Traumatic Brain Injury Among Veterans Returning From Afghanistan and Iraq

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By Bruce Capehart, MD, MBA [1] and Dale Bass, PhD [2]

This article addresses the epidemiology, diagnosis, and treatment of mild TBI among combat veterans, with a particular focus on blast injury and the presence of comorbid posttraumatic stress disorder (PTSD).

Causes of TBI

Although current media and scientific attention is focused on TBI from wartime incidents, the causes of TBI among US military service members and veterans include combat, training accidents, and nonmilitary accidents. The incidents are typically associated with blunt head injuries in military service resulting from motor vehicle accidents, military aircraft accidents, accidents during field training exercises or hand-to-hand combat, and combat. Penetrating head trauma occurs but is far less frequent than either blunt injury or injury from blast exposure. Combat experience can lead to blast exposure, but certain military occupational specialists, such as explosive ordnance disposal or military bomb disposal experts, will experience repeated blast exposure in their daily work. Military operations in Afghanistan and Iraq are associated with exposure to the IED, typically a small weapon that contains 20 to 30 pounds of explosive, and the often larger vehicle-borne IED, a device with charge sizes up to several thousand pounds of explosive.

There are 3 mechanisms by which an IED causes injury: blast, blunt impact, and fragment.

One of the most commonly encountered weapons in Operation Enduring Freedom and Operation Iraqi Freedom is the improvised explosive device (IED). From October 2001 until January 2005, explosive devices were responsible for nearly 80% of all casualties reported to the Joint Theater Trauma Registry. Compared with casualties in earlier conflicts, military casualties in Afghanistan and Iraq incur a greater percentage of injuries to the face, head, and neck. Improvements to personal protective equipment and vehicle armor have reduced death rates, but by improving survival, they may have increased the incidence of traumatic brain injury (TBI) among wartime casualties and perhaps also among veterans who appear physically uninjured. (We use the term “veteran” as an inclusive term for military personnel who served in a combat zone, regardless of the current status as a discharged veteran, active duty service member, or member of the military reserve or National Guard.)

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penetration. Of these mechanisms, penetrating injury is uncommon compared with blunt impact and blast injury, and when it does occur, the medical history readily informs the psychiatrist’s diagnostic task. The diagnosis of mild TBI often is far more challenging, particularly in the common clinical scenario of comorbid psychiatric illness. Although the clinical setting of a returning combat veteran suggests an epidemiological setting with elevated risk of TBI, cases of TBI are almost certainly being missed. Moderate or severe TBI is very likely to have resulted in medical care in a military setting, and records of this care should be available to the psychiatrist. Other factors contribute to the possibility of a missed TBI diagnosis, including an incomplete appreciation of TBI sequelae, attributing the clinical presentation to other psychiatric diagnoses, and misperceptions of blast injury biomechanics. On this latter point, we note that a study of blast injuries in civilians showed a missed diagnosis rate of 36% of primary blast–induced TBI cases. The complexity of trauma patients with other injuries may contribute to a missed TBI diagnosis, as also may the incorrect belief that loss of consciousness is necessary for TBI.

What is already known about traumatic brain injury (TBI) (and its comorbidities) in veterans?

- Combat veterans of Afghanistan and Iraq are at elevated risk for TBI and posttraumatic stress disorder (PTSD).
- The pathophysiology of blunt head injury is better understood than that for blast injury.
- Psychiatrists tend to be more comfortable diagnosing and treating mood and anxiety disorders than they are diagnosing and treating TBI.

What new information does this article provide?

- Most psychiatric consequences of TBI respond to a symptom-based approach established by the nearest equivalent DSM-IV disorder.
- Body armor is protective against blast injury to the lungs, thus allowing survival from injuries that were previously lethal.
- Stimulant medication can be useful for post-TBI cognitive difficulty, but it should be used with great caution in patients with comorbid anxiety.

What are the implications for psychiatric practice?

