

An Over View: Neuropathologic Changes and Neurometabolic Cascade in Traumatic Dementia Brain Injury

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Abstract

The pathology of a TBI is varied and typically classified as focal or diffuse. By definition, focal injuries are associated with moderate-to-severe TBI and typically result from direct head or brain impact (for example, gunshot wounds, blunt force, etc.). Cortical or subcortical contusions, lacerations, and intracranial bleedings (such as subarachnoid hemorrhage and subdural hematoma) are examples of focal injuries. After a traumatic brain injury (TBI), brain contusions are common, typically affecting the frontal and temporal poles, lateral and inferior aspects of the frontal and temporal lobes, and, less frequently, the inferior aspects of the cerebellum.

Keywords: Less frequently; Cerebellum; Pathophysiology

Introduction

The term “diffuse brain injury” refers to a variety of pathologies, including hemorrhages and tissue tears throughout the brain, that cause widespread stretching and tearing of brain tissue. It frequently occurs following acceleration and deceleration injuries (such as motor vehicle accidents). Shearing of neuronal axons is the most common form of diffuse injury, and it is referred to as diffuse axonal injury (DAI) [1]. Due to mechanical forces, the brainstem’s cerebral commissures and other white matter tracts are particularly susceptible to stretching and shearing. Reduced levels of arousal and the variety of neurologic deficits following brain injury may be primarily caused by the extent of DAI [2].

Brain trauma resulting from mTBI is typically not observable on standard structural neuroimaging, despite obvious transient cognitive and behavioral changes. However, it cannot be denied that mTBI causes a brain metabolic change or some diffuse, likely microscopic trauma. Cognitive and behavioral changes observed following mTBI are most likely the result of a multifaceted neurometabolic cascade, according to recent animal studies [3]. Neurotransmitters and ionic shifts, such as increases in extracellular potassium, sodium, and calcium, are thought to have been released abruptly and indiscriminately as a result of stretching and disruption of the neuronal and axonal cell membranes. Glutamate’s increased release, which binds to N-methyl-D-aspartate receptors, causes further depolarization, the influx of calcium ions, widespread suppression of neurons, and glucose hypometabolism.

As membrane pumps work harder to restore ionic balance, glucose consumption rises, further depleting energy stores. Endothelial calcium accumulation results in decreased cerebral blood flow and glucose availability in addition to this [4]. A widespread cellular crisis is brought on by the gap between glucose supply and demand. The majority of the studies that have been presented previously describe TBI in civilian populations. This whole process is brief, and within a few days, homeostasis is restored. The cognitive and behavioral effects of TBI on military personnel have been the primary focus of recent research due to the high rates of TBI among veterans and military personnel. These studies build on what civilian literacy has learned while also taking into account the particular characteristics of this particular population [5], which may have various injury mechanisms, risk factors, and comorbidities. Acute and chronic TBI symptoms, as well as frequently occurring comorbid conditions, have been the subject of research. It has been documented that military personnel experience severe mTBI effects. Headaches, dizziness, memory issues, balance issues, and irritability are the most frequently reported postconcussion

symptoms, just as they are for civilians.

Methods

Computerized testing reveals cognitive deficits in deployment-related mTBI patients during the acute postinjury phase, which occurs between three and ten days after the injury. These prospective studies demonstrate, in agreement with civilian research, that cognitive deficits resolve within days to weeks. Retrospective studies of more distant history of mTBI (for example, months to years after injury) during deployment typically do not reveal persistent cognitive effects or symptoms. For instance, for individuals who had returned from deployment within the previous two years, self-reported TBI during deployment did not increase the risk of cognitive impairment on postdeployment cognitive testing. In a study of the effects of deployment on cognition, a history of TBI had no effect on outcomes. 70% of service members who reported a history of mTBI during deployment showed no change in cognitive functioning in a study of cognitive change from baseline to routine postdeployment cognitive testing. However, a subset of people who reported both mTBI and ongoing no cognitive symptoms saw a decrease in performance when compared to the control group. Given the high prevalence of comorbid conditions among service members who have a history of traumatic brain injury [6] (TBI), particularly posttraumatic stress disorder (PTSD) and mood disorders, it is unknown from this study whether these declines are the result of the persistent effects of mTBI or some other comorbid condition. Subsets of people with a history of mTBI continue to report persistent cognitive complaints and somatic symptoms consistent with concussion, according to other retrospective studies of the chronic effects of mTBI.

