

Review on Pathology of Dementia after TBI

Rahman Farheen*

Department of pathology, University of California, USA

Abstract

Despite the growing body of evidence indicating an increased risk of DAT following a TBI, the specific underlying pathology responsible for this risk remains a mystery. The majority of mechanistic explanations focus on a presumption of a neuropathologic trigger that is activated at the time of the injury and continues to evolve over time, eventually leading to dementia [1]. Axonal damage is a hallmark of both traumatic brain injury (TBI) and Alzheimer's disease (AD), so many have looked into this possibility as a link between TBI and dementia. Amyloid- β (Ab), the primary protein most frequently associated with AD, has been shown to accumulate within neuronal cell bodies and injured axons within hours to days of TBI, according to human and animal models [2]. Amyloid precursor protein (APP) expression rises in injured axons and neuronal cell bodies in response to trauma, resulting in an increase in Ab production. It is hypothesized that this accumulation of APP and Ab plays a significant role in the subsequent Ab plaque formation, which is one of the hallmarks of AD. Additionally, it is hypothesized that the APOE 4 genotype may influence amyloid pathology and TBI outcome, putting individuals with this allele at increased risk for AD [3].

Keywords: Dementia; Amyloid pathology; Neuronal cell

Introduction

NFTs are also thought to be a major AD pathologic symptom. Tau proteins with abnormal phosphorylation make up NFTs, which are thought to be neurotoxic and may lead to the death of neurons. Tau accumulation has been observed in humans and animals following TBI. NFTs, on the other hand, have not been found to be significantly elevated following a single TBI, unlike Ab. Instead, it appears that multiple repetitive mTBIs increase the risk of early-onset behavioral and cognitive decline through accumulated tau protein pathology with little to no Ab deposition [4]. The brain's inflammatory response after a traumatic brain injury has been extensively studied. In addition, despite the fact that Ab formation and the tauopathy that goes along with it may appear to adequately explain the potential connection between TBI and dementia, the same proteins can also initiate processes that lead to inflammation. Despite the fact that acute inflammation is to be expected following a TBI, there is growing evidence that the inflammatory response may persist over time, suggesting that the initial effects of a TBI may last longer than previously thought.

In animal models and postmortem human studies, inflammation in the brain persists after a traumatic brain injury for at least a year. A recent study that looked at the inflammatory response to brain injury in vivo using positron emission tomography found that microglial activation increased up to 17 years after the injury, with activation in the thalamus being linked to more severe cognitive impairment [5].

Methods

As a result, traumatic brain injury (TBI) may set off an inflammatory response, particularly in subcortical areas, that may last and continue to develop over time. TBI-related dementia, neurodegenerative, or cerebrovascular disease may be sparked by this persistent inflammation as a precursor to a larger cascade. Given the findings that elevated inflammatory markers predict cognitive decline decades later, the presence of inflammation is concerning. Additionally, the recent findings of an increased risk of stroke in people with a history of TBI may be explained by persistent inflammation in TBI [6]. Moderate-to-severe traumatic brain injuries (TBIs) are frequently straightforward to identify and treat due to the obvious immediate effects of the injury (such as LOC, marked confusion, and coma). In empirical studies, the DoD and the VA are actively following these injured service

members over time. Longitudinal follow-up and data monitoring will help to clarify the initial care that helped to improve outcome and the natural course of brain injury and polytrauma over the lifespan for many service members who suffer not only brain injury but also other systemic injuries (such as amputation and sensory loss). Mild TBI, in contrast to moderate-to-severe TBI, can have subtle and difficult-to-detect initial symptoms [7].

Results

This is especially true in combat, where symptoms of mTBI may be mistaken for deployment stress or other psychological trauma or shock. The Department of Defense (DoD) initiated a number of policies and programs to assist in the detection of mTBI and its sequelae in the deployed environment. One of these policies and programs was the implementation of a neurocognitive baseline assessment program prior to deployment that enables comparisons to be made between injuries [8]. The Department of Defense established a policy that mandated screening, standardized evaluation of symptoms, documentation of the event, symptoms, and diagnosis following potentially concussive events [156]. The Department of Defense (DoD) updated their clinical care algorithms to take into account the most recent findings from theater-based research after this policy was implemented [9]. The Department of Defense (DoD) continues to place an emphasis on mTBI detection by mandating screening at multiple points in time (such as the injury site, prior to medical evacuation to the United States, and prior to redeployment).

