ORIGINAL ARTICLE

Development of Posttraumatic Stress Disorder After Mild Traumatic Brain Injury

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ABSTRACT. Hoffman JM, Dikmen S, Temkin N, Bell KR. Development of posttraumatic stress disorder after mild traumatic brain injury. Arch Phys Med Rehabil 2012;93:287-92.

Objective: To examine the incidence of and factors associated with the development of posttraumatic stress disorder (PTSD) 6 months after civilian mild traumatic brain injury (MTBI).

Design: Secondary analysis of a randomized controlled trial of telephone follow-up versus usual care to reduce MTBI symptoms and improve function.

Setting: In-person and telephone interviews.

Participants: Prospectively studied participants (N=239) with MTBI enrolled in the emergency department.

Interventions: Not applicable.

Main Outcome Measures: Secondary analysis with main outcome measure of Posttraumatic Stress Disorder Checklist–Civilian Version.

Results: At 6 months after MTBI, 17% of participants met criteria for diagnosis of PTSD. Logistic regression predicting PTSD from baseline characteristics showed that participants who were Hispanic versus white, non-Hispanic and who, at the time of injury, described themselves as less happy and believed they would be more affected by their injury were significantly more likely to have PTSD.

Conclusions: Rates of PTSD in civilian MTBI in this study are consistent with prior research. Results suggest that personality characteristics and attribution regarding the injury may impact the development of PTSD. Early interventions addressing risk factors may prevent or reduce the likelihood of developing PTSD.

Key Words: Traumatic brain injury; Posttraumatic stress disorders; Rehabilitation.

THE IMPORTANCE OF examining posttraumatic stress disorder (PTSD) in persons with mild traumatic brain injury (MTBI) has drawn considerable attention because of the frequent co-occurrence of the 2 in the current Operation Iraqi Freedom/Operation Enduring Freedom conflicts.[1] Those with both conditions appear to be at greater risk for postconcussion symptoms than either one alone. Vanderploeg et al[2] also found that MTBI prolongs recovery from PTSD. Such findings underscore the importance of monitoring the occurrence of both MTBI and PTSD, and investigating the implications for prevention and intervention efforts.[3] The co-occurrence of PTSD and MTBI clearly complicates assessment and treatment of these disorders.

There has been controversy about whether traumatic brain injury (TBI) and PTSD can actually co-occur, given the belief that to develop PTSD, one must remember the event, thereby "protecting" individuals with TBI who have posttraumatic amnesia and impaired recollection of events surrounding their injuries.[4] However, more recent research suggests that TBI and PTSD can and do co-occur at all severity levels of TBI. Disturbing recollections of the stressful event can derive from actual memory, "islands" of memory that could occur throughout the period of posttraumatic amnesia, reexperience of events through a fear-conditioned response, and memory that is reconstructed through imagined recall or by report of others.[4]

Bryant et al[5-9] have conducted several studies on PTSD after general trauma and specifically after TBI. Their results have contributed to estimates of incidence and prevalence of PTSD and have highlighted the importance of using standard assessments for TBI and PTSD. Overall, incidence rates have been found to differ by severity of TBI. In those with MTBI, PTSD rates have been found to range from 11% to 24%, while in those with more severe TBI, rates range from 0% to 27%. The large variation in rates found in those with severe TBI may be due to a variety of factors including methods to assess PTSD, the sample studied, and the impact of brain injury on the ability to respond accurately to the assessment.[4,10,11] While significant impairments in cognition are not expected months after MTBI,[12,13] PTSD has been found to occur more frequently in those with MTBI who report loss of consciousness (LOC), reflecting relatively more severe injury.[14] This is also consistent with reports from the military. For example, Hoge et al[1] reported that, of those individuals who reported concussion with LOC, PTSD has been diagnosed in 43.9%, compared with 27.3% with concussion with alteration of consciousness, and in only 9.1% of returning warriors with no TBI. However, rates of PTSD may start to decrease when LOC after TBI is prolonged (ie, ≥12h).[15] While previous studies have examined the co-occurrence of TBI and PTSD, there is little information about potential risk factors for developing PTSD in those with TBI.