- Psychiatrists must consider TBI when evaluating or treating veterans, especially combat veterans.
- The psychiatric signs and symptoms of TBI can be similar to those of PTSD and thus may be easily overlooked.
- TBI is associated with many medical and neurological comorbidities, and an interdisciplinary team approach provides optimal care.

Blast exposure, body armor, and TBI

Injury risk from blast exposure has been studied for nearly 100 years and has mostly focused on pulmonary injury and fatalities from the blast. For much of this time, it has been generally accepted that the lungs are much more vulnerable than the brain for blast fatalities. Indeed, our findings suggest that the pressure levels needed to cause fatalities from blast brain trauma are about twice the levels needed to cause fatalities from pulmonary injury. However, substantial questions remain about whether sublethal brain injury can occur at levels of blast that do not result in pulmonary mortality. Emerging results suggest that the apparent brain injury associated with a blast exposure in recent military conflicts likely results from 2 sources. First, unpublished research by our group has shown that current military body armor, especially systems with a hard armor plate, is highly protective against pulmonary blast. This protection substantially decreases the risk of pulmonary injuries at a
given distance from the explosion. So, service members can now survive much closer to blasts than is possible without body armor, saving many lives that would have been lost previously, but resulting in a greater risk of mild and moderate head injuries. Second, our findings from recent studies of large animal models suggest that organic neurotrauma may occur at levels of blast that are near or below the level of pulmonary threshold injury. This compelling finding suggests that even civilians exposed to blast may be at risk for organic brain injury in addition to the risk of PTSD. These 2 sources, increased protection of the body relative to the brain and previously unsuspected vulnerability to blast brain trauma, are the likely sources of much of the blast-associated traumatic brain injury anecdotally reported in military hospitals and Veterans Affairs (VA) medical centers.

**Neuropsychiatric symptoms, comorbidities, and diagnostic dilemmas**

TBI can present with many different affective, anxiety, or cognitive symptoms. In the combat veteran, TBI is also seen with comorbid conditions, with the most common diagnoses being PTSD, major depression, panic disorder, and substance use disorders. Until the associated pathophysiology is better understood, there is not a single coherent scientific model of TBI. These facts lead to the present diagnostic model for TBI and its similarity to the criteria-based diagnostic system in DSM-IV. Although the DSM-IV authors were careful to create exclusionary criteria for similar conditions (e.g., schizophrenia diagnosis requires the exclusion of mood disorders), there are no such exclusionary criteria for PTSD and TBI. This situation leads to the possibility of arbitrary assumptions for diagnosing PTSD and/or TBI in blast-exposed combat veterans.

Reexperiencing symptoms can suggest the presence of PTSD; if it is present, it should be treated following established guidelines, such as the 2010 VA/Department of Defense Clinical Practice Guidelines for PTSD. The presence of specific neurological abnormalities can indicate TBI. An experienced neuropsychologist can look for relative deficits that are atypical for PTSD. An extended, multidisciplinary evaluation is often required for diagnostic accuracy; without an accurate diagnosis, the psychiatrist will be challenged in prescribing appropriate treatment.

Standardized tests can help clarify the diagnosis of suspected TBI and can complement the clinical assessment. These tests include bedside tests, also useful in the outpatient psychiatry office, and more involved tests up to and including a complete neuropsychological test battery. Tests can be useful for detecting and tracking psychiatric symptoms. We like the Beck Depression Inventory and the PTSD Checklist (PCL) for quick assessment of veteran outpatients. The PCL is an excellent instrument for capturing PTSD symptoms, but it can lead to false-positive diagnoses when TBI might contribute to or cause irritability, insomnia, impaired concentration, and apathy.

