Early Diagnosis and Treatment of Traumatic Vestibulopathy and Postconcussive Dizziness

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KEYWORDS

• Mild traumatic brain injury (mTBI) • Neurosensory sequelae • Dizziness
• Vestibular rehabilitation

KEY POINTS

- Neurosensory disorders are the most common sequelae of mild traumatic brain injury (mTBI), and among these, balance disorders are the ones most frequently seen.
- Balance disorders seen after mTBI can be diagnosed and treated, and whereas some resolve with time, many of these disorders require treatment.
- Vestibular rehabilitation is one of the most important treatment modalities available for patients with mTBI and has been documented to be successful in this patient group.
- Untreated mTBI can produce long-term degenerative neurosensory disorders.

INTRODUCTION

mTBI is an increasingly common public health concern that has garnered increased attention in both the lay press and medical literature. Neurosensory effects are among the most common sequelae of mTBI, with balance-related findings being the most

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common.[1–6] Balance disorders present a unique opportunity with respect to mTBI because they are almost universally present, they can be documented easily with qualitative and quantitative tests, and prompt treatment can result in marked improvement and return to function. On the other hand, untreated acute balance disorders after mTBI represent a significant and underappreciated public health issue. Chronic balance issues from mTBI can affect quality of life for many years after the injury and, in some cases, balance function can deteriorate unpredictably over time. The vestibular system itself has been shown to be damaged in mTBI as evidenced by documented benign paroxysmal positional vertigo caused by blast or blunt trauma mechanisms,[7] semicircular canal dehiscence,[8] and peripheral vestibular hypofunction.[8] This article begins with a review of the prevalence of mTBI in general, and then discusses clinical evidence indicating balance disorders after mTBI and examines the most recent trends in diagnosis. Finally, it discusses the recent basic scientific hypotheses regarding the cause of mTBI-induced neurosensory disorders and provides a brief overview of vestibular rehabilitation for mTBI.

THE DEFINITION OF MILD TRAUMATIC BRAIN INJURY

There are a variety of definitions of mTBI. Although these definitions were developed by health organizations and government agencies, there is no consensus definition. For a detailed discussion of this particular topic, the reader is referred to the Centers for Disease Control and Prevention (CDC) Web site,[5] which is an excellent resource for traumatic brain injury (TBI)- and mTBI-related health issues. For this article, the authors have adopted a basic functional definition of TBI as follows:

1. A traumatic event affecting the head (such as blunt trauma, explosion, or large acceleration-deceleration)
2. An event that resulted in alteration or loss of consciousness
3. An event with resultant neurologic symptoms and signs

Among individuals with such events, mTBI is defined by the following inclusion criteria:

1. Glasgow Coma Score greater than 13
2. Loss of consciousness for less than 1 hour
3. No surgical intervention needed (including a burr hole for drainage of bleeding)

Stated simply, mTBI (as the authors address it in this article) is a concussion. Although penetrating injuries can cause TBI, they are rarely mild and are generally associated with other types of disorders, so penetrating TBI is not addressed in this article.

EPIDEMIOLOGY

It is difficult to draw reliable conclusions from the existing epidemiologic data regarding prevalence and long-term consequences of mTBI. The lack of consensus in definitions and the fact that many mTBI cases go unreported makes epidemiologic classification problematic. It is clear that the reported prevalence of mTBI is increasing. Several reports focused on selected populations, such as high-school athletes[9] or emergency departments (EDs), over limited time frames[10,11] do give estimates of the relative prevalence of mTBI in different populations. A recent study by Marin and colleagues[12] used the Nationwide Emergency Department Sample to investigate trends of visits to ED for TBI between 2006 and 2010. The data culled from a sample of 950 hospitals showed a sharp increase in the weighted rates of

ED visits from 2006 to 2010. There were 637 TBI visits per 100,000 ED visits in 2006, and by 2010, this figure was up to 822 per 100,000, with a disproportionate increase in the number of reported mTBI or concussion visits.[11] What also seems to be apparent is that mTBI occurs preferentially in certain age groups. The CDC reported that almost 75% of all TBI visits were from individuals aged 0 to 4 years or older than 65 years.[1] This 75% figure is likely an overestimate because of underreporting from the 5- to 64-year-old segment of the population.

