Explosive Blast Neurotrauma

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Abstract

Explosive blast traumatic brain injury (TBI) is one of the more serious wounds suffered by United States service members injured in the current conflicts in Iraq and Afghanistan. Some military medical treatments for blast TBI that have been introduced successfully in the war theater include decompressive craniectomy, cerebral angiography, transcranial Doppler, hypertonic resuscitation fluids, among others. Stateside neurosurgery, neurocritical care, and rehabilitation for these patients have similarly progressed. With experience, military physicians have been able to clinically describe blast TBI across the entire severity spectrum. One important clinical finding is that a significant number of severe blast TBI victims develop pseudoaneurysms and vasospasm, which can lead to delayed decompensation. Another is that mild blast TBI shares clinical features with post-traumatic stress disorder (PTSD). Observations suggest that the mechanism by which explosive blast injures the central nervous system may be more complex than initially assumed. Rigorous study at the basic science and clinical levels, including detailed biomechanical analysis, is needed to improve understanding of this disease. A comprehensive epidemiological study is also warranted to determine the prevalence of this disease and the factors that contribute most to the risk of developing it. Sadly, this military-specific disease has significant potential to become a civilian one as well.

Key words: clinical management of CNS injury; decompressive craniectomy; military injury; penetrating ballistic-like brain injury; traumatic brain injury

Introduction

Because of the ongoing Global War on Terror, there are increasing numbers of patients afflicted with traumatic brain injury (TBI). Many of these patients are injured by an explosive blast (bTBI). From this mounting clinical experience, bTBI is becoming recognized as a disease distinct from penetrating TBI (pTBI) and closed head TBI (cTBI). In this article the prevailing understanding of the basic mechanisms of injury and common approaches to clinical management will be presented.

In Operation Enduring Freedom in Afghanistan (OEF) and Operation Iraqi Freedom (OIF), explosive blast accounts for over 60% of combat casualties. For these wars, the most common explosive weapon is an improvised explosive device or IED (Shanker, 2007). Historically, the head is frequently injured in battle, accounting for about 20% of all combat-related injuries in past modern wars (Bellamy et al., 1986; Bellamy, 1992; Carey, 1996; Carey et al., 1998). The epidemiological data are still evolving for OIF and OEF. However, evidence to date from the Joint Theater Trauma Registry shows that injury patterns from OIF and OEF are similar to those noted previously (Shanker, 2007).

A distinct feature of OIF and OEF compared to wars of the 20th century is the high survival of combat-injured warfighters, even those who suffer TBI. An important contributor to this survival is the use of body armor. In the past, the severity of bTBI was thought to be related to pTBI from fragments or cTBI from the head striking an object after the victim was thrown (Bellamy, 1995; Carey, 1996). The modern combat helmet is preventing many fragments from causing pTBI. It also affords some protection against impact, but may not be optimized for this particular purpose. However, it is now known that bTBI can manifest at all severity levels, independent of a penetrating wound or being bodily thrown. Thus, unfortunately even this effective protective equipment cannot fully prevent all aspects of bTBI (Jaffe, 2004; Shanker, 2007).

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Advances in military medical care have also contributed to the lowest killed:wounded ratio (less than 1 in 10 patients die) in modern history. There have been a number of battlefield and in-theater medical innovations. Important clinical improvements used for treating TBI are early decompressive craniectomy, neuro-critical care, cerebral angiography, transcranial Doppler, hypertonic saline, TBI clinical management guidelines (battlefield and in-hospital), and others (Knuth et al., 2005; Ling and Ecklund, 2007; Ling et al., 2008). As a result, more warfighters are surviving what previously would have been fatal injuries. This growing number of veterans with serious disabilities necessitates new opportunities for advancing medical care (Zoroya, 2005).