Cognitive difficulties are a common complaint after TBI and thus should be evaluated. Our institution uses a panel of tests to capture several different cognitive domains. Including a neuropsychologist on the interdisciplinary TBI team is highly recommended. These subspecialty psychologists can assist with difficult diagnostic situations, such as the influence of premorbid attention-deficit/hyperactivity disorder on cognitive complaints in a combat veteran with suspected TBI and/or PTSD; determination of any regional specificity to neuropsychological testing results; evaluation of personality tests; and administration of tests of effort, motivation, and malingering. Psychiatrists may consider bedside tests for patients with cognitive disorders, but evidence supporting the use of such tests is inconsistent. The Mini Mental State Examination (MMSE) is not recommended as a TBI evaluation instrument, and while the Montreal Cognitive Assessment (MoCA) may be better than the MMSE for detecting subtle abnormalities, there are no published trials of the MoCA in patients with TBI. Another brief clinical evaluation tool is the Military Acute Concussion Evaluation (MACE). The MACE was developed by the Defense and Veterans Brain Injury Center and can be ordered from their Web site (www.dvbic.org). The MACE appears to be a reliable instrument for TBI assessment, but according to a recent military medical study, the MACE is only effective if it is administered within 12 hours of a head injury.

Until better brief diagnostic tests are shown to be effective, our recommendations for TBI diagnosis include paying careful attention to physical signs and symptoms, noting the presence of cognitive disturbances that cannot be explained by known psychiatric or neurological comorbidity, and noting the presence of psychiatric symptoms that do not respond to usual care.

Among the possible comorbid diagnoses and shared symptoms, one significant diagnostic challenge is impaired motivation. The patient with subcortical damage may appear to be depressed. The clinical picture may include apathy, cognitive slowing, motor slowing, and a blunted emotional response. Consultation-liaison psychiatrists may note a similarity to other conditions, such as HIV encephalopathy and a subcortical dementia.
When a depressive condition is present after TBI, it is important to assess for depressed mood and emotional response to daily events. Many patients with disordered motivation are not particularly bothered by the impaired motivation. Depressed mood and true anhedonia suggest a mood disorder rather than subcortical injury sequelae. If a subcortical motivation problem is present, cautious use of low-dose stimulant medications (as outlined below) may be appropriate.

CASE VIGNETTE

Sgt B, a 24-year-old Marine Corps veteran of Afghanistan, is admitted to inpatient psychiatry. He is convinced that his religious beliefs are permanently altered by the Buddhist statues he had seen in Bamiyan, Afghanistan. He also reports auditory hallucinations of derogatory comments about his appearance. There is no family history of psychotic illness, but he reports 1 blunt head injury with a loss of consciousness that lasted 10 to 60 minutes.

Certainly, the diagnosis of paranoid schizophrenia is a leading possibility in a young man with new-onset delusions and hallucinations. However, with reports of psychosis after TBI, the psychiatrist must elicit a careful longitudinal history of symptom onset and duration. A thorough review of systems for possible TBI-related physical or neurological conditions also is indicated.

Neurological signs can also support a TBI diagnosis. When vestibular and oculomotor testing is available, it can be a useful technique in diagnosing TBI. In one case, the TBI diagnosis was confirmed by the presence of an internuclear ophthalmoplegia that was not present before military service in Iraq, and in another case, a unique vestibular problem in a blast-exposed veteran was identified. Neurological localization can be suspected from psychiatric or cognitive symptoms. One veteran in our clinic experienced a blunt head injury that should have affected his inferior frontal lobes. On examination, this veteran lacked awareness of social cues and insight into his interpersonal deficits, none of which were present before his injury. The spouse of another veteran from our clinic reported unusual sexual behaviors in the veteran, and video electroencephalographic monitoring confirmed frontal lobe seizure activity that was temporally correlated with the sexual behavior.

Treatment recommendations

The typical treatment recommendation for mood, anxiety, psychotic, or cognitive disorders in the setting of TBI is to treat the psychiatric disorder as if the TBI was not present. It also is important to discuss carefully any proposed treatments with the patient and/or family members because there are no medications with FDA-approved indications for psychiatric sequelae of TBI.

Clinicians also must remember the possibility of comorbid medical and neurological disorders. Among combat veterans, the most common comorbid condition with TBI will be PTSD, but it also is important to assess for major depression, panic disorder, and substance use disorders. If an Axis I disorder is diagnosed, it should be appropriately treated with medication and/or psychotherapy. The treatment of comorbid PTSD and TBI can be difficult because of overlapping clinical symptoms. We recommend serial evaluation of the reexperiencing symptoms as an outcome measure because these symptoms should not be affected by comorbid TBI.

