Visual Dysfunction in Combat-related Mild Traumatic Brain Injury—A Review

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Abstract

Approximately half of all military personnel who have served in the conflicts in Iraq and Afghanistan are reported to have some degree of combat-related mild traumatic brain injury (TBI). Although in civilian concussion injuries symptoms typically resolve within several weeks, blast-induced mild TBI may be accompanied by prolonged symptoms and afferent and efferent visual dysfunction. Most commonly near vision problems and photophobia are the presenting symptoms. A complete eye exam including vision testing, oculomotor function, and near tasking, is highly recommended after blast-induced mild TBI to detect and improve symptoms in this young patient population. A review of the current literature is presented.

Traumatic brain injury (TBI) is the signature injury of the conflicts in Iraq and Afghanistan. The Department of Defense defines mild TBI by loss of consciousness for up to 30 minutes, or an alteration in mental state and/or memory loss, which lasts less than 24 hours and structural brain imaging yielding normal results.[1] Approximately 253,330 soldiers have been diagnosed with TBI since the year 2000.[1] The majority of these soldiers (76.8 %) were diagnosed with mild TBI while 17 % were diagnosed with moderate TBI, 2 % with penetrating and 1 % with severe TBI.[1] According to Lange, the overall numbers may be higher because many military personnel with mild TBI will never seek medical treatment.[2] Immediately after the TBI, post-concussion symptoms such as disorientation, slowed reaction time, headaches, and dizziness and blurred vision are common.[3,4] The natural history of mild TBI has been described to be self-limited with a predictable course.[5] In fact, studies show that most symptoms of mild TBI resolve completely by 6 months and only a small subset of patients has persistent symptoms beyond 1 year.[5–8] In recent years, observations of a more prolonged recovery after blast-related mild TBI have been discussed in the literature.[2,9,10] In a previous study, Wilk reported that among soldiers who reported loss of consciousness, blast mechanism of TBI was significantly associated with symptoms 3–6 months post deployment compared with a nonblast mechanism, although visual symptoms were not assessed in their study.[11] Explosive blast-related injuries are the predominant cause for TBI in the conflicts in Iraq and Afghanistan as a result of the preferred use of improvised explosive devices and improvised rocket-assisted mortars by the insurgency. The trauma to the brain from explosive blast injuries is thought to be more diffuse and complex than focal brain injuries from sports concussions commonly observed in the civilian population.[12–16] For example, malignant cerebral edema can develop rapidly over the course of one hour in blast-induced severe TBI as opposed to several hours to a day in closed head TBI.[15] Traumatic cerebral vasospasm in blast-induced TBI lasted for as long as

30 days compared to 14 days in closed head TBI. It has also been suggested that overpressure from the explosion may compress the abdomen and chest, inducing oscillating high pressure waves, which are transmitted upwards along the vessels, leading to perivascular brain damage.[16–18] In addition, hyperinflation of the lungs occurs, which can cause a vasovagal response leading to hypotension and possible cerebral hypoxemia.[16–18] Peskind recently reported decreased cerebrocerebellar metabolic rates in Iraq combat veterans with more than one episode of blast-induced mild TBI.[19] Significant brain injury can occur in blast-induced mild TBI. Several groups have recently diagnosed axonal damage in blast-related mild TBI patients with diffusion tensor imaging, despite normal MRI and CT studies.[20,21] Morey discusses a 'pepper-spray' diffuse pattern of white matter damage seen in Iraq and Afghanistan veterans with chronic mild TBI after blast injury.[20] These changes can appear as early as 1 month after the injury and extend further at 1 year.[20]

Blast Injury Mechanisms

There are several factors that can affect the degree of brain injury. At the time of the explosion a shock front is created followed by a blast wave, which expands until the pressure falls below atmospheric pressure.[16,17] Initially, the primary blast wave passes through body armor and bone, and is able to disrupt underlying tissues through embedded shear and stress waves.[13,22] Organ systems with high air content such as the pulmonary, gastrointestinal and auditory systems are the most susceptible, but overpressure also causes damage to the central nervous system, visual system, musculoskeletal and cardiovascular systems.[23] Secondary damage occurs when debris or fragmentation from the explosive device or surrounding objects penetrate the body.[24] Tertiary blast injury involves acceleration and deceleration forces, such as occur when a body is propelled and crashes into a fixed structure or the ground.[15] Quaternary injury occurs

Table 1: The Most Common Visual Symptoms after Mild Blast-induced Traumatic Brain Injury

