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Post-traumatic stress disorder vs traumatic brain injury

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Abstract

Post-traumatic stress disorder (PTSD) and traumatic brain injury (TBI) often coexist because brain injuries are often sustained in traumatic experiences. This review outlines the significant overlap between PTSD and TBI by commencing with a critical outline of the overlapping symptoms and problems of differential diagnosis. The impact of TBI on PTSD is then described, with increasing evidence suggesting that mild TBI can increase risk for PTSD. Several explanations are offered for this enhanced risk. Recent evidence suggests that impairment secondary to mild TBI is largely attributable to stress reactions after TBI, which challenges the long-held belief that postconcussive symptoms are a function of neurological insult This recent evidence is pointing to new directions for treatment of postconcussive symptoms that acknowledge that treating stress factors following TBI may be the optimal means to manage the effects of many TBIs,

Keywords: *post-traumatic stress disorder*, *traumatic brain injury*, *trauma*, *postconcussive syndrome*

Overview

The intersection between traumatic brain injury (TBI) and post-traumatic stress disorder (PTSD) has become a major focus of attention in recent years. Stimulated largely by injuries sustained in the Iraq and Afghanistan wars, this issue has been debated widely because these conditions, both independently and additively, are regarded as being responsible for much impairment following deployments. This review will commence with defining these conditions, explain potential overlaps between them, and discuss the differential diagnosis challenges of determining the extent to which presenting symptoms can be attributed to organic or psychological factors. The review then discusses evidence of PTSD following TBI, and possible mechanisms that may impact on the nature of PTSD following TBI. The respective roles of PTSD and TBI in impairment after TBI are then addressed, with specific focus on the understanding of postconcussive symptoms. Finally, the implications for managing the effects of TBI and PTSD are discussed in terms of recent developments in how each condition can affect the other.

Definitional issues

TBI

TBI involves damage to the brain from an external force.Brain injuries can involve contusion, brain laceration, intracranial hematoma, contrecoup injury, shearing of nerve fibers, intracranial hypertension, hypoxia, anemia, metabolic anomalies, hydrocephalus, and subarachnoid hemorrhage. Severity of TBI is typically described in terms of mild or moderate/severe; however, the exact definitions vary. Mild traumatic brain injury (MTBI) is usually defined as: (i) an external injur}' to the brain; (ii) confusion, disorientation, or loss of consciousness for 30 minutes or less; (iii) Glasgow Coma Scale score of 13 to 15; and (iv) post-traumatic amnesia for less than 24 hours.[1-3] Moderate TBI often involves loss of consciousness between 30 minutes and 24 hours, Glasgow Coma Scale score of 9 to 12, and post-traumatic amnesia between 1 and 7 days. Severe TBI involves more extended loss of consciousness and post-traumatic amnesia, which typically results in more severe cognitive impairment. These differences in TBI severity are important because they appear to interact differentially with PTSD.

PTSD

It is important to distinguish between immediate and longer-term PTSD reactions. Most diagnostic systems have distinguished between these two types of trauma response because acute stress reactions are frequent, but often transient, and they need to be distinguished from the less common persistent PTSD responses. In terms of the persistent responses, PTSD is described in the American Psychiatric Association's *DSM-IV* as an anxiety disorder that comprises five major criteria.[4] First, one must have been exposed to or witness an event that is threatening to safety, and one must respond to this event with fear, horror, or helplessness. Second, one must report a re-experiencing symptom, which may include intrusive memories, nightmares, a sense of reliving the trauma, or psychological or physiological distress when reminded of the trauma. Third, there need to be at least three avoidance symptoms, which can include active avoidance of thoughts, feelings, or reminders of the trauma, inability to recall some aspect of the trauma, withdrawal from others, or emotional numbing. Fourth, one must suffer marked arousal, which can include insomnia, irritability, difficulty concentrating, hypervigilence, or heightened startle response. These symptoms must cause marked impairment to one's functioning, and can only be diagnosed when they are present at least 1 month after the trauma.

DSM.-IV also introduced a new diagnosis, acute stress disorder (ASD), to describe acute trauma reactions that occur in the initial month following a trauma. As PTSD is only diagnosed 1 month after trauma, it was decided that there was a need to fill the nosological gap between the traumatic event and PTSD, in part to facilitate diagnosis and access to health care. ……..