Results

However, there is evidence to suggest that these persistent symptom

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complaints may be caused by non-neurologic conditions like emotional distress or PTSD rather than by mTBI. Others contend that the particular type of TBI associated with the military (such as blast injuries, repetitive injuries, etc.) may account for symptomatic cognitive and noncognitive recovery that is persistent or atypical. The likelihood of recurring mTBIs is high when deployments are prolonged and repeated [7]. Civilian research suggests that people with a history of multiple concussions are more likely to experience more severe symptoms and take longer to recover from multiple mTBIs, especially when they occur in close proximity. Initial research among veterans and members of the armed forces suggests that people who have suffered multiple mild brain injuries are more likely to complain of postconcussion syndrome and to report more symptoms in the immediate aftermath of the previous injury. Animal studies have led some to believe that blast injuries, which are common in deployment settings, may result in brain neuropathologic changes that are more severe and may last longer (for a review, see). However, clinical differences in the acute or chronic effects of blast injury versus nonblast injury on cognitive performance or somatic symptoms have not been documented in any studies to date.

Discussion

Additionally, there is a significant degree of overlap between TBI and a number of comorbidities, such as PTSD, anxiety, mood disorders, and other mental health diagnoses, as shown by the screening-based survey data. It is estimated that 30% of people who test positive for TBI also have PTSD and mTBI. A history of traumatic brain injury has been linked to a higher risk of PTSD symptoms.

Clinical diagnoses of PTSD, other anxiety disorders, and adjustment disorders were more common in veterans with a confirmed diagnosis of TBI. When these two conditions co-occur, it is thought to have additive effects on the brain, causing TBI to last longer and cause more cognitive problems. As depression can impede recovery, exacerbate neuropsychological impairment, and worsen overall outcomes, other studies demonstrate a specific increased risk for depression following a TBI across the spectrum of TBI severity. Some studies show an increased risk, even among those with milder injuries, of suicide after a TBI; other studies, including a large review conducted by the Institute of Medicine (IOM), conclude that there is no link between TBI and suicide [8]. The effects of a traumatic brain injury (TBI) can be exacerbated by other factors like persistent pain and disturbed sleep. A meta-analysis found that 43.1% of military personnel had pain disorders, and even when mental health diagnoses were controlled, TBI had an independent correlation with pain disorder diagnosis. 70% of veterans with TBI who participated in the study also had a diagnosis of head, back, or neck pain. After a TBI, insomnia is a common complaint. The link between TBI and later-life dementia risk has been repeatedly established in the literature. Sleep problems are significantly associated with TBI in military populations, and the risk of sleep disturbances increases with a history of multiple mild TBIs.

Conclusion

In particular, a prospective study of World War II veterans found

that veterans with a history of moderate-to-severe TBI had a two- to fourfold increased risk of dementia. According to other retrospective studies, individuals with dementia are more likely than controls to have a history of moderate-to-severe TBI earlier in life. Systematic reviews show that people with a history of at least one moderate-to-severe traumatic brain injury (TBI) are more likely to develop dementia than people without a history of TBI. However, given that some epidemiological studies failed to demonstrate a link between TBI and later dementia, consensus is not complete. A subsequent reanalysis of these studies confirmed the positive association between the development of DAT and a history of previous head trauma, despite some contradictory findings. DAT onset is accelerated at younger ages when there is a history of TBI, and the risk of developing DAT increases with TBI severity, according to other studies.

The risk of dementia following mTBI is less clear. According to previous systematic reviews and an IOM report, a history of mTBI without LOC does not raise the risk of DAT later in life. Veterans of World War II who had suffered from mTBI did not also show any increased risk for dementia. However, there is evidence to suggest that chronic mTBI, accumulated neuropathology, and eventually dementia may result from repeated mTBI. Since the 1920s, there have been connections between professional boxing and later dementia, and corresponding changes in neuropathology have been reported as early as the 1970s.

Declaration of Interest

The authors declared that there is no conflict of interest.

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