Prevalence of Blast Traumatic Brain Injury

The lay press and other popular media outlets report the prevalence of bTBI as approaching 40–60% of deployed U.S. warfighters (Zoroya, 2005; Shanker, 2007; Zoroya, 2007a, 2007b). A recent RAND report estimates that 320,000 service members or 20% of the deployed force potentially suffer from TBI (Tanielian and Jaycox, 2008). The supporting evidence for these claims is limited. A comprehensive scientifically rigorous epidemiological study of bTBI has not yet been done. Instead, the data used to support these assertions are from extrapolations from limited sample sizes, self-reported data, single centers and/or narrow inclusion criteria (Okie, 2005; Zoroya, 2006; Hoge et al., 2008; Tanielian and Jaycox, 2008). Importantly, the relationship between bTBI and post-traumatic stress disorder (PTSD) is incompletely understood (Hoge et al., 2008). As these two separate and distinct diseases share common clinical symptoms, which may affect accurate diagnosis, caution must be taken when trying to generalize such data. There are active efforts to elucidate the true epidemiology of this injury. Some of these efforts include the Joint Theater Trauma Registry, the Defense and Veterans Brain Injury Center study, and a new comprehensive database effort supported by the Hugh and Carolyn Shelton Military Neurotrauma Foundation.

 Explosive Blast Traumatic Brain Injury, a New Type of TBI

TBI has classically been divided into two groups, pTBI and cTBI. Blast TBI shares clinical features of both pTBI and cTBI, but also has unique aspects.

In pTBI, a foreign object penetrates the bony skull and then traverses through the brain parenchyma. This leads to physical disruption of neurons, glia, and fiber tracts, all of which are exacerbated by ischemia and hemorrhage. Clinically, patients usually have an impaired level of consciousness with neurological deficits referable to parenchyma injured by the foreign body tract of travel. If the object enters at very high velocity, the severity of clinical impairment may greatly exceed what one would expect from the size of the object (e.g., a rifle bullet). This is due to cavitation of brain tissue. The characteristic surface lesion is a breach in the skull where the foreign body entered. There may also be another skull defect where the foreign body exited. CSF leaks and an extruding brain are often observed emerging from the defect. CT scans typically reveal blood along the path of travel. Hemorrhage, cerebral edema, and macerated tissue are all hallmarks of pTBI.

In cTBI, disruption of brain function can occur from brain motion and deformation within the cranium, resulting in the typically observed injuries to the brain parenchyma, blood vessels, and fiber tracts (Bandak et al., 1996). A patient’s clinical presentation can be related to the severity of the mechanical insult the brain experiences. Focal impacts may result in localized lesions and neurological deficits. The clinical syndrome worsens sub-acute ly due to secondary injury processes of inflammation, ischemia, and free radical formation.

In bTBI, primary injury to the brain occurs when the physical forces emanating from detonation couple to the head and brain. The exact forces that contribute to the injury are not fully understood, but pressure transient appears to play an important but possibly not exclusive role. Furthermore, in bTBI the calvarium may or may not be breached, and thus patients may have findings ascribable to cTBI, pTBI, or both. There may be secondary, tertiary, and quaternary blast effects that may also contribute to a patient’s particular presentation. As described by the Centers for Disease Control and Prevention (CDC), primary blast injury is from physical forces emanating from the explosive (Centers for Disease Control and Prevention, 2006). Secondary injury occurs from matter thrown by the explosion, including fragments from the weapon casing and environmental debris. Tertiary injury results when the patient is thrown by the explosive blast and strikes an object such as a wall or the ground. Quaternary injury is from factors not included in the first three, such as burns or toxic fume inhalation.

Clinically, a patient’s condition may be as mild as a brief period of confusion to as severe as a coma. One apparent characteristic of severe bTBI is how commonly patients have diffuse cerebral edema and hyperemia. This develops rapidly, often within just an hour or so following blast injury. The presence of subarachnoid hemorrhage indicates a more severe injury and often heralds acute severe brain edema and hyperemia, and delayed vasospasm. This vasospasm is often the cause of delayed neurological deterioration (Armonda et al., 2006). This presentation seems to be more common following blast than the other types of TBI, and has led military neurosurgeons to perform decompressive craniectomies more often than commonly done for pTBI or cTBI (Grant, 2007; Schlüfka, 2007; Ling et al., 2008).

There is a challenge in confirming or refuting a unique signature clinical description for bTBI. It is difficult using epidemiologic data alone to separate the less understood explosive blast injury signs and symptoms from other better understood components attributable to classic cTBI or pTBI. Further complicating this effort is that these patients frequently have other serious injuries beyond TBI (such as traumatic limb amputation and hemorrhagic shock) that require aggressive resuscitation and other systemic therapies. The effects of such therapies on the clinical picture of bTBI need clarification.