Differential diagnosis

A key issue in this discussion is the overlap between symptoms accompanying each condition. In terms of the dissociative symptoms often observed in PTSD, and especially in the acute phase in ASD, there is much evidence that TBI can result in emotional numbing, derealization, reduced awareness of surroundings, depersonalization, and amnesia.[13-15] The issue of amnesia is particularly important in cases of TBI and PTSD because of the difficulty in differentiating between organic and psychogenic amnesia.[16] Some commentators have adopted the approach of excluding dissociative amnesia as a possible symptom of ASD and PTSD following TBI to reduce the likelihood of falsely increasing diagnostic rates.[17,18] In diagnosing PTSD, it is probably safer to not include dissociative amnesia as a potential symptom.

……. A further complicating issue in the differential diagnosis between PTSD and TBI is the range of other comorbid problems that commonly coexist with both TBI and PTSD. For example, depression is highly prevalent with both conditions. Numerous studies have suggested that TBI increases the risk for developing depression,[29,30] eg, refs 31,32,33. Some of the core symptoms noted across TBI and PTSD are also seen in depression, especially the more severe forms of TBI, including concentration problems, memory problems, irritability, reduced motivation, and fatigue. Highlighting this problem in one study was a finding that more than 50% of depressed patients met symptom criteria for moderate/severe postconcussive syndrome.[34] This contributes to the conclusion that some of the symptoms attributed to TBI may in fact be generic symptoms of

psychological malaise, which are observed across anxiety and depressive responses. Complicating the issue of comorbidity is compounded by the fact that TBI, PTSD, and depression commonly occur in the context of chronic pain, which also results in symptoms that overlap with each of these conditions.[35-41]

Prevalence

PTSD and TBI are not uncommon. Epidemiological studies indicate that most people in the community have been exposed to traumatic stressors,[42,43] although anly a minority develop PTSD. For example, the National Comorbidity Survey found that 21 % of the women and 8% of the men had developed PTSD.[42] Similarly, a Detroit study found that 13% of the women and 6% of the men had developed PTSD.[43] That is, although men are more likely to be exposed to trauma than women, women have at least a twofold risk of developing PTSD compared with men. [44] More severe traumas tend to result in more severe PTSD. Interpersonal violence leads to more PTSD than impersonal trauma; for example, whereas 55% of rape victims develop PTSD, only 7.5% of accident victims develop PTSD:[42,45]

In terms of TBI, there are between 1.5 and 2 million people in the USA alone who sustain a TBI, with approximately 70 000 to 90 000 experiencing persistent functional difficulties.[46] The Centers for Disease Control and Prevention estimates that approximately 5.3 million people in the USA are living with a disability due to TBI.[47] Certain populations appear to be more at risk of sustaining TBIs. For example, military estimates of mild TBI of deployed (non-mcdically evacuated) personnel indicate that between 10% and 20% may have suffered a mild TBI during deployment.[48] One study reported a rate as high as 23% in personnel assessed after returning to the USA.[49]

Postconcussive syndrome and PTSD

The issue of postconcussive syndrome is a vexed one, both in terms of its definition and its purported causes. It is also an issue that intersects with symptoms of PTSD. PCS is generally defined as a syndrome that involves headache, dizziness, fatigue, sensitivity to light or sound, sleep disturbance, and concentration difficulties.[93] The definitions of PCS vary, and generally overlap somewhat with symptoms of PTSD. For example, the *International Classification of Diseases (ICD-10)* [26] stipulates that PCS is defined by headaches, dizziness, general malaise, fatigue, noise intolerance, irritability, emotional lability, depression, or anxiety, concentration or memory difficulty, sleep disturbance, reduced tolerance to alcohol, and a preoccupation with these symptoms and fear of permanent brain damage. The Appendix of the *DSM.-IV* [4] describes PCS as fatigue, sleep disturbance, headaches, dizziness, irritability, anxiety or depression, changes in personality, and apathy. These descriptions clearly overlap with common symptoms of post-traumatic stress, and represent differential diagnosis problems insofar as how one attributes these symptoms to PCS or PTSD.

Recent evidence is highlighting that symptoms described as PCS are common in many populations, and actually reflect a diffuse collection of frequently experienced sensations. In healthy individuals, headaches, sleep difficulty, irritability, and memory failures are relatively common in daily life.[97-98] One study found that 72% to 79% of healthy adults reported at least three or more PCS symptoms; further, a significant minority of subjects met *DSM-TV* (14.6%) or *FCD-10* (12.5%) criteria for PCS.[99] Interestingly, these observed rates of PCS in non-MTBI are comparable to the rates noted in TBI populations, highlighting the fact that PCS are not unique to TBI.

