Review article

Ukrainian Neurosurgical Journal. 2025;31(4):20-25
doi: 10.25305/unj.333267

Post-concussion syndrome: Part 1. Overview of the epidemiology and pathophysiology of mild blast-related traumatic brain injury

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Received: 21 June 2025
Accepted: 19 July 2025

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This article provides a review of current data on the epidemiology and pathophysiology of mild blast-related traumatic brain injury (mbTBI), which has become the leading type of injury in modern military conflicts. The prevalence of mbTBI among U.S. military personnel during operations in Afghanistan and Iraq is described, with emphasis on the cumulative effect of repeated injuries that increases the risk of chronic traumatic encephalopathy. The paper discusses the main mechanisms of blast wave impact on the brain, including direct and indirect effects, pathomorphological changes, and differences from "civilian" mild traumatic brain injury (TBI). Current insights into molecular and cellular alterations underlying mbTBI are summarized, along with data from experimental and clinical studies. These findings are crucial for developing effective diagnostic approaches, treatment strategies, and rehabilitation programs for service members and veterans.

Keywords: *post-concussion syndrome; mild blast-related traumatic brain injury; pathophysiology; epidemiology; military medicine*

Epidemiology of mild blast-related traumatic brain injury

In countries involved in modern military conflicts, blast-related TBI has taken the leading position among injuries sustained by service members [1]. During the military operations in Iraq and Afghanistan—where the intensity of hostilities was lower compared to the current Russian-Ukrainian war, and regular Western forces often faced paramilitary groups—at least 10–20% of U.S. military personnel sustained TBI [2]. From the beginning of American involvement in military operations in 2000 through 2014, approximately 320,000 cases of TBI were registered [3]. According to the Defense and Veterans Brain Injury Center (DVBIC), between 2000 and the first quarter of 2016, about 348,000 active-duty service members sustained TBI. The annual number of such injuries increased from 10,958 in 2000 to a peak of 32,907 in 2011, subsequently decreasing to 22,594 in 2015. The majority of these injuries (82%) were classified as mild. Blast exposure accounted for 80% of mild TBI cases [4, 5].

In 2014, 7% of Iraq and Afghanistan war veterans receiving care within the U.S. Department of Veterans Affairs (VA) healthcare system had a TBI diagnosis [6]. A more detailed screening of one million veterans for head injuries between 2007 and 2015 revealed TBI in 8.4% of them. Given that an additional 45,000 service members had already been diagnosed with mild TBI, the total number of individuals with this type of injury reached 137,841, representing 13.8% of all those examined [7].

According to U.S. researchers, blast-related injuries—including those caused by explosive shells, landmines, and rocket-propelled grenades—accounted for 56–78% of all combat injuries sustained during operations in Iraq and Afghanistan [8]. These changes in the injury structure observed during recent military campaigns give reason to consider blast-related TBI to be the marker injuries sustained by soldiers in modern warfare [1]. Due to the prolonged nature of the conflicts in Afghanistan and Iraq and the frequent use of blast devices by enemy combatants, clinical and research attention has increasingly focused on blast-related TBI, especially its mild form [9]. It is noteworthy that only about 2.8% of blast-related head injuries were classified as severe, while the majority were mild. The true prevalence is believed to be underestimated, as mbTBI often remains undiagnosed and untreated due to the limitations of current screening tools, vague diagnostic criteria, and the lack of objective or imaging-based verification methods [10].

Reports indicating the frequent occurrence of repeated blast injuries among military personnel are alarming. A literature review [11] described a case of a U.S. Marine Corps Explosive Ordnance Disposal technician who sustained 50 significant blast exposures over 14 years of service. Other reports have documented groups of patients with an average of 13–14 blast exposures per individual during active combat deployment [11]. The high incidence of recurrent mbTBI increases the risk of developing chronic traumatic encephalopathy (a condition believed to result from multiple mild TBIs) as well as other forms of dementia [12].

The treatment and rehabilitation of service members who have sustained TBI are associated with substantial economic costs. In 2014, the average medical cost per injured service member diagnosed with TBI in the United States was USD 15,161, which was significantly higher than the cost for those without such a diagnosis (USD 5,058) [6].

