COMMENTARY

Models of blast TBI, and the implications for seizure risk

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ABSTRACT

TBI caused by explosive blast exposure is a primary cause of battle casualties. It is also suspected of being a major factor to war-related mental health disorders. A clinically significant outcome of all forms of TBI is an increased likelihood of developing seizures and epilepsy. Seizures have been observed in patients with bomb injuries during the Global War on Terror, although the actual occurrence is unknown. Seizures reinforce the theory that an explosive blast causes both cellular and structural brain damage. Unfortunately, the precise mechanism by which explosions induce brain damage remains unknown, complicating the development of effective medicines and mitigation techniques. A thorough neuropathological examination is required to aid comprehension. Histopathological procedures are incredibly helpful and necessary for this. Following that, we will go over the pathology findings from recent preclinical explosive explosion investigations, including those using immunohistochemistry and special staining methods.

Key Words: *Neurological illnesses*

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Blast-related Traumatic Brain Injury (TBI) is a common result of explosive device detonation exposure. The use of Improvised Ex-plosive Devices (IED), Vehicle-Borne IED (VBIED), and Improvised Rocket Assisted Mortars (IRAM) during the Global War on Terror (GWOT), which included both Operation Iraqi Freedom (OIF) and Operation Enduring Freedom (OEF) in Afghanistan, resulted in a significant number of blast-related TBI. During the over ten years of the GWOT, about 290,000 US military personnel got TBI, with explosive blast exposure accounting for 68% of the cases. When compared to prior conflicts in which similar protective equipment was not deployed, the deployment of Individual Body Armour Systems (IBAS) significantly reduces the occurrence of deadly thoracic and abdominal battle related injuries. As a result, many troops live who would not have survived if they had not worn IBAS. As a result of increasing survivability, blast-related TBI became more common than in prior battles. These victims suffer from a variety of neurological illnesses, ranging from modest moderate cognitive impairment, which affects a person's capacity to work under stressful settings, to severe disruption of brain function as serious as coma. These effects might be either momentary or long-term. If the latter, they can have a long-term detrimental impact on patients and their families, causing severe emotional and financial distress. It is uncertain how often epilepsy is among GWOT TBI patients. Based on information from previous wars, it is estimated that between 10%–25% of patients with closed head TBI and more than 50% of patients with penetrating TBI may develop Post-Traumatic Epilepsy (PTE). According to the Department of Defense, penetrating injury accounts for 1.5% of all combat-related GWOT TBI. PTE can manifest as any kind of epilepsy, although Temporal Lobe Epilepsy (TLE) predominates, accounting for up to 62% of TBI patients. Clinicians should be aware that up to 15% of TBI patients from previous conflicts did not experience seizures until five or more years following their injury. Recognizing this, the Veterans Administration (VA) has set up a national network of Centers of Excellence. Appropriate clinical and military-relevant experimental animal methodologies are required to define blast TBI injuries and pathologies. The injury model should be repeatable, with a clearly identifiable injurious component that mimics the characteristics of human blast TBI. The varied mechanical characteristics of the injurious agent should forecast harm severity, and the selected end-points of injury should be mirrored by the chosen injurious component of the blast. Various test methods are used to simulate human injuries caused by explosive blasts. Open-field blasts, blast tubes, and shock tubes are the most often utilised experimental models. When an explosive device is detonated in an open area, it is referred to as an open-field blast. It may be suspended from the ceiling or set directly on the ground. We present a brief summary of experimental models of brain trauma, the development of PTE, and the pathological/histological aspects of TBI, including explosion, in this paper. Our goal is to provide the reader with an overview of the most commonly used and reliable histological methodologies and neuropathological findings on blast TBI and PTE, since these reflect the cellular foundation of this

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damage and its clinical consequences, such as seizures. At this level, logical comparisons may be made between different TBI forms and, more crucially, between preclinical models and the actual state. The growing interest in the physics and pathology of blast-related brain damage has resulted in a huge number of research articles describing, often contradicting, findings, the outcomes of animal experiments These findings include both morphological changes in the CNS as well as neurophysiological and behavioral aspects of blast damage. While it is critical to investigate blast TBI and its repercussions in all aspects, pathological examination is likely the most effective technique to verify or disprove mechanistic ideas.

The pathological procedures and results discussed above explain various technical concerns that must be considered when working with tissue specimens, particularly brain. The ability to identify tissue and cellular changes and reactions to a noxious event is the most crucial. It is one of the most important guidelines to understand in order to distinguish common artefacts in CNS tissues that are not pathologically significant. Failure to do so will result in misleading and incorrect findings. Artifacts can be created by incorrect tissue handling, which is often inevitable; nonetheless, adhering to current recommendations may assist to overcome these possible technical challenges. Nonetheless, some of them deserve to be mentioned.

Some major results include the fact that an explosive blast, if severe enough, causes brain disease. Multifocal axonal and neuronal damage identified by silver staining, astroglial changes, inflammation with enhanced cytokine and reactive oxygen species activity, BBB abnormalities, and intracranial haemorrhages are the most consistent neuropathological findings. This disorder is associated with alterations in behavior such as spatial and cognitive performance and coordination. The clinical research demonstrating the advantages of body armour in minimizing blast TBI and torso protection is critical. This also lends credence to the idea that caudal propagation of shock waves through thoracic and cerebral blood vessels contributes to TBI genesis. Also significant is the finding that torso protection reduces diffuse axonal damage.

The function of the main blast in TBI is yet unknown. However, it appears that head acceleration is also a significant component to TBI. Seizures are a significant clinical complication of any TBI. Although the specific impact of this clinical condition on explosive blast TBI recovery is yet unknown, the discovery that an explosive blast causes persistent neuropathological brain alterations raises serious concerns that seizures and epilepsy are more common than previously thought. Fortunately, the VA is studying PTE in explosion TBI sufferers using a thorough prospective longitudinal strategy.