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Explosive Blast Mild Traumatic Brain Injury

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Abstract

In the recent wars in Iraq and Afghanistan, US military personnel have suffered over 333,000 traumatic brain injuries (TBIs), with over 85% being mild TBI. A variety of improvised munitions, such as improvised explosive devices (IEDs) and improvised rocket assisted mortars (IRAMs), have resulted in the explosive blast-induced TBI (bTBI). Due to its prevalence, TBI has been referred to as the signature wound of US warfighters in Afghanistan and Iraq. Explosive blast produces damage to the brain by creating a dynamic environment in forms of overpressure shock wave, heat impulse, blast-propelled projectiles, and debris, whose impact can cause complex injuries in the brain and other visceral organ systems, such as lung and bowel. Mechanisms of bTBI incorporate all forms of TBI such as falls, motor vehicle accidents, and coup-contrecoup injury. Some of the unique aspects of bTBI include the rate at which the injury occurs, the differential pressure load on and within the tissue, and the pressure-loaded tissue response. Mild bTBI is the most problematic injury within the US military in terms of number of warfighters affected and recognition of the injury. The pathobiology of mild bTBI is not fully understood. Here, we review mild bTBI injury, symptomology, and diagnosis. Finally, multi-modality testing is discussed, including functional, structural, and evidence-based evaluation with an intention to describe and diagnose mild bTBI affecting the US warfighter.

Keywords: brain injury, blast, concussion, TBI, military, IED, improvised explosive device

1. Introduction

Traumatic brain injury (TBI) in the most recent wars accounts for a significant percentage of combat-related injury. On average, about 1600 TBIs per month are suffered by US service members. The majority are due to explosive blast exposure. This is in spite of overall decreases in both combat-related injuries in general and increased wound survival rates. Active duty casualty

reports compiled by the Defense Casualty Analysis System (DCAS) indicate that since the start of the Overseas Contingency Operations (OCO) in 2001, beginning with Operation Enduring Freedom (OEF) in Afghanistan, Operation Iraqi Freedom (OIF), to the current Operation Inherent Resolve (OIR) and Operation Freedom's Sentinel (OFS), US combat casualties have a 90.7% casualty survival rate [1, 2]. In comparison, DCAS data from the Vietnam and Korean conflicts indicate 76.8 and 78.9% casualty survival rate, respectively [3, 4]. Increased survival in OCO is due to advancements in vehicle and body armor, ballistic helmets, and lenses, combined with forward-deployed surgical teams, faster evacuation times, and improved/enhanced training of medics, corpsman, and combat lifesavers based on the concepts of Tactical Combat Casualty Care (TCCC) [5–7]. Explosive blast continues to injure US warfighters leaving many with a mild blast-induced TBI (bTBI) that is difficult to diagnose and to treat.

2. Explosive blast-induced traumatic brain injury

During OCO from 2001 to mid-2016, US military service members have suffered over 333,000 traumatic brain injuries (TBIs), with over 85% being mild TBI [8]. These TBI statistics are often confusing when compared to casualty data reported by the DCAS. It must be kept in mind that DCAS casualty reporting is for active duty casualties and does not list the specific type of injury. In contrast, the TBI data collected by the Defense and Veterans Brain Injury Center (DVBIC) pools from the Armed Forces Health Surveillance Branch and does provide TBI data. In order to support US military personnel, it is essential to uncover the pathobiology of mild bTBI and develop improved treatment options. Modern combat will continue to cause bTBI, decreasing fighting strength to the point a combat team cannot accomplish mission objectives.

Explosions from various improvised munitions, such as improvised explosive devices (IEDs) and improvised rocket-assisted mortars (IRAMs), have resulted in explosive blast induced TBI (bTBI) becoming the signature wound of US warfighters in Afghanistan and Iraq [7, 8]. Explosive blast overpressure creates a dynamic environment, which can cause complex injuries in multiple organ systems [5, 9–11]. Brain injury due to explosive blast exposure is often overlooked. Realizing this, the Department of Defense (DoD) mandates that any service member exposed to a mandatory event such as a blast, vehicle collision, head injury, and so on, to be screened for concussion, followed by a neurologic evaluation.