There has been much debate over the extent to which persistent PCS develops as a result of neurological damage,[100] psychological distress,[101] or a combination of both.[102] One recent study that assessed PCS in both MTBI and non-MTBI injured patients found that comparable proportions of patients reported PCS (MTBI: 40%; no-TBI: 50 %).[103] A subsequent follow-up at 3 months post-injury found that a similar pattern (mild TBI: 46.8%; control: 48.3%).[104] Interestingly, across these studies, PCS was predicted by pain levels and PTSD symptoms. rFh cse data indicate that PCS is not unique to MTBI, and that these symptoms that are commonly

attributed to MTBI are more parsimoniously explained by the effects of high arousal associated with the stress of surviving a traumatic injury.

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The problem of confusing MTBI and PTSD

Military agencies have implemented programs for troops in Iraq and Afghanistan targeted towards treating the effects of MTBI. Much attention has been given to the "problem" of mild TBI, communicating to troops that MTBI is a syndrome that causes marked problems. Given the evidence that so-called postconcussion-like symptoms and general health problems are largely related to psychological factors, there are likely risks in suggesting to troops that the problems experienced following MTBI should be attributed to neurological damage. Communicating to personnel who sustained a MTBI that a range of nonspecific symptoms are caused by brain damage communicates a cause with a poor prognosis. This expectation that common sensations are signs of permanent dysfunction can result in hypervigilance to every sensation, followed by catastrophic attributions about the adverse consequences of the sensations. This pattern has been well-documented across a range of disorders, including panic disorder, health anxiety, and hypochondriasis.[105-107] In these disorders, people tend to be hypervigilant to somatic cues because they believe they represent a threat to their physical well-being. For example, the patient with panic disorder may believe that an alteration in his or her respiration is a sign of imminent choking or that a slight pain in the chest is indicative of an approaching cardiac arrest. Similarly, someone with health anxiety may constantly search their body for any alterations in appearance of function to determine if there arc signs of malignancy. Once the sensation or sign is detected, the person can catastrophize the sign in an extremely negative manner, such that the slightest somatic cue is perceived as indicative of dire outcomes. This is a common pattern in people with PTSD. Fear network models of PTSD propose that these individuals preferentially allocate attention to stimuli of concern because of their fear of threat.[108] Consistent with this proposal, people with PTSD are hypervigilent to threat on a range of paradigms.[109-111] Further, people with PTSD not only catastrophize about external threats,[112] they also catastrophize about somatic and physical sensations.[113] Therefore, people who are suffering the effects of PTSD will be attentive to any information that is perceived as threatening, and will likely attribute a range of physical, cognitive, and emotional responses to brain injury if this is provided as a salient explanation. This response may exacerbate the PTSD reaction, as well as promote continued hypervigilence to sensations and subsecpent maladaptive appraisals that these reactions arc indicative of permanent brain injury.

This pattern was reflected in the aftermath of the 1991 Gulf War, when there were widespread concern of chemical weapons, which apparently contributed to medically unexplained symptoms that were linked to concerns about somatic sensations purportedly linked to chemical agents. [106,114,115] It seems that a cohort of soldiers after the 1991 Gulf War misattributed somatic experiences to chemical agents, which led to persistent concerns about their health. There are potential similarities between Gulf War Syndrome and the manner in which MTBI is currently being understood; both comprise general sensations that are commonly reported in stress responses, and both mistakenly attributed to common stress reactions. This can be problematic because it can reduce people's optimism or expectancy for recovery.

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Implications for treatment

This review has several implications for how symptoms following TBI are addressed in treatment. In terms of treating the symptoms of PCS, current evidence suggests that simple neuropsychological education is modestly useful in reducing symptoms of PCS.[116] The emerging evidence that PCS is predominantly influenced by posttraumatic stress reactions suggests that addressing these problems may be crucial in alleviating PCS. That is, by reducing the arousal-inducing symptoms of PTSD, it is possible that many of the symptoms associated

with PCS will be alleviated. Similarly, by minimizing catastrophic appraisals that exaggerate the severity or adversity of PCS sensations it is probable that anxiety about these reactions would be eased. For example, patients who are overly concerned about the adverse outcomes of dizziness or sensitivity to light can be taught to normalize these reactions in ways that minimize distress about these sensations. Cognitively reframing the perception of these reactions is akin to established treatments for panic disorder or health anxiety, in which patients are taught to tolerate somatic experiences in ways that discourage inferences involving an adverse outcome. Although this approach has been proven to be very effective in treating panic disorder[117] and health anxiety,[118] it has yet to be tested with PCS.

