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Research Article

The Role of Blame Attribution in Severity of Prolonged Concussion Recovery

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Abstract

Psychological risk factors are associated with prolonged recovery from Concussion. We investigated whether blame attribution is another risk factors for prolonged recovery. 91 new patients presenting with prolonged recovery from Concussion at a Canadian subspecialty concussion clinic were included in the study. Participants were separated into three groups: those attributing external blame for their concussion (n=70), those describing the incident as accidental (n=20), and those attributing internal blame (n=1). Observations included: population variables, subjective percent recovered (SPR), duration of symptoms thus far, and presence of symptoms. Participants were more likely to belong to the external blame group versus the accidental or internal blame groups ($p<0.0001$). Mean SPR in accidental was 73%, compared to 44% in external ($p<0.0001$). The following occurred more in external versus accidental (relative risk[95% confidence interval]): PTSD (32.24[2.08-500.12]), depression (3.78[1.56-9.19]), anxiety (3[1.53-5.89]), headache (1.21 [0.97-1.51]), irritability (2.76[1.40-5.44]), and cognitive symptoms (1.77[1.13-2.77]). There were no differences in the distribution of sleep disorders and vestibular symptoms. Mean symptom duration thus far was 10.2 months in accidental and 22.5 months in external ($p=0.001$). External and accidental groups did not differ in age ($p=0.938$), number of concussions ($p=0.72$), sex ($p=0.908$), or pre-existing mental illness ($p=0.735$). Patients with prolonged recovery are more likely to externally attribute blame for their initiating concussion, which is associated with a longer, more severe course. This study suggests blame attribution could be used to determine patients at risk of prolonged recovery from concussion.

Keywords: *Post-concussion syndrome, PCS, Blame, Concussion, Recovery*

Introduction

Prolonged recovery after concussion consists of a range of symptoms, previously recognized as Post-Concussion Syndrome (PCS). Specifically: an mTBI is defined as a Glasgow Coma Scale (GCS) score of 13-15, 30 minutes after a head injury.[1] Common symptoms of prolonged recovery after concussion include: headaches, irritability, dizziness, cognitive deficits, fatigue, visual disturbances, anxiety and depression.[2]

Historically, records show approximately 110 per 100,000 Canadians suffered a concussion from 1996-1997.[3] More recent studies from Ontario show that the average annual incidence of concussion between 2008 and 2016 was 1153 per 100,000.[4] These records still likely underestimate the actual number of concussions in the population, as concussions are not always recognized by a patient and/or diagnosed by a clinician. Estimations of PCS prevalence vary greatly depending on the precise classification method being used, with PCS being diagnosed in 5-43% in subjects after an mTBI.[5]

Considering the prevalence of prolonged recovery and the debilitating nature of its symptoms, a deeper understanding of the syndrome is required. The precise etiology of the syndrome is not yet known. Some researchers point to neurobiological changes, such as regional volume loss in patients with an mTBI.[6] Other studies have noted the organic brain pathology after an mTBI and commented on its similarity to the neurobiological patterns observed in depression,[7] and anxiety.[8]

In contrast to the above neurobiological explanations are several convincing critiques that oppose the existence of PCS as a standalone "syndrome". Some argue that the vague PCS symptoms exist at a high rate amongst a healthy population and are highly correlated with depressive symptoms, thus implying PCS is an example of misattribution.[9] In a study on young patients, pre-injury somatization, wherein patients with mental illness demonstrate somatic symptoms unrelated to physical disease,[10] was positively correlated with symptoms of PCS after an acute concussion.[11]

The uncertainties surrounding the etiology of prolonged recovery from concussion present a serious conundrum for researchers. Regardless, the symptoms that accompany the syndrome have a profound effect on the daily functioning of patients. Therefore, while the existential debate rages on, we must examine the collection of factors influencing severity, in the hopes of identifying targets for clinician intervention. For those concerned about the existential debate, insight gained from the exploration of risk factors may also contribute to further understanding of the syndrome.