Furthermore, categorizing and describing the different types of blast-induced head injury must continually evolve in order to better diagnose and treat the injuries.

3. Categorization of bTBI

Categorization of bTBI can initially be described as open or closed based upon the integrity of the skull and overlying tissue. Open head injury (OHI) indicates that the skull has been fractured. This can be by a foreign body, such as a bomb fragment or bullet, or from depressed skull fragments pushed into the skull interior by impact. Penetrating TBI (pTBI) is often used

synonymously with OHI, but here it is presented as a subcategory of OHI, as the brain is not necessarily involved. Bomb case fragments, shrapnel, and debris are all types of explosive ejecta, which can penetrate the brain, resulting in a pTBI. When severe, brain tissue is violated, extrusion of brain matter and cerebrospinal fluid (CSF) are often noted at the site of penetration [11]. When ejecta enters the brain, it may leave wound tracts and cavitation disproportionate to objects' size [12]. The resulting hemorrhage, edema, and macerated tissue are hallmarks of pTBI [11]. Further evaluation by CT imaging often indicates blood along the wound tract [11]. Presentation of the OHI may be as apparent as brain herniation or more subtle such as a linear skull fracture with intact skin and mild swelling. Closed head injury (CHI) is much more common. Most typically, it is due to blunt force. This causes the head to move. The brain moves slower than the head because it is surrounded by fluid, that is, CSF. This lag causes the faster moving skull to strike the brain, leading to a contusion. If the head rebounds, such as in an acceleration/deceleration injury, the brain will be struck on the opposite side as well, causing a coup-contrecoup injury pattern. During an explosive blast, the detonation generates an overpressure shock wave that moves rapidly through the air, striking the head. The pressure is transmitted through the skull to the brain. Consequently, the patient has an intact skull but underlying damage to the brain parenchyma. Both categories of bTBI can be caused by multiple injury mechanisms.

4. Explosive blast injury mechanisms

There are five injury mechanisms which contribute to bTBI individually or in combination: primary, secondary, tertiary, quaternary, and quinary injuries [13, 14]. Primary barotrauma (overpressure) injury results from the detonation/shock front impingement on and transmission through the tissue [15, 16]. Differential density of juxtaposed body structures can result in reflective waves and turbulent spalling effects within the tissue [16]. Secondary (ejecta/fragment) injury is caused by shrapnel, ejecta, and/or foreign body impact. Tertiary (acceleration) injury is due to the body being thrown by the explosive, which may result in the traditional coup-contrecoup injury and/or rotation stress causing tissue shearing or membrane disruption. Quaternary (burn) injury is caused by heat, chemicals, and/or toxidromes [17]. Finally, quinary (contaminant) injury is due to environment (infection from soil bacteria in the ejecta) or detonation (radiation) contamination [14]. Clinical identification of bTBI and severity stratification are based on post-event signs and clinical symptoms.

5. Mild bTBI severity criteria

According to VA/DoD Clinical Practice Guidelines, bTBI clinical diagnosis is primarily based on history of event (blast exposure) and any one of the following: any period of loss of consciousness (LOC), post-traumatic amnesia (PTA), alteration of consciousness/mental state (AOC), transient or persistent neurological deficits, and/or intracranial lesion [18]. Blast-induced TBI can further be categorized by severity based on degree or duration of LOC, PTA,

AOC, and imaging findings. Open head injury is considered a severe injury, while CHI severity can be classified as mild, moderate, or severe.

Mild bTBI is the most common explosive blast injury affecting US warfighters and is largely a diagnosis of exclusion. The patient may present with decreased LOC <30 min, AOC at event to <24 h, PTA <1d, best Glasgow coma scale (GCS) score within first 24 h [1, 3, 4], and no gross abnormalities on imaging such as CT [18]. For a more thorough discussion of TBI evaluation, the VA/DoD Clinical Guidelines for Management of Concussion/mTBI is suggested.