Mechanisms of Explosive Blast Traumatic Brain Injury

The precise causes and mechanisms of primary bTBI remain unknown and the current understanding is incomplete. Specifically, the “cause” is the action generated by the explosive blast, and the “mechanism” is the process by which the interaction of the explosive blast with the body produces injury (i.e., the wounding process or how the body is injured).
In trauma, there are primary and secondary injury mechanisms. Primary injury mechanisms are those which can be attributed directly to the cause (i.e., periorbital vascular disruption from a blow to the eye). Secondary injury mechanisms constitute the physiological response to the primary injury (i.e., the inflammatory swelling associated with the “black eye”). For bTBI, it is probable that more than one primary injury mechanism exists and that there are potentially several coupled mechanisms.

Blast produced by an explosive device is a transient pressure wave disturbance that travels through a medium like air or water. This is accomplished through detonation of an energetic material such as a conventional explosive. An explosive is a material capable of storing high molecular binding energy in a relatively small volume, but has the important characteristic of relative instability. Thus this quiescent stored chemical energy can be released very rapidly when a chemical reaction is induced. This energy release leads to a phase change that generates rapidly expanding gas. This in turn transfers mechanical, thermal, and electromagnetic energy into the surrounding medium as well as into objects or people within the blast proximity. The extent and intensity of this process depends on several factors such as charge size, chemical composition, and confinement. This rapid, chemically driven process is referred to as a “detonation.”

A critical aspect of a detonation is the rapid pressure waves that can travel faster than the characteristic wave speed of the host medium, and therefore compress to become shock waves. Figure 1 schematically shows a steepening pressure wave front experiencing a shock wave front in the formation of a shock wave. The shock wave represents a very rapid and steep rise in the pressure and can be a direct mechanism of injury in the brain exposed to blast.

FIG. 1. Schematic of the steepening process that a pressure wave front experiences in the formation of a shock wave. The shock wave represents a very rapid and steep rise in the pressure and can be a direct mechanism of injury in the brain exposed to blast.

The non-free field or enclosed condition (i.e., inside a structure) is more complex. In an enclosure, blast pressure waves reflect off walls, ceilings, barriers, and other objects, creating a “complex wave field.” Under such conditions, each explosive blast must be analyzed on an individual basis and cannot be generalized by a single relation as was done by Friedlander for the free field example. For a complex blast wave, the contributions from the shape and the peak of the initial blast wave as well as each reflected wave need to be accounted for spatially and temporally in the assessment of injury.

The assumption that the cause of bTBI is dependent only on peak pressure and not the shock wave may not be valid. This original assumption is based on the work of Bowen and Richmond, who described a relationship between peak pressure and lethality in a sheep model (Bowen et al., 1968; Richmond et al., 1968). The impression is that high pressures lead to air-filled organ failure (i.e., lung and bowel) (Phillips and Richmond, 1991). If the lung is the most susceptible organ then a high prevalence of blast lung injury is to be expected. However, the OIF and OEF clinical experience reveals that blast lung injury occurs infrequently. Acute respiratory distress syndrome (ARDS) is reported, but only in conjunction with other severe injuries such as traumatic amputation and massive hemorrhagic shock. ARDS is rarely, if ever, seen in isolation (Holcomb, 2005). The bowel has not been found to be injured by explosive blast unless there is shrapnel penetration. In contrast, bTBI is much more common. The brain is not an air-filled organ and so other mechanisms such as those from shock wave effects must be responsible. Brain injury was not studied in the earlier studies (Bowen et al., 1968; Richmond et al., 1968). One consideration is that the interceptor body armor vest may be protecting the lungs and bowel from blast forces. Another possibility is that other physical forces may contribute or play a more prominent role in explosive blast injury.
An explosive detonation produces effects other than just pressure waves. Conventional military ordnance also releases light, acoustic, thermal, and electromagnetic energies, some of which can be injurious to the CNS. In addition, there are toxic fumes. The relative contribution of each of these forces to bTBI is uncertain. The evidence is lacking for including or excluding them as potential causes of bTBI.