Several psychological risk factors, such as premorbid anxiety or depression disorders, acute post-traumatic stress and post-traumatic stress disorder (PTSD) severity have been correlated with the development of prolonged symptoms.[12] Even a patient's sex has been argued to be a risk factor, however, studies report contrasting results regarding which sex is at a greater risk of PCS.[12,13] A meta-analysis has shown that ongoing litigation, financial compensation, and compensation-seeking behaviour are positively correlated with PCS duration and/or severity.[14] Furthermore, there may be ingrained societal ideals that put individuals at risk for prolonged recovery. Compared to Canada, where expectations of long-lasting symptoms and litigation after a motor vehicle collision are more common, subjects in Lithuania, where litigation is less common, expected less chronic symptoms after a motor vehicle collision.[15]

Blame attribution is a psychological risk factor that has been correlated to the prognosis of patients in other studies. After suffering facial trauma, patients who attributed external blame for the traumatic incident had greater rates of anxiety and depression compared to those that attributed internal blame.[16] A similar correlation was found between external blame attribution for a TBI and subsequent depression.[17] With regard to mTBI's specifically, external blame attribution for the incident is associated with a worsened prognosis in the form of increased PTSD, anxiety, depression and post-concussive symptoms.[18]

We hypothesize that patients who externally attribute the blame for their causative concussion are demonstrating an additional psychological risk for prolonged recovery. This hypothesis is based on the positive correlations between other psychological risk factors and severity of reported symptoms.[12,14] In this study, we expect that patients who attribute external blame will go on to experience a lengthier and/or more symptomatic course of the syndrome.

Methods

Study design and participants

PCS was defined by DSM-V criteria as the persistence of neurocognitive changes including the presence of headache, memory changes, concentration difficulty, vestibular symptoms, irritability, sleep disorder, anxiety, and depression, in a patient diagnosed with an mTBI $\geq$3 months ago.

111 new patients presenting at a Canadian subspecialty concussion clinic with a diagnosis of concussion with prolonged recovery from 2017-2019 were questioned about whom they blamed for their initiating concussion. Inclusion criteria were that the patient must remember the causative incident, and the patient must not have suffered organic brain damage during the incident. These inclusion criteria were met by 91 patients (56 females, 35 males). 20 patients were excluded from the study due to one of the following: the inability to define the specific causative event of their concussion (n=13), the presence of an underlying structural lesion (n=1), or evidence of an intracranial bleed (n=6). These inclusion and exclusion criteria allowed us to clearly separate participants according to their pattern of blame attribution, as well as remove the confounding effect of organic brain pathology.

Patients were then assigned to either an external blame attribution (n=70), an accidental (n=20) or an internal blame attribution group (n=1). Those in the external blame attribution group claimed an external party was to blame for their concussion. Those in the internal blame attribution group claimed they were responsible for their concussion. Those in the accidental group described the event leading to their concussion as "accidental" and did not attribute any blame for the event. Patients were followed over an approximately 2-year period at the clinic, where the presence, severity and duration of symptoms were recorded.

Clinical variables

The patients' electronic medical records (EMR) were examined for diagnoses of mental illnesses occurring before the incident concussion. The mental illnesses seen throughout our record review were anxiety, depression, and bipolar disorder. The total number of previous concussions were collected from the EMR, as well as verbal confirmation with the patients. Subjective percent recovered (SPR) was recorded by asking the patients to rate their perceived recovery after their mTBI, ranging from 0% to 100%, at their most recent visit to the clinic. Patients were asked to report whether they were experiencing headaches, irritability, cognitive symptoms (impaired memory and/or concentration), sleep disorders (difficulty falling asleep, staying asleep, hypersomnolence, sleep apnea), and vestibular symptoms (tinnitus, imbalance, vertigo) during their time at the clinic. Post-mTBI anxiety, depression and/or post-traumatic stress disorder (PTSD) were noted as present if the patient manifested symptoms and signs that reached a diagnostic threshold for the mental illness during their time at the clinic.

Statistical analyses

Statistical analyses were performed with the XLSTAT program. Continuous data were analyzed with a t-test, preceded by a Shapiro-Wilk test to ensure normal distribution of data, along with Fisher's F-test to confirm homoscedasticity. If continuous data failed to pass both the above tests, they were instead analyzed with a Mann-Whitney U test.

Categorical data meant to be analyzed with a chi-squared test were first examined to ensure there were >5 samples in each category of the contingency table. Failure to satisfy this requirement resulted in analysis using Fisher's exact test. The α-value for all statistical tests was set at 0.05.

The internal blame group was excluded from all statistical analyses except for the distribution of patients between blame groups, because of its extremely small sample size.

The age of the external blame group was compared to the accidental group using a t-test. A Mann-Whitney U test was used to compare total number of concussions before the incident concussion in the external blame group to the accidental group. The distributions of patients by sex and by presence of previous mental illnesses were examined with chi-squared tests. The distribution of patients between blame groups was compared with Fisher's exact test.

Rates of post-concussion irritability, cognitive symptoms, anxiety, sleep disorders, and vestibular symptoms were compared between the external blame group and the accidental group using a chi-squared test. Fisher's exact test was used to compare rates of PTSD, depression, and headaches between the external fault group and the accidental group.