Visual Symptoms	Lew (2007)	Brahm (2009)		Stelmack (2009)	Capo'-Aponte (2012)	Magone (2012)
Time since TBI	Not reported	Not reported		Not reported	15–45 days	51±20 months (mean±SD) 16–91 months (range)
Degree of TBI	Not reported	Only mild TBI subset reported here		Not reported (TBI only, no polytrauma patients reported here)	Mild	Mild
Mechanism of Injury	79 % blast	Non-blast	Blast	Not reported	Blast	Blast
Number of patients	62	12	112	36	20	31
Polytrauma	No	No	No	No	No	No
Inpatient	No	No	No	No	No	No
Outpatients	Yes	Yes	Yes	Yes	Yes	Yes
Best corrected visual acuity	Normal to near-normal	12 patients 20/60 or better	110 patients 20/60 or better, 2 patients worse than 20/100	97 % 20/20 2 % Legally Blind 1 % No Light Perception	20/20	20/20
Total subjective visual symptoms	75 %	75 %	76 %	42 %	Not reported	68 %
Near vision/reading problems	70 %	83 %	88 %	50 %	65 %	32 %
Visual field deficits	0 %	3.2 %		14 %	0 %	0 %
Light sensitivity	59 %	Not reported		59 %	50 %	55 %
Accommodative insufficiency	21 %	71 %	46 %	47 %	35 %	22.5 %
Convergence insufficiency	46 %	64 %	47 %	28 %	45 %	25 %

TBI = traumatic brain injury.

from burns or any other damage. In addition, damage can occur from blast wave reflection off surrounding structures.[21,23] If the explosion occurs in an enclosed space such as a vehicle or a building, then the reflected blast waves can exert potentiated damage.[22,24] Finally, the distance to the explosion plays an important role in the severity of the injury.[22,24]

Binocular Vision Dysfunction after Blast-induced Mild Traumatic Brain Injury

Cranial nerves three through six are involved in the fine alignment of the extraocular muscles and therefore it is not surprising that oculomotor dysfunctions occur in approximately 90 % of individuals early after TBI.[25] Capo'-Aponte studied in detail the near oculomotor functions in patients 15–45 days after blast-induced mild TBI and found that most patients had impaired reading abilities.[26] Compared to control subjects without mild TBI affected subjects had a higher rate of near exophoria, abnormal near point of convergence, decreased amplitude of accommodation and abnormal pursuit.[26] Magone found a much lower rate of convergence and accommodative insufficiency 50 months after blast-induced mild TBI, which indicates possible improvement with time.[27] However, the reported rate of 25 % for accommodative and convergence insufficiency remains at a 2.5-fold higher rate compared to mild TBI without a blast mechanism.[27,28]

Convergence insufficiency is common after TBI and is characterized by exophoria at near fixation, more than distance, and an abnormal near

point of convergence.[8,28] The rate of convergence insufficiency has been reported to be about 3–5 % in the general population and 9 % in civilian patients after TBI.[27,29] In patients after blast-induced mild TBI it ranges between twenty-five to forty-seven percent.[26,27,30-32] (see *Table 1*). Similarly, accommodative insufficiency ranged between 23–46 % in the previously mentioned studies.[26,27,30,31] It is recommended that all patients after blast-induced mild TBI be assessed for near oculomotor functions. Ideally, an ophthalmic evaluation prior to deployment to the warzone would allow a more accurate post-injury dysfunction assessment.

Occult visual dysfunction can impact reading performance and the patient's work performance and ultimately quality of life. In a recent retrospective study, 80 % of unemployed patients had accommodative and/or convergence insufficiency compared to only 31 % of employed.[27] The majority of patients after combat-related TBI will re-enter the civilian workforce after discharge from the military and therefore it is important to evaluate patients after mild TBI to recognize and treat associated visual dysfunction. Treatment options are tailored to the individual patient, but include reading glasses, fusional prisms, large print texts, and vision therapy to assist the patient with visual processing techniques.[33]

Photophobia after Traumatic Brain Injury

Light sensitivity after TBI in the absence of ocular inflammation is a common complaint, affecting 40–50 % of patients early on.[32-34] The

exact mechanism of persistent photophobia remains unknown.[31,33–36] Although rods and cones are the primary light transmitters of the eye, other pathways exist for light to activate the pain circuit.[37,38] Intrinsically photosensitive retinal ganglion cells were discovered in recent years and have multiple functions including pathways to pupil constriction and light avoidance.[39] Melanopsin photoreceptors of the iris in mammals can bypass the optic nerve to activate nociceptors outside the globe.[40] Experimental studies also describe a light-induced sensitization of the trigeminal pathway independent of the central visual pathways.[35,37–38]