Categories of Severity for bTBI

Blast TBI is presently categorized similarly as TBI from cTBI and pTBI. Military providers use the same criteria as their civilian counterparts. The TBI definition from the Mild Traumatic Brain Injury Committee of the American Congress of Rehabilitation Medicine (1993) applies to bTBI when explosive blast is the etiology of loss of consciousness, loss of memory preceding or following injury (amnesia), alteration in mental status at the time of injury, and/or focal neurological deficit. The level of bTBI severity is based mainly on the duration of altered mental status. Mild bTBI is associated with brief (<5 min) loss of consciousness or awareness. As in classic cTBI, mild bTBI can lead to headache, confusion, and amnesia, as well as other symptoms such as difficulty concentrating, mood alteration, sleep disturbance, and anxiety. After injury, these symptoms often resolve within a few hours or days. Very troubling to patients is post-concussive syndrome, which may develop up to days later (Ling and Maher, 2006).

A joint Veterans Administration and Department of Defense committee is developing guidelines for management of this condition. In general, this syndrome usually responds to reassurance and specific symptomatic treatment such as non-narcotic analgesics, anti-migraine medication for headache, and antidepressants. As with civilian cTBI, it may last only a few weeks, but in some cases can persist for up to a year or more (Jarrell and Ecklund, 2003; Ryan and Warden, 2003; Ling and Maher, 2006).

Again using civilian criteria, moderate bTBI is associated with a presenting Glasgow Coma Scale (GCS) score of 9–13, often with prolonged loss of consciousness and/or neurological deficit (Geocadin, 2004). Combat-injured patients suffering from moderate bTBI will require prompt evacuation to an echelon 3 medical treatment facility (i.e., combat support hospital), and may require neurosurgical care. They too may develop post-concussive syndrome (Jarrell and Ecklund, 2003; Ling and Maher, 2006; Ling et al., 2008).

Severe bTBI occurs when the injury causes the patient to be obtunded or comatose (i.e., presenting with a GCS score of 8 or less). Such injury is typically associated with significant neurological injury, often with abnormal neuroimaging (e.g., head CT scan revealing skull fracture, intracranial hemorrhage, and early diffuse cerebral edema). These patients require advanced medical care while still on the battlefield. The “Guidelines for Field Management of Combat-Related Head Trauma” is an effort to optimize far-forward medical care of...
TBI (Knuth et al., 2005). After initial stabilization, these patients must be evacuated to the nearest combat support hospital that has neurosurgical capability. Such patients will most likely need airway protection, mechanical ventilation, neurosurgical intervention, intracranial pressure monitoring, and neuro-critical care in an intensive care unit setting. Recovery is prolonged and usually incomplete if at all. Based on the civilian TBI experience, it is expected that a significant percentage of severe bTBI patients will not survive to 1 year post-injury (Geocadin, 2004; Ling et al., 2008; Multi-Society Task Force on PVS, 1994a, 1994b).

Explosive blast TBI can also lead to concussion. bTBI concussion is considered by some investigators as a subtype of cTBI (Warden, 2006). It too can be classified as mild (grade 1), moderate (grade 2), or severe (grade 3), using the same criteria established for civilian concussion. Mild concussion is defined as brief confusion lasting less than 15 min, but with no loss of consciousness. At the moderate level, confusion lasts longer than 15 min, but consciousness is maintained. Severe concussion is whenever there is any loss of consciousness.

Recognizing that applying criteria developed for classic civilian TBI may be inappropriate, a new classification specific for bTBI is being proposed (Okie, 2005; Warden and French, 2005). Mild bTBI is defined as loss of consciousness <1 h and post-traumatic amnesia <24 h after exposure to an explosive blast. Moderate bTBI is loss of consciousness for >1 but less than 24 h and amnesia lasting >1 but <7 days. Severe bTBI is loss of consciousness >24 h and amnesia >7 days. It needs to be emphasized that this classification is a proposal and has not yet gained wide acceptance in the medical community.