List of Abbreviations

ED	emergency department
GOAT	Galveston Orientation and Amnesia Test
LOC	loss of consciousness
MTBI	mild traumatic brain injury
PCL-C	PTSD Checklist–Civilian Version
PTSD	posttraumatic stress disorder
TBI	traumatic brain injury

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In this study, we sought to examine (1) the rates of PTSD in a representative group of people diagnosed with MTBI in the emergency department (ED), which has not previously been reported; and (2) potential risk factors including demographics, preinjury depression, anxiety, and personality factors (information collected within hours of injury) for the development of PTSD at 6 months after injury.

METHODS

The current study is a post hoc analysis that used a subsample of 239 participants who completed the PTSD Checklist–Civilian Version (PCL-C) at 6 months postinjury. These participants were originally enrolled in a clinical trial aimed at improving postconcussion symptoms after MTBI, with the intervention delivered by telephone.[16] The larger study enrolled 366 participants with diagnosed MTBI who completed an initial baseline evaluation less than 48 hours after injury, with 4.4 hours being the median time between injury and initial baseline evaluation. Participants were randomly assigned to either telephone follow-up or usual care and a total of 312 completed 6-month assessments. The PCL-C was added to the outcome assessment battery after the study began, and therefore only the 239 consecutive participants in the current study were given and completed this measure. Participants were recruited through the EDs of 2 university-based hospitals, one of which is a level I trauma center. Enrollment criteria were (1) admission to the ED within 48 hours of injury; (2) likely circumstances for MTBI (eg, motor vehicle collision or fall); (3) ED Glasgow Coma Scale score of 13 to 15[17]; (4) documented, self-reported, or witnessed LOC for 30 minutes or less, a period of impaired consciousness (eg, confusion) for 24 hours or less, or posttraumatic amnesia for 24 hours or less (consistent with the operational definition of MTBI recommended by the Centers for Disease Control and Prevention[18]); (5) sufficient knowledge of the English language to participate in the intervention calls; and (6) permanent home address and phone number.

Exclusion criteria were (1) age less than 16 years; (2) abnormal findings on intracranial computed tomography; (3) admission to the intensive care unit; (4) serious nonextremity injuries (eg, organ lacerations, pelvic injuries); (5) extremity injury(ies) more severe than a single-limb closed fracture; (6) current or progressive neurologic disease (eg, multiple sclerosis, stroke, brain tumor) or end-stage terminal disease; (7) self-report or medical record evidence of recent or current major psychiatric illness such as schizophrenia or bipolar disorder; (8) injury received during sexual assault; and (9) hospitalization of more than 2 nights for head injury in the past year. Persons with evidence of serious or long-standing alcohol abuse (ie, ED blood alcohol level $\geq$250mg/dL or self-report of alcohol consumption on waking up in the morning) and/or current dependence on illegal or prescription drugs were excluded, as were prisoners or those in custody.

The initial clinical trial was approved by the Institutional Review Board of the University of Washington, and details on enrollment and study design have been previously published.[16] Briefly, the study was a randomized 2-group design with outcome assessment at 6 months after injury. The control group received standard care for MTBI in the ED, and the treatment group received standard care plus scheduled telephone contacts during the first 3 months after injury. We found that those participants who received telephone follow-up aimed at symptom self-management and activation had fewer and less severe postconcussion symptoms and were less negatively impacted by persisting symptoms. However, analyses indicated that there was no difference between the 2 groups on the PCL-C, and thus the data were pooled for the current analyses.