Studies of blast-related TBI based on data from military campaigns in Iraq and Afghanistan concluded that, although many medical principles and treatment protocols used in the civilian sector for the assessment and management of TBI can be applied to wounded service members, there are several distinctive factors specific to this injury in military and veteran populations. These include the combat-related mechanisms of injury, unique blast dynamics, comorbid psychiatric conditions, and the influence of military culture, which often minimizes the perceived significance of mild injuries [9].

The military operations in Ukraine, ongoing since 2014 as a result of Russian aggression, are characterized by the simultaneous use of modern personal protective equipment and advanced weaponry, including barrel and rocket artillery, rocket-propelled grenades, and landmines. This has led to a growing number of blast injury cases and an urgent need for effective medical care and rehabilitation for the affected service members [13]. Accurate epidemiological data on the prevalence and long-term consequences of mbTBI will only be available after the war concludes. However, based on international research findings, it is evident that the long-term rehabilitation of a large number of affected individuals will become one of the major challenges for Ukraine's healthcare system.

Pathophysiology of mild blast-related traumatic brain injury

The mechanisms underlying blast-related TBI differ fundamentally from those of "civilian" TBI (hereinafter, the term civilian TBI is used in a nonstandard sense to distinguish injuries sustained in non-combat conditions from blast-related TBI). In peacetime injuries, brain damage typically results from rotational and inertial forces as well as from local traumatic impact. In contrast, blast-related injuries are caused by a pressure wave transmitted over a distance through the air [14]. According to the classical definition by C.J. Clemedson (1956), a blast injury represents the biophysical and pathophysiological changes resulting from direct exposure to an explosion or its associated shock wave [15]. Although blast-related TBI may present with the clinical features of mild TBI, it can be accompanied by significant morphological alterations, including cerebral edema, neuroinflammation, vasospasm, diffuse axonal injury, neuronal death, and secondary astrogliosis. At the cellular level, blast-related brain injury is characterized by multiple heterogeneous disturbances, such as increased accumulation of β-amyloid precursor protein, upregulated expression of proto-oncogenes c-Myc, c-Fos, and c-Jun, enhanced nitric oxide synthesis leading to oxidative stress, disruption of axonal transport, and elevated levels of TBI biomarkers including neuron-specific enolase (NSE), ubiquitin C-terminal hydrolase-L1 (UCH-L1), and glial fibrillary acidic protein (GFAP) [1].

Blast-related TBI represents a distinct form of traumatic injury resulting from direct or indirect exposure to an explosion, most commonly under combat conditions. Injury caused by excessive blast pressure occurs as a result of the rapid release of energy over a short time interval and within a limited volume, generating a nonlinear wave of shock and pressure. The resulting "shock wave" consists of a high-pressure front that compresses the surrounding air within submillisecond–millisecond timeframes, followed by an abrupt pressure drop, often below atmospheric levels (negative pressure)—before returning to ambient conditions. The zone of negative pressure then rapidly expands and is replaced by an equivalent volume of air. This air displacement produces the so-called "blast wind," a powerful stream of superheated air constituting a large mass of gas capable of propelling the victim's body against surrounding objects. The blast wind, together with the shock wave, forms the primary components of the "blast wave." Blast waves exert complex effects on the head–brain system, and the severity of blast-related TBI depends on both the magnitude and the duration of the pressure cycle [1, 16, 17].

From a physical standpoint, tissue injury resulting from the primary blast effect occurs through several mechanisms, including spallation, implosion, inertial effects, and cavitation. Spallation arises when a shock wave transitions from a denser to a less dense medium, causing the fragmentation of the denser material into the less dense one. A relatively simple example is an underwater explosion, in which the denser water is dispersed into the less dense air. Cavitation and implosion are interrelated phenomena that occur when the negative pressure phase of the blast wave forces dissolved gases in fluids to form bubbles. These bubbles subsequently collapse under negative pressure (implosion) and then explosively expand once the negative pressure phase passes. Spallation, cavitation, and implosion can lead to primary pulmonary blast injury. Inertial forces develop at the interfaces of tissues with different densities when the blast pressure accelerates materials of varying density at different rates, generating shear stresses [18–20]. Experimental models have demonstrated that cavitation can also result in secondary tissue damage under blast exposure. In particular, cavitation of the cerebrospinal fluid may occur at pressure levels and exposure durations consistent with those observed during real-life explosive events [19, 21].