Persistent and prominent arousal or avoidance symptoms, such as irritability and an apathy syndrome, may be caused by TBI, perhaps due to medical or neurological sequelae. When identified, these conditions should be treated or referred to the appropriate specialty physician. For example, endocrine deficiencies after TBI are not common but should be considered when facing “treatment-resistant depression.”

TBI affects psychosocial treatments. The presence of cognitive problems may affect the type or delivery of psychotherapy. There are no useful data to guide psychotherapy selection for veterans with comorbid psychiatric disorders and TBI. Our group often reviews neuropsychological test results before starting psychotherapy in the patient with a known TBI.

Neuropsychological testing may show specific deficits and relative strength. This information can facilitate psychotherapy by engaging the patient’s strongest cognitive domains whenever possible. Interdisciplinary team meetings between the mental health and TBI teams can be extremely helpful in planning a coordinated biopsychosocial treatment response. There remains a definite need for research into the optimal psychotherapeutic approach for comorbid psychiatric conditions and TBI.

The comorbid medical and neurological conditions associated with TBI can adversely affect clinical outcome. Common comorbid conditions include chronic pain, insomnia, and substance use disorders. Chronic pain has been reported in 43% of veterans with TBI. Approximately 50% of patients with TBI reported one or more sleep problems, including sleep apnea, periodic limb movements of sleep, and narcolepsy. Substance abuse can further complicate any cognitive difficulties, either directly as substance effect (eg, alcohol use) or indirectly by interfering with sleep. Mitigating these associated
conditions will improve patient outcome.

When addressing insomnia, psychiatrists should minimize the use of or avoid medications that may impair cognition. The medications to be avoided include anticholinergic or antihistaminic medications for sleep, tricyclic antidepressants for headache or neuropathy, and benzodiazepines for insomnia. However, short-term use of a hypnotic agent taken 3 to 5 nights a week may promote sleep; not taking the medication every night avoids both physical and psychological dependence. In most circumstances, the beneficial impact of improved sleep on mood, anxiety, and cognition more than offsets the adverse effects of the hypnotic agent. We have found good results with both lorazepam and zolpidem in this situation: these agents promote sleep without adverse effects. Promoting sleep with trazodone or hydroxyzine, while effective at night, may appear to worsen cognition the following day, and in a relatively young veteran population—a high percentage of whom are college students—morning sedation can be bothersome. Benzodiazepines are best avoided in the setting of TBI. These medications tend to exacerbate postinjury deficits in short-term memory. Of course, any combat veteran should receive a careful evaluation for PTSD; benzodiazepines are relatively contraindicated in patients who have PTSD. Before starting treatment with a benzodiazepine, the psychiatrist should be sure that the target symptoms are not just PTSD-related anxiety for which a PTSD-specific intervention would be more appropriate.

Because of the elevated seizure risk with bupropion, it is relatively contraindicated in patients with TBI. For mood disorders with comorbid TBI, augmentation with an alternative such as buspirone or triiodothyronine (T3) is suggested, with the former having more usefulness against anxiety and the latter showing more efficacy against depressive symptoms. Both buspirone and T3 were effective in the Sequenced Treatment Alternatives to Relieve Depression (STAR*D) study, but neither augmentation strategy has been evaluated in patients with TBI.

Many veterans with TBI complain of cognitive difficulties. Choosing the right medications can avoid iatrogenic worsening of cognition and even mitigate some cognitive problems. Make sure that the patient is not using substances that aggravate memory or attention problems, such as alcohol, excessive caffeine, illicit drugs, benzodiazepines, sedating antihistamines, and anticholinergics. Next, treat any comorbid Axis I conditions (eg, anxiety, depression) with an SSRI or serotonin-norepinephrine reuptake inhibitor and psychotherapy. It is important to recognize and manage chronic pain or a sleep disorder.