**Clinical Management**

Clinical care for TBI begins on the battlefield and is carried out in accordance with the “Guidelines for Field Management of Combat-Related Head Trauma” (Knuth et al., 2005). The combat medic’s first responsibility is to protect the patient from further harm. After this, the ABCs of airway, breathing, and circulation are attended to. During this initial stabilization, the patient’s GCS score should be determined. The GCS score can be helpful in making triage decisions, especially in isolated head injury. If the GCS score is low, the patient should be evacuated to higher echelons of care. If GCS score is
13 or less, this should be done by helicopter or in another expeditious manner. Typically, IED blast injuries result in multiple injuries to the same patient, all requiring simultaneous management.

Once at the combat support hospital, a more detailed clinical assessment should be made. Neuroimaging with CT should be done as soon as possible to identify lesions such as intracranial hemorrhage, skull fracture, or cerebral edema. It is crucial that such conditions are diagnosed so that early neurosurgical intervention can be rendered. Military TBI patients receive the same standard of care in a combat support hospital as they would in a civilian hospital (The Brain Trauma Foundation, 2007). Important issues to attend to include maintaining adequate oxygenation, controlling intracranial pressure (ICP), and ensuring proper cerebral perfusion pressure. Intracranial hypertension is common in severe bTBI. One frequent observation among operating neurosurgeons is the presence of hyperemia and severe edema in the acute period (Fig. 4A). This tends to occur more frequently when a traumatic subarachnoid hemorrhage is noted on CT (Fig. 4B). The presence of blood in the basilar cisterns may be one marker for higher severity of blast injury. Patients have also been noted to develop delayed increased ICP, sometimes 14–21 days after severe bTBI. This phenomenon may be related to other insults such as vasospasm (Armonda et al., 2006). One medical therapy that has been used with great success in these types of injuries is hypertonic saline (Ling and Marshall, 2008; Ling et al., 2008). Intravenous boluses of 23% NaCl can be used for acute elevations of ICP with continuous IV infusions of 2% and 3% NaCl solutions to maintain ICP control (Qureshi and Suarez, 2000; Geocadin, 2004). The benefit of hypertonic saline solutions is that serum osmolality may be increased without compromising intravascular volume. This is critical, as severely injured warfighters are often suffering from hemorrhagic shock. Other therapies that have been promising in mitigating delayed intracranial hypertension are mild hypothermia (34–36°C) and early decompressive craniectomy (Ling et al., 2008). Presently, factor VIIa is not used to treat intracranial hemorrhage. However, some TBI patients may receive factor VIIa for the clinical indication of systemic hemorrhage (Holcomb, 2005).

Decompressive craniectomy is commonly used in severe bTBI patients (Okie, 2005; Grant, 2007; Schlifka, 2007; Ling et al., 2008). A decompressive craniectomy permits the swelling brain to avoid compression by the skull and may also provide local brain cooling. From a practical military standpoint, craniectomy provides an additional measure of safety for ICP control during the evacuation process where, at times, ICP management can be challenging. The other benefit of early decompressive craniectomy is that it may obviate the need to use more conventional methods of ICP control such as pharmacological coma. Barbiturate coma is difficult to execute in the deployed setting due to the limited number of neuro-critical-care specialists and lack of EEG support. Thus for many reasons, decompressive craniectomy is the most practical, if seemingly aggressive, approach. Cranioplasty is delayed for at least 3 months from the time of the patient’s last infection. The availability of computer-generated cranial flaps made from methyl methacrylate has improved cosmetic results, shortened operative times, and reduced the need for subcutaneous placement of the bone flap with its attendant risks (Fig. 5A and 5B).

IED blast injuries are complex. There are frequently associated injuries, including traumatically amputated limbs, multiple penetrating wounds, and severe hemorrhage. Management is multidisciplinary and rapid with several subspecialists treating the patient simultaneously under the direction and coordination of a trauma surgeon. There is often extensive soft-tissue loss from the blast with possible severe facial and scalp burns (Fig. 6). The helmet provides excellent protection so penetrating objects usually enter through the face, orbit, or skull base (i.e., areas not covered by the helmet). Prevention of CSF leakage and good tissue coverage are of paramount importance. When the helmet stops a fragment,
there can also be an associated cTBI ranging from mild concussion to severe contusions and skull fractures from the resulting helmet deformation (Fig. 7A and 7B).