The U.S. Centers for Disease Control and Prevention (CDC) classifies blast-related injuries into four categories [22]:

1) primary injuries, caused by the direct effect of the initial overpressure wave on body surfaces (primary blast);

2) secondary injuries, resulting from penetrating or blunt trauma caused by fragments and projectiles;

3) tertiary injuries, sustained when the body is propelled by the blast wind;

4) quaternary injuries, encompassing all other explosion-related injuries, illnesses, or diseases not caused by the primary, secondary, or tertiary mechanisms, as well as the exacerbation or complication of pre-existing medical conditions [22].

Although blast injuries are often combined or complex, this study focuses exclusively on mbTBI resulting from the shock wave.

There is a limited number of studies reporting chronic neuropathological changes associated with blast-related TBI. The results of such investigations are summarized in a literature review [23], which notes that these reports began to emerge after 2011. Notably, in the brain tissue of a former U.S. Marine, neurofibrillary tangles and tau protein pathology were identified, resembling the pattern characteristic of chronic traumatic encephalopathy (CTE). Similar pathological alterations were observed at autopsy in four military veterans exposed to blasts. In another series of pathohistological studies (five blast-exposed servicemen, four of whom survived longer than two months after the incident), axonal pathology was described. In five additional cases of chronic blast-related TBI (with survival beyond six months post-injury), the authors reported pronounced astroglial pathology (astrogliosis), indicating the presence of reactive gliosis, as well as tau protein abnormalities in two of the five cases. In addition to these limited human studies investigating both acute and chronic neuropathological outcomes, a considerable number of animal models have been developed. However, these models have inherent limitations in accurately replicating the pathophysiological mechanisms and neuropathological features of blast-related TBI observed in humans.

The blast wave can affect the brain through several mechanisms. First, as kinetic energy passes through the skull, it can directly induce acceleration or rotational motion of the brain, leading to diffuse axonal injury (DAI) followed by secondary axonal degeneration. Although these injury types may initially appear similar to non-blast, "civilian" TBI, recent evidence indicates that the axonal injury pattern caused by blast exposure is unique to blast-related TBI. Neuroimaging studies of military personnel using diffusion tensor magnetic resonance imaging (DT-MRI) after blast exposure have demonstrated that axonal injury in blast-related TBI is more widespread and spatially variable compared to non-blast "civilian" trauma. Affected brain regions include the superior corona radiata of the frontal cortex, the cerebellum, and the optic tracts [24, 25]. These findings are consistent with results obtained from rat models of blast-related TBI, which showed that rotational brain injury (analogous to "civilian" trauma) produces distinct behavioral disturbances compared with blast-induced neurotrauma [26]. Collectively, these data support the notion that blast-induced axonal injury should be regarded as a separate subtype of diffuse axonal injury. This is further supported by the more pronounced inflammatory response observed in blast-related TBI relative to non-blast trauma, including increased expression of pro-inflammatory cytokines and heightened neuroglial activation [27, 28]. Additionally, in rat models of blast-related TBI, brain edema and vasospasm are more prevalent due to primary arterial constriction and compromised vascular integrity, leading to secondary neuronal tissue damage [29].

According to K.J. Dixon (2017), in addition to the direct mechanical impact on brain tissue, the blast wave may also act indirectly through two possible mechanisms. First, a blast can cause compression and subsequent expansion of gas-containing compartments within the brain, resulting in damage to surrounding tissues. Second, the blast wave may generate pressure waves within the blood or cerebrospinal fluid, which propagate to the brain within seconds. Such a shock wave can accelerate tissue components from rest to a velocity dependent on medium density, potentially causing deformation and injury of the affected neural structures [30]. These indirect effects of blast exposure are thought to result both from the transmission of explosive energy through the blood vessels of the thoracic and abdominal cavities and from vagus-mediated bradycardia, arterial hypotension, and possibly cerebral hypoperfusion [31].

The described effects of blast waves on the central nervous system have been confirmed in experimental studies. Following a single sublethal blast exposure generating overpressure levels of 48.9–77.3 kPa in open-field conditions (a model of mbTBI), rat brains examined one day post-injury exhibited "darkened", shrunken cortical neurons and spastic blood vessels. Histological analyses revealed apoptotic oligodendrocytes and astrocytes (TUNEL-positive staining) within the white matter, along with acute axonal injury (increased amyloid precursor protein immunoreactivity) but without evidence of macrophage or microglial activation. Signs of recovery were observed on days 4 and 7 post-blast, indicating the mild nature of the cellular injury and white matter alterations [32].