Measures

Baseline assessment. The baseline assessment at the time of enrollment consisted of demographic questions on age, sex, income, education, and living situation (living alone vs with others); a history of physical and mental health problems including a past diagnosis of PTSD; and questions related to the injury including the Galveston Orientation and Amnesia Test (GOAT) score[19] and the cause of injury. The cause of injury was divided into 5 categories: (1) moving vehicle related (motor vehicle, motorcycle, all-terrain vehicle, bicycle, or pedestrian crash); (2) assault (by use of blunt force); (3) fall; (4) sports related; and (5) other (eg, unintentionally struck by object). Race and ethnicity were coded into 3 distinct groups including those who were of (1) Hispanic descent (both white and nonwhite); (2) white, non-Hispanic; and (3) nonwhite, non-Hispanic.

Subjects were given the GOAT[19] in the ED as part of their eligibility determination, and to determine their ability to give informed consent. Baseline testing in most cases took place within 24 hours of injury. As part of the GOAT, subjects' recollection of events (rather than what they were told happened) and their timing after the injury was determined by careful, nonleading questioning. The duration of posttraumatic amnesia was determined to be the period from injury to when continuous memory returned.

In addition, we developed Likert scales (0–10) to assess each individual's rating of worry ("I never worry" to "I worry all of the time"), happiness ("very unhappy" to "very happy"), sense of control ("I have no control" to "I am fully in control"), and energy level ("completely worn out" to "I have good energy") in the 2 weeks before injury. Also, we asked individuals to rate on a 0 to 10 Likert scale their perception of injury severity ("minor" to "very serious") and how much they expected the injury to affect their life ("not at all" to "large impact"). These items were formulated to examine possible individual personality factors that might influence outcomes after MTBI. Finally, we asked 4 questions, rated on a scale from 0 to 100, to assess attribution of blame for their injury to (1) self, (2) others, (3) circumstances, or (4) chance.

Outcome assessment. All participants were assessed by telephone at 6 months postinjury by an examiner blinded to their treatment assignment. Included in the assessment was the PCL-C.[20] The PCL-C is a reliable and valid measure of PTSD with an estimated 10th grade reading and comprehension level requirement.[21,22] Subjects were also administered the same Likert scales originally given at baseline, but asked about their current level of worry, happiness, control, energy level, perceived injury severity, and impact of injury.

Statistical Analyses

A diagnosis of PTSD was assigned when a participant scored in the moderate range or above (3–5) on each of the required symptom clusters in the PCL-C to meet the criteria delineated in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*.[23] Univariate comparisons were conducted with *t* tests or Mann-Whitney tests for continuous measures, and chi-square tests or Fisher exact tests for categorical data. Regression (when the variable was normally distributed) and rank regression (when normal distribution was not found) were used to relate the Likert items at 6-month outcome to PTSD while controlling for baseline assessment. Finally, we examined the strength of the relationship between individual baseline char-

Table 1: Univariate Comparison of Baseline and Outcome by PTSD

Baseline Variables	PTSD No (n=198)	PTSD Yes (n=41)	*P*
Age (y)	32.4±13.5	31.9±12.5	.842
GOAT	95.0±5.2	94.7±5.4	.736
Sex (male)	132 (67)	23 (56)	.212
Income (monthly)			.696*
- None	38 (19)	10 (24)	
- ≤$2000	69 (35)	16 (39)	
- >$2000	81 (41)	15 (37)	
- Unknown	10 (5)	0 (0)	
Education			.012*
- Less than high school	7 (4)	6 (15)	
- High school or above	190 (96)	35 (85)	
Cause of TBI			.348*
- Moving vehicle related	116 (59)	26 (63)	
- Assault	24 (12)	8 (20)	
- Fall	13 (7)	0 (0)	
- Sports	31 (16)	5 (12)	
- Other	14 (7)	2 (5)	
Living alone and unmarried	126 (64)	27 (66)	.468
Race/ethnicity			.001*
- Hispanic	10 (5)	9 (22)	
- White, non-Hispanic	142 (72)	20 (49)	
- Nonwhite, non-Hispanic	46 (23)	12 (29)	
Diagnosis of PTSD before TBI	3 (2)	0 (0)	1.000*
Treatment group	103 (52)	20 (49)	.734

NOTE. Values are mean ± SD, n (%), or as otherwise indicated.
*By Fisher's exact test.

acteristics, including demographics, history of PTSD, and the Likert items examining personality factors, and the development of PTSD at 6 months by using exact logistic regression. To evaluate independent contribution, we selected predictors with the use of asymptotic logistic regression with forward stepwise selection of variables, using $P<.05$ for both inclusion and removal of variables, and refined the resulting model by getting exact estimates and confidence intervals. Statistical significance was set at $P\leq.05$. No adjustment was made for multiple comparisons. *P* values are provided in tables 1 and 2, and confidence intervals are provided in table 3 for interpretation.