The kinetic energy imparted by a penetrating projectile and the location of the injury tract are important. Low-velocity projectiles may penetrate the skull but cause little underlying parenchymal damage (Fig. 8), whereas high-velocity projectiles can cause massive tissue damage from a large secondary cavity. The cavity creates large volumes that can be of such pressure that skull fracture occurs with fragments expanding in an outward displacement fashion. Fragments from explosions travel at low velocity and thus do not result in significant cavitation. The decisions with regard to removal of shrapnel debris or bone fragments lodged in the brain have evolved since the Vietnam and Korea Wars. Previously, surgeons aggressively sought to remove all foreign bodies including bone fragments in an effort to reduce infection risk and risk of developing post-traumatic epilepsy. Longitudinal studies of treated Vietnam War patients and other more recent studies from other conflicts have shown that aggressive removal of all fragments is unnecessary, but removal of gross contamination with débridement of large injury tracts is beneficial (Carey et al., 1970; Carey et al., 1972; Carey et al., 1974; Carey et al., 1974; Carey, 1987; Carey et al., 1998). Currently, any accessible fragments are removed if they can be safely debrided along the tract, but fragments that may be deep or subcortical, contralateral, or otherwise inaccessible are left in place (Grant, 2007; Schlifka, 2007; Ling et al., 2008). The reconstruction of the dura and scalp closure are important to reduce the incidence of CSF leakage and meningitis. A soldier who presents with a good neurological status, no significant mass effect, and small penetrating fragments in the brain will likely get local débridement and scalp closure with an emphasis on preventing CSF leakage. On the contrary, a soldier who presents with significant tissue destruction, mass effect, and/or an extensive wound tract with large penetrating fragments will likely undergo a large decompressive craniectomy, débridement of the tract, and removal of the fragments (Carey et al., 1972; Carey et al., 1998; Grant, 2007; Schlifka, 2007).

If bodily thrown by the explosion, the patient’s head can strike a solid object resulting in tertiary blast injury. As a result, there can be skull base injuries to the middle fossa, temporal bone, or frontal sinus, which should be suspected if patients have otorrhea or rhinorrhea. There is also a
have complex Le Fort fractures, as well as a ventriculostomy, lumbar drain, or both in place. Early maxillofacial fixation is performed, even in the setting of a head injury. The compounded morbidity of drug-resistant *Acinetobacter baumannii* meningitis with external drains in place is much greater than that of early surgical intervention. Anecdotal reports suggest that early reduction of facial fractures has been effective in minimizing CSF leaks.

Finally, there can be critical vascular effects on both arteries and veins, such as sagittal sinus injury and lacerated cortical arteries. Blast injuries appear to have a high risk for traumatic pseudoaneurysm formation. Our recent work revealed a 35% incidence of pseudoaneurysms in 47 blast-injured patients who underwent cerebral angiography at the National Naval Medical Center during the first 2 years of OIF (Armonda et al., 2006). Patients who underwent angiography were those who sustained an injury tract near a vascular territory or had an unexplained decompensation in neurological status or ICU monitoring indicators. Blast-related pseudoaneurysms have been observed to expand and rupture, and require treatment by appropriate endovascular or open surgical techniques.

Vasospasm is a particularly common finding after bTBI. Our study revealed that 47% of the patients who underwent angiography developed cerebral vasospasm (Armonda et al., 2006). Early transcranial Doppler studies in-theater demonstrate that this vasospasm can develop early, often within 48 h of injury. Not surprisingly, this occurs with moderate and severe bTBI, and is worse with higher injury severity (Ling and Ecklund, 2007). Vasospasm can also present later in the course of this disease, typically 10 or more days after initial injury, and does appear to be more prevalent when traumatic subarachnoid hemorrhage is also present acutely.