Symptoms found to have a statistically significant difference in distribution between blame groups via either chi-squared test or Fisher's exact test had their relative risks and 95% confidence intervals calculated.

The duration of concussion symptoms thus far, and the subjective percent recovered in the external blame group versus the accidental group were compared using Mann-Whitney U tests.

Results

Blame group distribution

Patients presenting to the clinic with prolonged recovery from concussion were asymmetrically distributed between the external blame group (n=70), the accidental blame group (n=20), and the internal blame group (n=1) ($p<0.0001$).

Blame group populations

Our population had more females than males and the majority of the patients had no history of mental illness across groups. Average age was 46 years in the external blame group and 46.3 years in the accidental group. Mean number of concussions occurring prior to the incident event were 1 ($\pm$0.28) for the external blame group and 0.7 ($\pm$0.23) for the accidental group. There was no statistical difference between the groups for any of these variables (Table 1).

Table 1. Results of chi-squared tests, *t-test and **Mann-Whitney U test on patient populations.

Population variable	External blame group	Accidental group	p-value
Sex			
Male (%)	27 (39%)	8 (40%)	
Female (%)	43 (61%)	12 (60%)	0.908
Past medical history of mental illness			
Yes (%)	15 (21%)	5 (25%)	
No (%)	55 (79%)	15 (75%)	0.735
Mean age (years)* ± SE	46 ± 2	46.3 ± 3.58	0.94
Mean number of previous concussions ± SE**	1 ± 0.28	0.7 ± 0.23	0.72

Data on sex and past medical history of mental illness presented as numbers of patients per group with percentages in parentheses, and p-value derived from chi-squared tests. Data on mean age presented with ± standard error (SE), and p-value derived from t-test. Data on mean number of previous concussions presented with ± SE, and p-value derived from Mann-Whitney U test.

Symptom occurrence among blame groups

Rates of PTSD, depression, anxiety, irritability, cognitive features, and headache were asymmetrically distributed between the external blame and accidental groups (Table 2). Patients belonging to the external blame group had a higher relative risk of experiencing these symptoms compared to those in the accidental group (Table 3). In contrast, rates of sleep disorders and vestibular symptoms appear to be randomly distributed between the groups.

Table 2. Results of chi-squared tests and *Fisher's exact tests on presence of symptoms in blame groups.

Symptom	External blame group	Accidental group	p-value
PTSD*			
Yes (%)	54 (77%)	0 (0%)	
No (%)	16 (23%)	20 (100%)	**<0.0001**
Depression*			
Yes (%)	53 (76%)	4 (20%)	
No (%)	17 (24%)	16 (80%)	**<0.0001**
Anxiety			
Yes (%)	63 (90%)	6 (30%)	
No (%)	7 (10%)	14 (70%)	**<0.0001**
Irritability			
Yes (%)	58 (83%)	6 (30%)	
No (%)	12 (17%)	14 (70%)	**<0.0001**
Cognitive			
Yes (%)	62 (89%)	10 (50%)	
No (%)	8 (11%)	10 (50%)	**0.00014**
Headache*			
Yes (%)	68 (97%)	16 (80%)	
No (%)	2 (3%)	4 (20%)	**0.021**
Sleep disorder			
Yes (%)	46 (66%)	9 (45%)	
No (%)	24 (34%)	11 (55%)	0.094
Vestibular			
Yes (%)	44 (63%)	10 (50%)	
No (%)	26 (37%)	10 (50%)	0.301

Data presented as numbers with percentages in parentheses. ***Abbreviations:*** *PTSD, Post-Traumatic Stress Disorder.*

Table 3. Relative risks and 95% confidence intervals of symptom occurring in external blame group compared to the accidental group. Symptoms arranged from greatest relative risk (top) to least relative risk (bottom).

Symptom	Relative risk in external blame vs accidental	Relative risk in external blame vs accidental
PTSD	32.2	2.1 - 500.1
Depression	3.8	1.6 - 9.2
Anxiety	3	1.5 - 5.9
Irritability	2.8	1.4 - 5.4
Cognitive	1.8	1.1 - 2.8
Headache	1.2	1 - 1.5

Duration and recovery

The mean duration of concussion symptoms thus far, along with the mean SPR, were compared between the external blame group and accidental group with Mann-Whitney U tests. The mean duration of symptoms in the external blame group (mean=22.5, SE=$\pm$2.24) was longer than the duration in the accidental group (mean=10.2, SE=$\pm$1.52) (p=0.001). The external blame group SPR (mean=44, SE=$\pm$3.43) was lower than the accidental group SPR (mean=73, SE=$\pm$3.9) (p<0.0001).