6. Mild bTBI symptoms and sequela

As with concussion, majority of mild bTBI patients recover within hours to days [18]. Often, warfighters are not aware they have suffered a bTBI or endure the symptoms to continue the mission and support fellow warfighters. It is important for all combat medical staff from the squad level to the company level and above to educate the team on the importance of prompt reporting and evaluation of those exposed to blast. Initial evaluation is the most important in order to determine severity of the injury and avoid obfuscating the classification with worsening PCS symptoms. Prompt medical evaluation of initial bTBI allows determination of timing for recovery, ensuring avoidance of a second head injury [9]. A head injury occurring within the recovery window can cause life threatening second impact syndrome (SIS) [11]. Military first providers are given the military acute concussion evaluation (MACE) as a screening tool. With the MACE is the standardized assessment of concussion (SAC). This is a paper-and-pencil clinical device that tests for alterations in attention, consciousness, memory, and orientation. If abnormal, the patient is referred to an advanced health care provider, typically the unit's physician, for diagnosis.

If concussed, patients are then placed into a Concussion Care Center for recovery. *Guidelines for Concussion Management* from the American Academy of Neurology lists recommended recovery periods [19]. A step-wise approach to rest, rehabilitation, and recovery is conducted in the Military Concussion Care Centers. Basically, patients are educated on their condition and reassured that they will likely recover quickly. Adequate sleep and rehydration are important. Behavior health issues such as post-traumatic stress disorder (PTSD) are also addressed. Once symptoms abate, the patient is allowed to resume mild physical activity and cognitive tasks. This progresses until the patient is able to conduct full physical and cognitive activities without symptom manifestation without the need for any medications. Fortunately, the vast majority of patients, over 95%, will fully recover without sequelae within a few days.

Unfortunately, some bTBI patients develop postconcussion syndrome (PCS) days after initial injury [20]. PCS is a set of symptoms (headaches, nausea, balance or coordination deficits, slurred speech, confusion, sensitivity to noise, sensitivity to light, tinnitus) that usually resolve within days to weeks after blast exposure. In general, the syndrome responds with patient reassurance and symptomatic treatment such as non-narcotic analgesics/antimigraine for headache and antidepressants for depression [11]. A subset of PCS suffers may continue to have persistent or chronic symptoms.

Persistent postconcussion syndrome (PPCS) has been identified in non-blast TBI as a condition of at least three nonresolving neurologic and behavioral PCS symptoms lasting longer than 3 months following the injury [21]. Clinical data from 181 blast-exposed veterans indicate that these criteria are inadequate to properly diagnose blast-related PPCS and that more focused testing is required [22]. The relative contribution of mild bTBI versus PTSD for PPCS is under investigation. Davenport et al. interviewed 122 veterans with a diagnosis of mild TBI, 88% having been exposed to explosive blast, and found that both mild bTBI and PTSD contribute to PPCS; however, the relative contribution of PTSD is substantially diminished after accounting for personality traits [23]. These data indicate timely description of the initial injury, correlation of ≥ 4 symptoms, and accounting for experience and personality traits aid in isolating diagnosis of PPCS.

Post-traumatic stress disorder and mild bTBI share many common symptoms such as difficulty in concentration, sleep disturbances, and mood alteration. However, there are differences. For example, bTBI patients typically complain of headaches and vertigo, whereas PTSD patients much less commonly complain of headaches and vertigo. Conversely, PTSD patients experience flashback, whereas bTBI patients typically do not. The American Psychiatric Association DMS V describes post-traumatic stress disorder as a mental health condition triggered by witnessing, experiencing, or learning a traumatic event. Symptoms of PTSD may include severe anxiety, flashbacks, nightmares, and uncontrolled thoughts about the event. Avoidance of stimuli about the event is common. Alteration in arousal may include outbursts, difficulty in concentration, hypervigilance, exaggerated startle response, and sleep disturbances [24]. All of these symptoms interfere with the person's ability to live a normal enjoyable life. Due to the extreme situations in which blast injuries occur, it is not surprising to find that mild bTBI and PTSD afflict the blast-injured patient.

7. Noninvasive evaluation of mild bTBI

Diagnosis of mild bTBI has been difficult due to limited understanding of its pathobiology. Inability to identify brain structural and functional changes in mild bTBI is a further complication. Traditional methods of imaging (CT, MRI) generally do not show gross changes for mild bTBI even though physiologic symptoms are present. Negative imaging with persistent symptoms likely indicates an injury below the limit of resolution of the image scanner. High-resolution imaging studies and electrophysiology recordings are two noninvasive tools that may aid in diagnosis of mild bTBI.