Many moderate and severe bTBI victims arrive intubated and medicated from the field and have severe facial trauma that prevents a reliable neurological examination, especially of pupillary reaction. CT can reveal tight basilar cisterns and poor sulcal definition indicative of diffuse edema, without any apparent penetrating brain fragments or intracranial...
Blast Concussive Injury

Diagnosis of concussion or mild TBI is particularly vexing. It is critical to recognize early that a warfighter has suffered a concussion. This is so that patients who are diagnosed can receive appropriate medical care, and more importantly, so that they do not return to duty with compromised mental status, which at times can be quite subtle. In the war theater, the first providers are generally medics, who typically do not have sufficient training in recognizing the subtleties of mild TBI. This is exacerbated by the lack of overt evidence of trauma (e.g., lacerations or hematoma). The patient may not recognize that she or he has suffered an injury. Some patients will knowingly try to hide their injury so that they can remain with their unit (Zoroya, 2007a, 2007b). Thus, for medics and other medical providers, there needs to be increased suspicion that bTBI may have occurred in any warfighter who has been exposed to an explosive blast so that an appropriate clinical work-up can be initiated. If necessary, the patient should be referred to higher echelons of care, ideally to a neurologist, neurosurgeon, or emergency medicine physician (Ling and Maher, 2006; Warden, 2006).

After their first blast exposure, many soldiers do not recognize that they may have been injured and thus will not seek medical care. It may take two or more blast exposures before they realize that they may have been injured. The first indication of injury may be persistent post-concussive symptoms, such as headaches, vertigo, short-term memory loss, and difficulty concentrating or multi-tasking (Ryan and Warden, 2003; Okie, 2005; Ropper and Gorson, 2007). As these symptoms can be subtle, these patients should undergo a detailed mental status evaluation by a physician or psychologist. Part of this evaluation should be objective neuropsychological testing, even though it may consist only of limited bedside testing. There are efforts underway to develop neuropsychological tests that can be automated on a laptop computer or are easy enough for use by less-trained providers (Zoroya, 2007a, 2007b).

If a patient is suspected of having PTSD, a combat stress team provider or psychiatrist should be consulted. It cannot be overemphasized that bTBI and PTSD can occur alone or together and may be difficult to differentiate.

If TBI is suspected, the patient must be removed from combat-related duties. This may mean garrison or light duty for a period of time until symptoms fully resolve, or evacuation out of theater where advanced neuroimaging (such as MRI) and more detailed evaluation may be instituted.

One important clinical condition to be avoided is second-impact syndrome (SIS). SIS is a disease that can develop if a subsequent head injury occurs before full recovery (McQuillen et al., 1988; Ling, 2007). This can lead to a worse clinical outcome, especially in the young, in whom mortality rates can reach 50%. In order to allow for sufficient recovery, military medical providers use established civilian practice guidelines, such as The American Academy of Neurology’s “Guidelines for Concussion Management,” that includes recommended periods of recovery (Quality Standards Subcommittee of the American Academy of Neurology, 1997). There are other guidelines that can also be used, such as the Cantu grading system and the Colorado Medical Society Guidelines (Bailes and Cantu, 2001; Bleiberg et al., 2004). Although developed for sports-related TBI, these are still useful tools for military health care providers (Ling, 2007; Ropper and Gorson, 2007).

Conclusion

Blast TBI is a disease afflicting many combat-injured warfighters, and it potentially constitutes a new category of TBI.
Clinically it shares many features with traditional TBI, but there are unique aspects. In its mild form, bTBI also shares many features with PTSD, but here again it has distinct aspects. Although military medical providers depend on civilian standard-of-care TBI guidelines when managing bTBI, they are continually adapting their medical practice in order to optimize the treatment of this disease, particularly in a theater of war. It is clear that further rigorous scientific studies of bTBI at both the basic science and clinical levels are needed. This research must include improved understanding of the causes and mechanisms by which explosive blast leads to TBI. Also needed are comprehensive epidemiological studies to determine the prevalence of this disease, and the factors that contribute most to the risk of developing it. A widely accepted unambiguous clinical description of bTBI with diagnostic criteria would greatly improve diagnosis. It is hoped that through appropriate research, meaningful prevention, mitigation, and treatment strategies for bTBI can be realized quickly. It should be recognized that although bTBI is currently a disease suffered largely by soldiers, there is the unfortunately high likelihood that it may become a civilian disease as well.

Author Disclosure Statement

The views and opinions expressed herein are solely those of the authors. They do not represent, and should not be interpreted as, stated or implied endorsement by the Uniformed Services University of the Health Sciences, U.S. Army, U.S. Air Force, Department of Defense, or the U.S. government.

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