In terms of treating symptoms of PTSD, prevailing cognitive models posit that recovery from a traumatic experience involves integrating the trauma memory into one's autobiographical memory base in a way that allows a coherent narrative of the experience in which the person can contextualize the experience and consequently currently feel safe.[112] This perspective proposes that a major reason trauma memories are difficult to integrate into memory is the manner in which they are encoded[119]; specifically, experiences are often fragmented because they are encoded under conditions of extreme arousal, and this purportedly disturbs the ability to form the required coherent narrative. Fragmented memories of the traumatic experience can also occur in the context of TBI because of the impaired consciousness secondary to the injury. As noted above, TBI patients can reconstruct aspects of the traumatic experience that were not adequately encoded during the period of impaired consciousness. This scenario raises the possibility that treating PTSD after TBI will require adaptive reconstruction of this narrative in a way that facilitates adaptation rather than retraumatization. For example, a patient who reconstructs their memory of a car accident in which they were excessively responsible for someone's death will have marked depressive responses relative to a patient who reconstructs the memory in a way that accepts a more reasonable level of responsibility. Alternately, a patient can be encouraged to tolerate a level of uncertainty insofar as there is permanent amnesia of some aspect of the event; inability to tolerate uncertainty is linked to enhanced anxiety and worry.[120] One of the challenges for treating PTSD after TBI is the patient's ability to either reconstruct events in a coherent and adaptive way or to accept the uncertainty of how events transpired when they suffered their TBI.

The extent to which a person with TBI needs to reconstruct the trauma narrative to recover from PTSD has yet to be empirically determined. As noted above, several large-scale studies have reported that MTBI is associated with increased risk for PTSD.[59,92,78] One possibility for this observation may be that people who sustain a MTBI do not have a coherent narrative of their traumatic experience because of the impaired consciousness secondary to the brain injury, and this may impede their capacity to contextualize the experience in their autobiographical memory base.

A second implication for PTSD treatment after TBI is that the treatment of choice for PTSD involves traumafocused exposure therapy.[121] This treatment is based on extinction learning, which occurs when a conditioned stimulus is repeatedly presented in the absence of an aversive outcome, thereby facilitating new learning that the stimulus is no longer signaling threat. In the context of therapy, presenting memories or reminders of the trauma to the patient in the safety of therapy typically leads to symptom reduction. Exposure can either be *imaginai,* which involves focusing on one's memories of the traumatic event, or *in vivo,* in which approaches and remains with reminders that usually trigger anxiety about the event. On the premise that fear conditioning and extinction still occurs in the context of TBI, it would seem that that exposure-based therapy is the indicated intervention for PTSD following TBI. Supporting this conclusion is evidence in one controlled trial of patients with acute stress disorder following MTBI that CBT effectively treated PTSD symptoms to a similar extent as when applied to non -TBI samples.[122]

Imaginal exposure with people following TBI will usually be dependent on the amount of memory that the patient is reporting. It may not be as useful to patients with more severe TBI because they are largely amnesic of their trauma. As noted above, some severe TBI patients can have nightmares or intrusive memories on the basis of reconstructions of their trauma; in these cases, imaginable exposure to those mental representations that are causing anxiety. In most cases of moderate/severe TBI, however, it is more useful to employ in vivo

exposure because reminders of the trauma can elicit stronger anxiety in the absence of actual memories or images. A survivor of a motor vehicle accident who sustained a severe TBI may experience marked fear when watching film footage of traffic; in such a case, the patient could complete exposure by repeatedly watching traffic footage. Through these techniques it would be hoped that extinction learning can be achieved, even though the patient may never retrieve direct memories of the traumatic event.

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Conclusions

The coexistence of TBI and PTSD is frequent, and the extent to which the symptoms of TBI and PTSD are confused may be as frequent. Increasing evidence indicates that many previously termed PCS responses are a function of psychological responses, and it hampers a patient's recovery if they mistakenly perceive these reactions as indicators of a brain injury that may be permanent. In this sense, the field is recognizing the distinction between TBI as an event rather than a syndrome, whereas PTSD or PCS are symptoms that arise secondary to the event. The likelihood that the presumed secpelae of MTBI are actually attributed to psychological responses to the traumatic experience is becoming more apparent. Accurate identification of the true nature and cause of the symptoms experienced after TBI is important because if stress-related disturbances are mistakenly attributed to neurological factors, patients may be deprived of effective treatments that can, in most cases, alleviate the symptoms. As we learn more about the interaction of TBI and PTSD, it seems that we will be discovering much about how the brain responds to traumatic experiences, both in cases when there has and has not been a TBI. Understanding this interaction between neurological insult and psychological response has the potential to shed light on the key mechanisms underpinning trauma response generally, and how it is impacted by different levels of brain injury.

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