Cortical thinning on MRI scans has been noted in 11 active-duty military persons with a diagnosis of mild bTBI at an average of 1-month post-injury. Cortical thinning was identified in the superior temporal and superior frontal gyri and two areas in lateral orbitofrontal gyrus [25]. Another study of 38 veterans diagnosed with bTBI measured cortical thickness utilizing a T1 weighted 3-T MRI with 1-mm isotropic resolution and 8-channel birdcage head coil. The cortical thinning was noted in the inferior frontal, temporal, and insula regions [26]. The data is an important initial step toward a structurally relevant diagnosis and quantification of mild

bTBI. Cooperation among radiologist, clinicians, and researchers could immediately establish a database for correlating mild bTBI events, presenting symptoms, PCS, PPCS, and PTSD with MRI cortical thinning.

Diffusion tensor imaging (DTI) can be used to image white matter (WM-) integrity, which may be a predictor of mild bTBI [27]. White matter in the brain constrains water and causes it to diffuse along the myelinated axons in an anisotropic pattern of diffusion. Large linear fractional anisotropy (FA) combined with a low mean diffusivity (MD) of water is characteristic of healthy WM. On the contrary, disruption in WM-integrity allows diffusion of water away from the WM, indicated by a decrease in FA and increases MD. There are several DTI studies of veterans where loss of WM-integrity predicts mTBI [28–30]. A study of 125 veterans, 2–5 years post-deployment, indicated that mild TBI was correlated with number of deployments and increased PCS, however, indicated normal WM-integrity on DTI [23]. In addition, veterans with PTSD initially showed a larger loss of WM-integrity, but the effect was negated when accounting for behavioral sequela. As indicated by other investigators, the relative contribution of mild bTBI, PCS, PTSD, and WM-integrity, during the chronic phase of injury, still needs to be determined [23].

Evidenced-based standardized approach to evaluation of head injury has been proposed by emergency medicine physicians so as to better describe and diagnose mild bTBI. Physical exam should look for neurological abnormalities, ocular dysfunction, vestibular dysfunction, cervical injury/tenderness, ocular motor performance, signs of vestibular dysfunction, and orthostatic blood pressure. All parameters can be evaluated at the initial clinical presentation and throughout treatment, which should enhance the traditional symptomology based MACE and SAC [31].

Electrocortical potentials generated by the pyramidal neurons of the cortex can be used to monitor brain function with transdermal electrodes. Passive recording of electroencephalograms (EEGs) is often used in sleep studies and aids in the diagnosis of epilepsy. The spatial resolution is inversely proportional to width of recording electrode and distance from cortex. Temporal resolution is good and an advantage of EEG recording. Evoke response recording provides a method to evaluate cortical activity in response sensory stimulus. Visual evoked responses (VERs) are well characterized and aid in diagnosis of multiple sclerosis. Pattern reversal and flash stimuli are two of many protocols, which can be used to evaluate retinal to visual cortex integrity. Auditory evoked responses (AERs) can be used to evaluate inner ear to auditory cortex [32]. Somatosensory evoked response (SER), olfactory evoke response (OER), and gustatory evoked response (GER) all can be used to evaluate the integrity of the sensory apparatus to the sensory cortex through repeated stimuli-responses [33]. The multiple and variable symptoms of mild bTBI presentation and later transient PCS and chronic PPCS suggest diffuse low-level injury. Multisensory evaluation, combined with repeated follow-up, will aid in bridging the gap for understanding the pathobiology of this insidious condition.

8. Conclusion

Mild bTBI still lacks sufficient understanding of the mechanisms that lead to structural and functional alterations of the brain. Multidisciplinary scientific and clinical investigation can provide the coordinated efforts required to sufficiently elucidate the pathobiology of mild

bTBI. Identification of what is injured will allow development of effective treatments and protective strategies. Improvements in electroencephalography deconvolution, removable discrete electrode arrays, combined with multisensory-evoked response protocols, will make the electrophysiology techniques more clinically useful as diagnostic and, potentially, prognostic tools. Functional and structural imaging, evoked responses, and symptomology assessment are all useful in describing parts of TBI. The best approach is likely a multitest protocol to identify and diagnose the diffuse patterns of mild bTBI.

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