Discussion

This study shows that patients with prolonged recovery from concussion who blame someone else for their concussion have a worsened prognosis compared to those who described their concussion as an accident. This worsened prognosis is composed of three aspects in our study. Firstly, with external blame attribution comes an increased relative risk of PTSD, depression, headache, anxiety, irritability, and cognitive symptoms. Secondly, patients with prolonged recovery who attribute blame externally had endured a longer course of symptoms at the time of the study. Thirdly, those with external blame demonstrated a reduced subjective percent recovered, even though they were on average further along in the course of the syndrome. The association between external blame and worsened prognosis in our study echoes the findings of others, who all saw external blame as a predictor of negative recovery outcomes.[16-18]

Considering the worsened outcome of patients who attribute external blame, it is concerning that the 77% of patients included in the study belonged to this group. It is still too soon to say what the exact relationship is between external blame attribution and prolonged recovery. The simplest answer is that an external party causing a concussion is itself an independent risk factor. We theorize that the removal of the locus of control from the patient during the incident leads to increased perceptions of trauma.

It could also be that external blame attribution is a manifestation of an inappropriate coping mechanism. We would suspect this if the stories of patients in the external blame group did not match independent incident reports. A hypothetical example of this phenomenon could be a police report stating that a patient who identified themselves in the external blame attribution group was actually at fault for collision, and should have been placed in the internal blame attribution group instead. While this study did not examine the accuracy of blame attribution, it should be considered in future studies on the topic.

Another possibility is that patients in the external blame attribution group are incentivized to be sicker, as their symptom severity may be seen as support for ongoing litigation or other forms of compensation. This incentivization hypothesis could also explain the increased number of patients in the external blame group. The possibility for reward by society would prompt those that experienced an ambiguous incident to characterize another as at fault, instead of describing the event as an accident. Previous research supports this theory, as it goes with the narrative that litigious societies are associated with worsened outcomes after similar motor vehicle collisions.[15]

Although the details of the relationship between prolonged recovery from concussion and blame attribution are unclear, this study demonstrates that blame attribution is associated with a more severe course after concussion. This association translates into real-world consequences for patients experiencing the syndrome. We believe the association between blame attribution and prolonged recovery presents an excellent opportunity for clinical intervention. A recent review on the subject suggests that counselling on resilience, cognitive behavioural therapy (CBT), and other psychotherapeutic interventions may be helpful for post-concussion syndrome.[19] CBT has been used to successfully treat symptoms of post-concussion anxiety and depression in athletes, symptoms which were common among our cohort of patients with PCS.[20] Therefore, we would like to see a prospective study in which blame attribution is targeted by psychotherapy in patients who have sustained a concussion. Perhaps this early intervention, which could reinforce the concept of avoiding a blame-based injury narrative, would lessen the severity of symptoms, or even lessen the occurrence of prolonged recovery altogether. A prospective study in which pediatric patients that have experienced a concussion receive significant educational and psychological interventions compared standard care is currently underway and promises to shed more light on this area.[21]

Our analyses of the blame group populations found no difference in age, sex, number of concussions, and previous illnesses. Based on this lack of difference, we expect the external blame attribution and worsened symptoms association is not being confounded by one of the previously mentioned variables. However, we recognize that the group variables that were collected in this study were not comprehensive. Future research on the topic by our team will include the collection of more data on the social determinants of health like ethnicity, income, neighbourhood, and occupation, as these could all be associated with symptoms severity and/or blame attribution. Similarly, a study that collects data on litigation status, return to work/play, and full recovery, would allow us to more closely delineate the influence of blame attribution from other independent variables.

This study was performed using a small patient population at a single subspecialty clinic, and so its generalizability may be limited. As well, the retrospective design was lacking in a concussion positive, prolonged recovery negative control group, which limited our ability to comment on external blame attribution as a risk factor for the development of prolonged recovery. The clinical team that cared for the concussion patients was not blinded to the study, which may have biased their EMR reporting.

Conclusion

Patients with prolonged recovery from concussion are more likely to attribute external blame for their concussion, compared to internal blame or describing the incident as accidental. Those that attribute external blame experience more symptoms, for longer, with a lower subjective rate of recovery. This association between external blame attribution and worsened clinical picture is not explained by age, sex, number of previous concussions, or pre-existing mental illness.

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Conflicts of Interest

None to declare

Ethics Approval

University of Alberta REB 4: HREB

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