Several anatomophysiological prerequisites may underlie the mechanisms described above. These include the direct propagation of the blast wave through the skull or paranasal sinuses [33]. Conversely, the indirect action of the blast wave is associated with compression of the abdomen and thoracic cavity, which transmits kinetic energy through the body's biological fluids. This effect generates oscillatory pressure waves that travel from the bloodstream to the brain—remote from the site of maximal explosive energy impact. Such kinetic energy transfer induces functional and morphological alterations within brain structures, constituting a specific feature of blast-induced brain injury, which is not observed in other known forms of TBI. The complex mechanism of blast-related damage also involves the primary blast impact on the autonomic nervous system [1]. In an experimental mouse model, the effects of mbTBI were assessed at overpressures of 68, 103, and 183 kPa generated by a shock tube on parenchymal organs and the brain. The principal injuries to extracerebral organs included pulmonary hemorrhages and hemorrhagic infarctions of the liver, spleen, and kidneys. Multifocal axonal injury was observed in the cerebellum, corticospinal tracts, and

optic pathways, accompanied by persistent behavioral and motor impairments, such as deficits in social interaction, spatial memory, and motor coordination. Notably, torso protection significantly reduced both axonal damage and behavioral deficits [34]. In another study, mbTBI was modeled in rats using a shock tube producing overpressures of 126 and 147 kPa. The thoracic cage and abdominal region of the animals were protected with a Kevlar vest, which reduced blast-related mortality, as well as the extent of axonal degeneration in the brain and the severity of neurological and behavioral disturbances [35].

Taken together, these findings support the increasingly recognized theory that thoracoabdominal vascular/hydrodynamic transmission of blast waves may represent a primary mechanism of blast-related TBI. Notably, certain patients exposed to blast events exhibit clinical signs of TBI in the absence of direct head trauma [20]. This theory has received experimental confirmation. The authors of the study [36] investigated the effects of a blast wave on rats placed within a shock tube apparatus, where overpressure of 70 and 130 kPa was applied exclusively to the torso, while carotid artery and intracranial pressure were simultaneously measured. The researchers demonstrated that the blast energy transmitted through the torso resulted in a 255% increase in peak bulk blood flow velocity in the basal brain regions and a 289% increase in shear stress within cerebral vessels. They concluded that the indirect mechanism of blast injury provokes a sudden, high-magnitude blood surge that rapidly propagates from the torso through the neck to the cerebral vasculature. This surge markedly increases vascular wall shear stress within the brain's circulatory network, potentially leading to functional and structural alterations of cerebral veins and arteries and, ultimately, to vascular pathology [36]. An increase in cerebral vascular pressure resulting from thoracoabdominal compression compromises the integrity of the blood–brain barrier (BBB), leading to damage of small cerebral vessels. As noted above, blast-related injury to air-filled organs, such as the lungs, may cause air embolism through the spallation mechanism. Air emboli can enter cerebral vessels, resulting in cerebral ischemia and infarction. In addition, blast overpressure may provoke structural alterations of arteries due to blast-relayed vasospasm [20,37–39].

According to researchers [40], experimental models should compare the severity of blast-related TBI using two types of special protective devices. The first device ("iron lungs") allows the blast wave to act exclusively on the head of the animal, while the second directs the blast exposure to the thoracic and abdominal regions. Such experiments would help explain and substantiate the clinical observation that enhanced thoracic protection provided by ballistic body armor likely plays a significant role in mitigating the severity of blast-related TBI by preventing pulmonary injury at blast intensities that would otherwise cause TBI [41]. However, it has been reported [11] that under real combat conditions in the Iraq and Afghanistan wars, body armor and helmets did not protect soldiers from closed blast-related TBI.

Taken together, the literature evidence convincingly demonstrates that the biomechanics and pathophysiology of neurotrauma resulting from blast exposure differ fundamentally from those of civilian peacetime injuries or penetrating ballistic trauma sustained in combat. These findings are of critical importance for developing effective diagnostic, therapeutic, and rehabilitation strategies for military personnel and veterans affected by blast-related brain injury.

Disclosure

Conflict of Interest

The authors declare no conflict of interest.

Funding

This research received no external funding.

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