For cognitive problems that may be caused or aggravated by difficulties with attention, a cautious trial of stimulant medication is a consideration. Methylphenidate is known to improve cognition and mood after TBI, and it is recommended for patients who do not have an anxiety or substance use disorder. However, among combat veterans in a psychiatry clinic, the prevalence of both PTSD and substance use disorders will be elevated; thus, any stimulant use in this population must proceed cautiously and should be managed by a psychiatrist experienced in managing PTSD.

The rule of “start low, go slow” applies to methylphenidate in TBI. We find a starting dosage of 2.5 mg bid can be effective for mild attention problems, without any adverse effects on PTSD-related anxiety. At higher doses, certain patients may report improved cognition but increased anxiety. Each patient will respond differently, thus a careful dose titration is the only method for finding the optimal balance between improved cognition and increased anxiety. If a patient reports anxiety after starting methylphenidate treatment, make sure that he or she has eliminated caffeine intake because it can worsen anxiety.

One alternative to methylphenidate may be modafinil. This medication is more expensive and appears to be less potent that methylphenidate. It may be an appropriate alternative for the patient who cannot tolerate even small doses of methylphenidate, but it requires a stimulant to bolster attention. Any stimulant use should include a careful discussion of off-label use and the possibility of a significant increase in anxiety. Caution is warranted with the use of methylphenidate: an adverse reaction to methylphenidate may include new-onset suicidal ideation.

Substance abuse is another consideration when prescribing stimulant medication, because both clinical and medicolegal issues exist. Some patients with TBI may benefit from stimulant medication despite the presence of substance abuse. Neuropsychological testing can help establish which patients may benefit from stimulant medication. If the patient participates in a substance use program and demonstrates progress toward substance use recovery, then a stimulant medication may be an appropriate option. Close cooperation with the substance use disorder clinician or team is highly recommended; periodic urine drug screens also are appropriate. The overall clinical goal
should be to promote better cognition through substance abstinence and, if necessary, stimulant medication. A substance use disorder diagnosis by itself should not prevent appropriate medical therapy for impaired cognition if the patient demonstrates a willingness to participate in substance use treatment. State and/or local medical practice regulations may provide additional guidance on prescribing a controlled substance for patients with known substance use disorders. Psychiatrists in this clinical situation may wish to review the pertinent medical practice laws or regulations, consult with the state medical or pharmacy board, or seek advice from the hospital or clinic attorney.

Conclusions

Medical diagnosis begins with recognizing the epidemiological setting in which a patient presents. The combat veteran returns home from an occupational environment that presented physical, moral, emotional, and existential challenges that can only be imagined by most mental health clinicians. While psychiatrists are familiar with the possible diagnosis of a postcombat mood or anxiety condition, most medical education includes little exposure to brain injuries, and the natural tendency is to think PTSD when hearing “insomnia, irritability, and vague depressive symptoms.” This reaction often will be correct in combat veterans. However, the presence of PTSD or major depression does not rule out the presence of TBI. Among veterans of Afghanistan and Iraq, estimates of TBI prevalence range from 8% to 23%, and TBI must be considered as a possible comorbid condition. When the psychiatrist working with combat veterans finds either an atypical symptom cluster or an Axis I condition that does not respond to usual interventions, the wise choice is to consider TBI as an alternative or comorbid diagnosis.

Making an accurate TBI diagnosis in a combat veteran includes obtaining a history of past head injuries, including those injuries not considered significant by the veteran; performing a careful clinical assessment of psychiatric symptoms; possibly referring for neuropsychological testing; and providing symptom-focused treatment. Appropriate treatment can result in significant clinical benefit for the veteran.

References:

Dr Capehart reports that he is listed as the sole inventor on a patent application describing tizanidine for the treatment of certain psychiatric disorders. This patent is owned by the federal government and there are no current licensing agreements with this patent. Dr Bass reports that he has no conflicts of interest concerning the subject matter of this article.

References


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