Bohnen showed that tolerance to light and sound was decreased in patients 6 months after mild head injury compared to control subjects and suggested that the cause was a cortical and subcortical lack of inhibitory control.[34] Abnormal responses to light conditions and nonuniform cortical excitability have been described in other brain disorders associated with photophobia such as migraines and epilepsy.[41,42] A cortical hyperresponsivity could interfere with visual perception leading to photophobia.[42] These findings indicate that photophobia is triggered by more complex pathways than previously assumed and future research will elucidate the exact mechanism of this condition after mild blast-induced TBI. The mainstay of therapy for photophobia after mild TBI is the prescription of tinted lenses. Huang et al. found a reduction of cortical hyperactivation on functional magnetic resonance imaging in patients with migraine by using precision tinted lenses that block blue wavelengths compared to just grey lenses.[43] Suppression of stimulatory high frequency wavelengths of light associated with photophobia may explain the symptom reduction in patients suffering from light sensitivity.[44]

Table 1 summarizes the prevalence of visual dysfunction in outpatients diagnosed with TBI in the literature. Polytrauma and inpatient data from some articles are excluded as they typically have moderate to severe degrees of TBI and the emphasis of this review was to evaluate visual dysfunction after mild blast-related TBI (see Table 1).

Eye trauma associated with blast injuries is most commonly related to superficial or penetrating secondary blast injuries.[45–47] Fortunately, combat-related eye injuries have significantly decreased over the last years, because of better compliance with eye protection wear.[48–49] In polytrauma-associated TBI, closed globe injuries are more common than penetrating injuries.[50] Forty-three percent of combat blast survivors in a VA Polytrauma Rehabilitation Center had closed-globe injury, often affecting multiple zones; injury occurred despite use of protective eyewear.[50]

The first studies focusing on combat-related visual dysfunction in veterans who had been deployed to Iraq and/or Afghanistan were published in 2007. Goodrich and colleagues reported the incidence of visual injuries and dysfunction in 50 inpatients in a subacute rehabilitation setting after TBI.[51] Half of the patients had suffered blast-induced TBI, however, the degree of TBI was not listed. Overall, 74 % of patients complained of visual symptoms, and 20 % had near vision problems. In addition, 24 % of patients also had visual field abnormalities and 26 % of non-visually impaired patients had damage to the eye, orbit, or cranial nerve.[51] Cockeram described the early impact of blast-induced TBI on the visual system in an inpatient population without direct eye injury.[52] Findings included visual field defects, decrease in contrast sensitivity, and occult ocular injuries from blunt trauma despite good visual acuity.[52] Seventy percent of patients self reported vision problems and over 45 % had accommodative and convergence insufficiency.

A retrospective study by Brahm and colleagues from a polytrauma rehabilitation center reported a similar visual complaint rate of 76 % for blast-induced mild TBI and 75 % percent for nonblast-induced mild TBI.[31] Accommodation and convergence insufficiency were present in 46 and 47 % respectively, and were even higher in nonblast-induced mild TBI. Unfortunately, the time since injury was not studied, although the institution mostly treats subacute stages of rehabilitation both in inpatients and outpatients. Stelmack and colleagues performed a retrospective study in a polytrauma rehabilitation center.[32] Among 36 patients with TBI but without other injuries, half complained about reading problems. Twenty-eight percent of the TBI patients were diagnosed with a convergence insufficiency disorder and 47 % percent had accommodative insufficiency. The authors did not specify the severity of TBI in their study.[32]

A recent clinical study compared visual dysfunction in soldiers 15–45 days after blast mild TBI compared to deployed controls without TBI.[26] This is so far the first study with matched controls, which deployed to the warzone but did not suffer TBI. The authors found significant early visual dysfunction in soldiers after blast induced mild TBI despite excellent visual acuity.[26] Binocular vision problems, eye fatigue, and photophobia were the most commonly reported symptoms. Lew et al. reported persistent physical, vision and emotional problems in 38 patients up to 2 years after combat-related TBI, with the majority suffering a blast-related injury.[53,54] In a retrospective study of routine eye exams in 31 veterans with blast-related mild TBI, Magone found significant visual dysfunction in 68 % of patients almost 6 years after injury.[27] Patients complained predominantly of photophobia and binocular function problems.[27] Visual symptoms were more common after a dismounted mechanism of injury, where the patient was exposed directly to the blast wave. A subset of seventeen patients was not taking any systemic or topical medications and 59 % reported vision problems 49 months after the injury. Forty-seven percent complained of photophobia, 35 % had near vision problems, and 24 % had accommodative and convergence insufficiency. Fortunately, the incidence of symptoms in Magone's study decreased significantly with time, but the recovery was much longer than previously reported.[27]

In conclusion, it is becoming evident that significant visual dysfunctions occur in blast-induced brain injuries, and can result in prolonged disability and symptoms compared to non-blast mild TBI. Eye care providers should be aware that combat veterans might have occult closed-globe injuries, visual symptoms and dysfunction related to blast-induced mild TBI despite excellent visual acuity. A complete eye exam including near oculomotor function is highly recommended to detect and improve visual symptoms in this predominately young patient population. ■

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