RESULTS

A total of 41 participants (17%) were found to meet criteria for PTSD using the PCL-C. These participants differed from those without PTSD on some demographic and injury characteristics (see table 1). Those with PTSD were more likely to be nonwhite, Hispanic and to have not completed high school.

Likert Item Comparison

Table 2 compares those with and without PTSD at 6 months after injury on each Likert item at baseline and 6 months postinjury. Results suggest that participants with PTSD at 6 months reported more worry and less happiness before their injury on baseline measures compared with those without PTSD. In addition, those with PTSD at 6 months also reported stronger beliefs at baseline that the injury would affect their lives, and they were less likely to blame themselves but significantly more likely to blame others for their injury. At the 6-month postinjury assessment, both with and without adjusting for baseline values, those with PTSD described themselves as significantly more worried and less happy than those without PTSD. The difference also increased from the baseline assessment. They additionally reported having less control over their lives and lower energy than those without PTSD. They continued to describe themselves as being more affected by their injury, even after adjusting for the baseline difference. At 6 months, those with PTSD assessed their injury as more severe than those without PTSD. There was no difference in reports of the role of circumstances or chance impacting injury between the 2 groups at baseline.

Logistic Regression

In the stepwise forward logistic model examining baseline factors independently related to the development of PTSD, results suggest that those participants who were Hispanic versus those who were white, non-Hispanic; those who rated themselves as less happy at baseline; and those who reported that they would be more affected by injury at baseline were significantly more likely to develop PTSD at 6 months postinjury (see table 3).

DISCUSSION

Our study is the first to identify potential preinjury personality factors as increasing the risk for the development of PTSD after MTBI. These data contribute to the existing literature on the contribution of personality characteristics[24-26] and the role of individual expectations of recovery after injury to the development of PTSD in persons without TBI.[27,28] While we did not perform extensive personality assessments in this study, significant differences found at baseline in self-reported levels of worry and happiness suggest personality differences between those who later developed PTSD and those who did not. The strengths of our study include (1) the relatively large, nonselect sample that was prospectively studied; and (2) assessment of preinjury personality characteristics, blame, and expectations soon after injury as predictors of 6-month outcome rather than concurrently at 6 months when PTSD had already developed.

The role of personality characteristics in the development of PTSD has been examined for individuals exposed to trauma, but not in those who have TBI.[24] Studies have focused on neuroticism as being the trait behind the development of PTSD, with neuroticism describing the tendency to react negatively and strongly to adverse events.[24,25] Although personality traits have been examined in individuals with TBI, the focus has been on whether any personality change occurs after injury rather than how preinjury personality traits may predispose to the development of PTSD. Rush et al[29] found no evidence of personality change after TBI, but did not examine whether personality traits had any impact on outcome. In addition, much of the research that has examined PTSD after traumatic injury, such as in spinal cord injury, has typically been cross-sectional and without assessment near the time of injury.[30]

Our findings that those participants who early after injury expected that they would be significantly impacted by injury had a higher likelihood of developing PTSD are similar to the findings of studies that have examined the persistence of postconcussional symptoms. Whittaker et al[31] noted that patients who believe that their MTBI will have serious consequences to their life after injury tend to have more persistent symptoms at 3 months postinjury. A recent study[32] examined the impact of preinjury stressful events and found that those with new MTBI who had more stressors tended to have poorer outcomes at 3 months postinjury. These findings together support the need for additional research to examine person factors, including per-