Recent revisions to survey questions aimed at postemployment TBI detection and postemployment reassessment (see Section 8.1) encouraged symptom reporting to connect service members with care. In polytrauma centers, where other critical or life-threatening

***Corresponding author:** Rahman Farheen, Department of pathology, University of California, USA, E-mail: farheen@gmail.com

Received: 2-Jan-2023, Manuscript No: dementia-23-86433, **Editor assigned:** 4-Jan-2023, Pre QC No: dementia-23-86433 (PQ), **Reviewed:** 18-Jan-2023, QC No: dementia-23-86433, **Revised:** 23-Jan-2023, Manuscript No: dementia-23-86433 (R), **Published:** 30-Jan-2023, DOI: 10.4172/dementia.1000144

Citation: Farheen R (2023) Review on Pathology of Dementia after TBI. J Dement 7: 144.

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multisystem injuries may mask initial symptoms of mTBI, there are also coordinated efforts to screen for TBI. In the future, efforts are being made to evaluate the efficacy of biomarkers, neuroimaging, and other innovative methods for definitively diagnosing TBI. The Post-Deployment Health Assessment/Post-Deployment Health Re-Assessment (PDHA/PDHRA) from the Department of Defense or the Veteran's Health Administration's TBI Screening Questionnaire are used to screen for TBI because of the growing concern about the health effects of TBI. These tests look for ongoing symptoms and the possibility of being exposed to risk factors. However, the service member's willingness to report can be significantly affected by when these measures are administered. In order to avoid lengthy follow-up evaluations, some service members may minimize symptoms. Others might not realize how bad their symptoms are until they get home and go back to their normal activities, or they might try to hide how bad they are. The course of symptom relief and recovery can be adversely affected by treatment delays; Consequently, it is essential to continue making efforts to better identify injuries as soon as possible after they occur. There are now mandatory evaluations and prescribed algorithms for follow-up care for those who are thought to be at risk for TBI as the DoD continues to develop its care model in theater. In order to connect Service members with care, the questions that are asked during the PDHA and PDHRA have been refined to address underreporting and encourage symptoms to be acknowledged [10].

Discussion

The focus on screening and follow-up evaluation enables better evaluation of long-term outcomes and dementia risk, records multiple exposures and/or injuries in the population, and provides documentation of a TBI diagnosis. In order to address the growing concern regarding the possibility of cognitive insult during military deployment, Congress mandated a baseline predeployment neurocognitive assessment for all US service members in 2008. Within concussion monitoring and management programs for preventing or reducing concussion risk, the empirical validity of baseline cognitive testing has recently been questioned. Although some studies have suggested that civilian concussion monitoring programs do not benefit from baseline testing, there is evidence that baseline testing reduces the likelihood of false-positive concussion detection in healthy service members. 66% of individuals classified as "atypical" actually showed no change from baseline when norm-referenced postdeployment scores were considered in isolation, according to a large study of military service members (n = 5 8002). A person's cognitive trajectory can be tracked over time and factors that cause a change from baseline can be identified through baseline testing, especially testing that can be repeated over time. The sensitivity of dementia monitoring protocols could be increased by monitoring these results over time and controlling for the effects of aging or other typical causes of cognitive change.

Conclusion

The advantages of longitudinal monitoring are hypothetically demonstrated. These examples show how service members' diagnoses and clinical management might benefit from longitudinal testing. The standardized cognitive testing scores, represented by the y-axis (mean 5 100; average deviation includes a representation of a person who had a mTBI while they were deployed. This individual exhibits a drop in cognitive performance of approximately two standard deviations following the injury. However, over time, their functioning returns to baseline, and they continue to perform at this level on routine

postdeployment testing. This provides an illustration of the expected recovery of functioning as well as an example of a possible false-positive error in someone who was premorbidly functioning below average before deployment. This individual performed two standard deviations below the mean on cognitive tests following a suspected concussion-causing injury. Post-injury performance may be interpreted as evidence of a concussion-related cognitive impairment absent additional information. However, a longitudinal assessment reveals that cognitive functioning remained stable at follow-up testing points and that the individual did not exhibit any changes from baseline. This person may receive unnecessary treatment and be misdiagnosed as having had a concussion if longitudinal testing, which includes a baseline assessment, is not used. demonstrates a hypothetical case of late-onset symptoms in a person who has had a previous mTBI. After the documented mTBI, longitudinal testing clearly demonstrates a successful cognitive recovery. Late-onset symptoms may incorrectly be attributed to the previous mTBI if longitudinal testing is not utilized. The clinician would be better equipped to investigate and treat more precise etiologies of these symptoms if routine cognitive screening was available. Last but not least, although it is not shown in this study, longitudinal cognitive testing over the course of a person's lifetime, when the expected effects of aging are taken into account, would make it possible to identify future functional declines that, if found to be progressive in nature, could indicate the onset of a neurodegenerative process.

Declaration of Interest

The authors declared that there is no conflict of interest.

Acknowledgement

None

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