)					1.17	0.92−1.49	1.30	.193
Extracranial injury severity (ISS)§								
Mild (1−8)					1.32	0.75−2.29	0.96	.335
Moderate (9−15)					1.58	0.86−2.89	1.47	.142
Severe (>15)					2.73*	1.55−4.82*	3.46*	.001*

Abbreviations: AIS, Abbreviated Injury Scale; CI, confidence interval; OR, odds ratio; PHQ-8, Patient Health Questionnaire-8.
* Statistical significance of $P<.05$.
† Referent group is accident.
‡ Measured over time concurrently with outcome.
§ Referent group is 0 (no extracranial injury).

Questionnaire-8 score, there was a 1.27 times greater odds for SI, which translates to 3.3 times greater odds for SI with a 5-point increase in the Patient Health Questionnaire-8 score (1.27^5). Drug use at time of injury was associated with an increased odds of SI (odds ratio=1.69; 95% confidence interval, 1.11−2.86; $P=.015$). There were no other significant variables associated with SI, including FIM scores at follow-up.

The second model included head injury (maximum Abbreviated Injury Scale head score) and ECI (ISS categories) severity variables (see table 3). ECI, measured through ISSs, carried a higher likelihood of SI, but only when the ECI was severe (ISS>15). With a severe ECI, participants were nearly 3 times more likely to have SI (odds ratio=2.73; 95% confidence interval, 1.55−4.82; $P=.001$) compared with those with no ECI. The likelihood ratio test showed that head injury severity (maximum Abbreviated Injury Scale head score) and ECI (ISS) significantly contributed to the variance explained by the model ($\chi^2_4=13.74$, $P=.008$).

SA models

The only covariate that was significantly associated with SA in either the base or full model was Patient Health Questionnaire-8 (odds ratio=1.14; 95% confidence interval, 1.06−1.23; $P<.001$).

Exploratory analysis

We also explored whether ECI predicted depression, to assess whether the relation between ECI and SI might be explained (mediated) by depression. However, ECI was not significantly associated with depression severity and it did not interact with depression to predict SI (data not shown). Further, both ECI and depression severity remained significant in the SI model, indicating that depression severity does not explain the relation between ECI and SI. We also explored whether drug use at injury interacted with ECI to predict SI; this was not statistically significant.

Discussion

Recent work suggests that self-injury is the eighth leading cause of death in the United States,[29] and understanding the underlying reasons for suicide remains a significant public health problem. To our knowledge, this is the first study to link acute trauma factors to suicidality across the first 5 years post-TBI, and the analysis revealed a novel finding that those with severe ECI may be at the greatest risk for SI even many years after TBI. Although psychological factors (eg, higher prevalence of depression after TBI)[30] are strong contributors to the elevated risk of suicidality after TBI, there is also evidence that neurobiologic factors (eg, TBI-induced tissue loss, neuroinflammation) may contribute to suicidality after TBI.[31-33] Consistent with previous research,[13,34,35] we did not find significant associations between SI/SA and multiple indicators of TBI severity (maximum Abbreviated Injury Scale head score, Glasgow Coma Scale, and time to follow commands). However, we did find that drug use at the time of injury and substance abuse at follow-up, severe ECI, and depressive symptoms were associated with SI after TBI. It was surprising to discover that motor disability over time (FIM motor) was not meaningfully associated with SI, contrary to our hypothesis, suggesting that the relation between ECI and SI was not the result of greater motor disability or less functional independence with basic activities of daily living. Additionally, ECI was not predictive of depression, suggesting a more direct or alternatively mediated (eg, through other psychosocial factors over time) relation between ECI and SI. Perhaps individuals' overall adjustment to disability, a construct likely determined by a constellation of physical, psychological, and social factors, and not adequately captured by discrete measures of mood or basic functional ability, is a major determinant of postinjury suicidality. Future research is needed to better define and measure adjustment to disability after traumatic injury.

Within the general literature on suicidality, most research focuses on suicidal gestures or attempts, rather than SI in particular. Differences in the significant predictors and associated factors between SI and SA in this study suggest that SI and SA are different, although related, outcomes. Other latent factors not measured in this study may explain more of the variance in SA. A literature review of studies investigating SI after TBI found SI among those with a range of time postinjury was reported in 3% to 33% of individuals with TBI.[2] Those reporting SI have >5 times the risk of SA.[2] In our sample, participants with SI and participants with SA shared similar baseline and follow-up characteristics on all factors measured, with the one exception being that attempters were more likely to have substance abuse problems than ideators only. This finding is consistent with recent work suggesting that substance abuse problems contribute significantly to suicide rates nationally.[29,36]

Recent studies with non-TBI cohorts report that substance use disorders, second only to depression in some clinical samples,[37] are highly correlated with both SI and SA.[38-41] These data are also consistent with our findings because both substance use at injury and postinjury problematic substance use distinguish between those with and without SI in our sample. Interestingly, some studies examining the relation between substance use and SI or SA in the non-TBI literature suggest that these relations are bidirectional and that suicidality may either precede or follow the onset of substance use.[42,43] If drug use at injury in our sample is reflective of premorbid patterns of use, this behavior could be associated with underlying SI prior to the injury, which then persisted through the follow-up period. This idea is consistent with our finding that preinjury SAs are also associated with long-term SI. These findings reflect the fact that preinjury psychological and behavioral challenges, including mental health conditions and substance use disorders, can persist postinjury and often complicate recovery,[44-46] highlighting the importance of assessing and accounting for these factors in rehabilitation.