Table 2: Comparison of Likert Items From Baseline to 6 Months Postinjury

Measure	PTSD No		PTSD Yes		Assessment of Difference at Baseline	Assessment of Difference at 6mo	Regression* (Group Effect on 6-mo Value, Adjusting for Baseline)
	Baseline	6mo	Baseline	6mo			
Worry	4.02±2.59	4.50±2.55	5.44±2.83	6.90±2.23	1.42†	2.40†	1.74
					P=.002	*P*<.000	*P*<.001
Happiness	7.97±1.85	7.72±1.99	6.73±2.76	5.80±2.99	−1‡	−2‡	*P*=.002
					P=.014	*P*<.001	
Control	7.76±2.18	7.56±2.09	7.73±1.73	6.52±2.44	0‡	−1‡	*P*=.009
					P=.467	*P*=.007	
Energy	5.16±2.55	5.38±2.64	5.20±2.67	3.76±3.15	0.04†	−1.63†	−1.64
					P=.930	*P*=.001	*P*<.001
Injury severity	4.93±2.32	5.80±2.38	5.53±2.59	7.22±1.99	0.59†	1.42†	1.12
					P=.152	*P*<.001	*P*=.003
Affected by injury	3.59±3.17	4.79±3.10	5.38±3.27	7.88±2.40	1.79†	3.09†	2.53
					P=.001	*P*<.001	*P*<.001
Blame self§	43.0±41.0		23.7±34.6		−10‡		
					P=.002		
Blame others§	38.6±42.8		61.1±43.0		20‡		
					P=.001		
Blame circumstances§	36.2±38.2		33.3±39.5		0‡		
					P=.506		
Blame chance§	41.0±37.5		49.1±41.2		0‡		
					P=.244		

NOTE. Values are mean ± SD or as otherwise indicated.
*Linear regression was used for normally distributed variables and rank regression for nonnormally distributed variables. Regression coefficients are given for the normally distributed variables.
†*t* test used for normally distributed variables. Mean difference and level of significance are given.
‡Mann-Whitney *U* test used for nonnormally distributed variables. Median difference and level of significance are given.
§Blame questions were only included at baseline for the entire sample.

sonality and attribution of blame, in PTSD and other outcomes after TBI.

The results from the current study are consistent with prior research on incidence of PTSD and association with ethnicity. The incidence of PTSD for individuals 6 months after MTBI was found to be 17%, which is consistent with ranges from other civilian and military studies.[33,34] In addition, we found that Hispanic individuals with MTBI had a higher likelihood of developing PTSD, extending the findings of previous studies.[35,36] We did not collect specific measures of the broader category of social disadvantage, which may be the contributing factor for these individuals developing PTSD[37] and other mental health conditions.[38]

Study Limitations

A number of limitations of this study may impact findings. Assessment of PTSD was done through self-report on the PCL-C and was not confirmed with a structured clinical interview. In addition, we had a relatively small population of ethnic/racial minority subjects and cannot comment fully on other potential ethnic differences in stress reactions. In addition, although there was a significant relationship between the perception of blame and later development of PTSD, it is possible that specific differences in the circumstances of the injuries influenced the perception of blame but were not captured by our study. Future research on blame attribution should include a detailed analysis of injury circumstances. Finally, while we asked participants to rate their level of worry, happiness, control, and energy before their injury, this was done retrospectively and may have been biased by the experience of their injury and the ED.

Future Directions for Interventions

No difference was found between the treatment and control group in the primary treatment study for the development of PTSD, which suggests that telephone follow-up focused on minimizing postconcussion symptoms did not address factors related to the development of PTSD. Results from the current study suggest that future interventions might benefit from targeting individuals with MTBI who are at risk for developing PTSD, and addressing comorbid mental health difficulties as well as individual expectations.

Identifying personality characteristics and factors related to triggers of PTSD may be an important next step for understanding and potentially identifying individuals who are likely to develop PTSD and who may benefit from early intervention. Given that a recent Cochrane review[39] suggests that treatment for PTSD for those exposed to stressful situations, but not reporting symptoms, may actually lead to an increase in report of symptoms, such identification would better target treatment for only those who need it. Reviews of current preventive interventions have not provided strong recommendations for any specific interventions.[40,41] The Institute of Medicine report on treatment of PTSD suggested that future research is needed to reach the level of certainty that current interventions are effective. The Institute of Medicine is also continuing to study ongoing efforts in the treatment of PTSD, which may produce evidence for specific recommendations.[42]

CONCLUSIONS

The results of the current study suggest a role for personality characteristics in the development of PTSD after MTBI. Knowledge about such risk factors in the development of PTSD provides an opportunity to develop tailored interventions

Table 3: Logistic Regression Examining the Association of PTSD With Baseline Factors

Variable	Descriptive	Univariate (Exact Regression) OR	Univariate (Exact Regression) 95% CI	Multivariate* (Exact Regression) OR	Multivariate* (Exact Regression) 95% CI
PTSD (outcome)	41/239 (17)				
Age (per 10y)	32.3±13.3	0.97	0.75–1.25		
GOAT (per 10 points)	95.0±5.2	0.90	0.48–1.73		
Sex (male)	155 (65)	1.57	0.31–1.35		
Income (none)	48 (20)				
- ≤$2000 vs none	85 (36)	0.88	0.34–2.40		
- >$2000 vs none	96 (40)	0.71	0.27–1.93		
- Unknown vs none	10 (4)	0.29	0.00–2.08		
High school or above (vs less than high school)	225 (95)	0.22	0.06–0.83		
Cause (MVR)*	142 (59)				
- Assault vs MVR	32 (13)	1.48	0.52–3.92		
- Fall vs MVR	13 (5)	0.25	0.00–1.58		
- Sports vs MVR	36 (15)	0.72	0.20–2.13		
- Other vs MVR	16 (7)	0.64	0.07–3.06		
Living single	153 (64)	0.91	0.41–1.93		
Race/ethnicity (Hispanic)	19 (8)				
- White, non-Hispanic vs Hispanic	162 (68)	0.16	0.05–0.50	0.21	0.06–0.74
- nonwhite, non-Hispanic vs Hispanic	58 (24)	0.30	0.08–1.02	0.32	0.08–1.25
Diagnosis of PTSD before injury	3 (1)	1.25	0.00–11.81		
Telephone group	123 (51)	0.88	0.42–1.82		
Stress (0–10)	4.7±2.9	1.13	1.00–1.28		
Worry (0–10)	4.3±2.7	1.21	1.07–1.38		
Happiness (0–10)	7.8±2.1	0.78	0.67–0.91	0.80	0.68–0.94
Control (0–10)	7.8±2.1	0.99	0.85–1.18		
Energy (0–10)	5.2±2.6	1.01	0.88 –1.15		
Injury severity (0–10)	5.0±2.4	1.11	0.96–1.28		
Affected by injury (0–10)	3.9±3.2	1.18	1.06–1.31	1.14	1.01–1.28
Blame self (0–10)	4.0±4.1	0.88	0.79–0.96		
Blame others (0–10)	4.2±4.4	1.13	1.04–1.22		
Blame circumstances (0–10)	3.6±3.8	0.98	0.89–1.07		
Blame chance (0–10)	4.2±3.8	1.06	0.97–1.16		

NOTE. Values are n (%), mean ± SD, or as otherwise indicated.
Abbreviations: CI, confidence interval; MVR, motor vehicle related; OR, odds ratio.
*Forward stepwise ($P \leq .05$ to enter, $P > .05$ to exit).

that may prevent or reduce the likelihood of development of PTSD. Because baseline perceptions appear related to outcome, interventions immediately after injury should be studied. Further research should develop better characterization of patient personality profiles, in addition to severity of injury and circumstances of incidents leading to the development of PTSD, to allow for targeted selection of patients for early intervention to prevent PTSD and other complicated outcomes.

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