Although not often studied, full injury severity measured by ISSs has been informative for some TBI outcomes (eg, discharge disposition,[20] community integration[47]). Our study demonstrates a nearly 3-fold increased likelihood of SI among participants with severe ECI (ISS>15), representing major polytrauma, compared with those with isolated head injuries. However, the likelihood among those with mild to moderate ECI was not significantly higher. Although the association between TBI and suicidality has been well studied in both civilian and military populations,[1,48,49] there are few studies reporting on the association between ECI severity and SI. March[15] reported significantly higher odds of suicide completion or SA among those with severe traumatic injuries compared with participants without traumatic injuries. That study, however, did not separate cranial and ECI, examine mild to moderate traumatic injuries, or assess SI risk. Individuals sustaining polytraumatic injuries, including TBI, have poorer recovery over time than those with TBI alone or ECI alone.[50,51] Therefore, this is the first study specifically examining ECI severity after TBI as it relates to both SI and SA. Our results may have parallel implications for ECI in the military setting, a population at high risk for polytrauma.

Contrary to our hypothesis, we did not find motor disability to differ to a clinically meaningful degree (eg, ≥17 points) for those with SI or SA. Extrapolating from the SCI literature, SI/SA was most often associated with control of one's community activities, spiritual well-being, younger age at injury, and severity of injury.[52] This literature suggests that restrictions in community-based participation (rather than just basic activities of daily living, as measured with the FIM) or differences in age at injury may be factors explaining the association between severity of ECI and SI after TBI. However, these factors require further exploration.

Study limitations

This study is not without limitations. Large existing datasets, such as that used in this study, provide unique opportunities to study complex and clinically challenging populations, but they are also subject to the limitations and challenges associated with the use of large databases. The limitation of available data in this dataset regarding both pre- and postinjury psychosocial variables (eg, anxiety, cognitive functioning, social support, coping) reduces the opportunity to evaluate these factors in this study. For example, premorbid limitations in coping may influence both pre- and postinjury substance use and SI. Additionally, there are inconsistencies in the scales commonly used for assessing severity of initial head injury (eg, Glasgow Coma Scale score, maximum Abbreviated Injury Scale head score). For example, defining the time frame and severity of initial injury may vary between existing measures and determination and documentation of how medical interventions (eg, intubation, sedation) influence measurement. Further, the data have limited temporal resolution because they are collected at 1, 2, and 5 years after injury but with reference to the varying time frames (eg, since last assessment, in the last year, and in the last 2wk are all referent time frames of varying assessment tools). This limitation is perhaps most significant in our inability to assess temporal relations between SI and SA because SI is assessed with reference to the 2 weeks prior to the follow-up visit, whereas SA is assessed any time within the last year.

We also do not have complete data on completed suicides, and although extrapolations could be made with regard to the percentage of those with SI or SA who go on to complete suicide, there are also cases of completed suicide in the absence of either SI or SA. Despite a robust sample size overall for a TBI cohort, the incidence of SA was relatively low (approximately 3%) compared with other studies (1%−18%)[2] reporting postinjury SA. Therefore, this study may be underpowered to identify significant factors associated with SA that may have smaller effect sizes. This low incidence may be caused by a number of factors, including differences in study samples (evaluating a general TBI cohort vs a cohort specifically screened for depressive symptoms/SA), differences in how SA was measured (in the year prior to injury vs any postinjury SA), and differences in services received that may reduce post-TBI risk factors for SA (eg, all participants in the TBIMS National Database participated in inpatient rehabilitation). Although a strength of this study is its robust longitudinal modeling, this research design does not allow for differentiation of temporal relations between associated factors and SI or SA.

Conclusions

Suicidality may develop through multiple pathways, whether related to the injury itself, to individual differences in adjustment to disability and in coping, or to other personal, biologic, and environmental factors unaccounted for or obscured when data are aggregated. Developing proactive strategies to monitor at-risk individuals and provide early intervention for SI may have a positive public health effect by decreasing the incidence of suicide after TBI.

Supplier

a. Stata version 13; StataCorp.

Keywords

Brain injuries; Injury Severity Score; Multiple trauma; Rehabilitation; Suicidal ideation; Suicide, attempted

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