Certain established trends in TBI include a preponderance in men older than 75 years and motor vehicle accidents being the most common cause of TBI in individuals aged 15 to 24 years.[13] It is hard to know if these observations apply to mTBI as well. Although football injuries receive the most attention in the press,[14] there is a substantial literature regarding the occupational incidence of mTBI from the military alone.[1–5,15–17]

BALANCE DISORDERS AFTER BLAST INJURIES

There have been several studies examining balance disorders after blast exposure. An explosives detonation produces a shock wave, a blast wind, and an electromagnetic pulse. Primary blast injury is produced by shock wave propagation through tissue. The leading edge of the shock wave, the blast front, is an overpressure that propagates supersonically; it is followed by a negative pressure termed the underpressure. Reflections off environmental surfaces can produce more complicated exposures and greater injury. A single overpressure-underpressure blast wave sequence, described by a Friedlander wave profile, propagates into the brain case as a positive-negative shift in intracranial pressure.[18,19] Computer simulation studies have estimated deformations of the brain from blast waves,[20] which predict the greatest peak-to-peak effects in the posterior fossa.[21] Secondary blast injury is produced by shrapnel or fragments. Tertiary blast injury can produce blunt trauma by impact with objects in the environment. Quaternary blast injury is produced by other detonation products such as heat, electromagnetic pulses, and detonation toxins.

Hoffer and colleagues[1] described the symptom complex in blast-exposed service members with primary blast mTBI in the acute (81 subjects, within 72 hours), subacute (25 subjects, 4–30 days), and chronic (42 subjects, >30 days) periods.[1] Significant differences were noted in the prevalence of dizziness among the acute group (98%) compared with the subacute (76%) and chronic (84%) groups. Also, there was a significant difference in vertigo prevalence: 4% in the acute group, in contrast to 47% and 36% in the subacute and chronic groups, respectively. There was no statistical difference in frequency of hearing loss (33%–49%) and headache (72%–82%). From these data, 4 descriptive diagnostic classifications were established for dizziness after blast exposure: positional vertigo, post-blast-induced dizziness, postblast exercise-induced dizziness, and blast-induced dizziness with vertigo. These categories help to differentiate these patients from the more well-known entity of blunt trauma TBI (concussion). Differences include more constant symptom presence and dizziness that occurs during exertion rather than after exertion. Eye movements during rotational chair testing suggested worsening horizontal vestibuloocular reflex (VOR) function over time, but a larger study is needed to draw statistically sound conclusions.

Scherer and colleagues[2] examined vestibular test results in a group of blast-exposed service members divided into 2 groups based on the presence or absence of vestibular symptoms. In both groups, 83% had a concomitant blast and blunt trauma. All subjects were examined at least 30 days postinjury except for 1 symptomatic subject examined 14 days from injury. Each group had 12 subjects.

Videonystagmography results were abnormal in both groups, although slightly (but not significantly) more prevalent in the symptomatic group (6/12 vs 4/12). The rotational chair testing showed more of a difference between the 2 groups with 6 of 12 symptomatic patients exhibiting abnormalities in contrast to 1 of 10 in the asymptomatic group. Again, small sample size precluded any definitive conclusions. The abnormal videonystagmography results in the nonsymptomatic side were thought to be due to confounding medications and possible central pathology. Subjective self-report measures were significantly different between the 2 groups. The same group of patients was examined by Scherer and colleagues[22] who reported their results separately. This study was statistically powered to 0.80 with 12 subjects each, and active and passive head impulse testing was performed resulting in 4 different impulse rotation conditions: passive yaw, active yaw, passive pitch, and active pitch. Only 11 subjects from each group were reported because of excessive noise during signal recording for 2 subjects. Gain differences were found for active yaw and active and passive pitch, although there was variability in the findings. For example, 6 of 12 symptomatic subjects displayed angular VOR gains less than 0.85 indicating vestibular hypofunction, yet 2 of 12 had abnormally elevated gains that indicated cerebellar pathology. The study also included correlation of the angular VOR with vestibular symptoms during exertion. Passive pitch angular VOR gain showed an association, although the study was not adequately powered to draw definitive conclusions. Nonetheless, it suggests a possible avenue of investigation to find an objective measure of postblast exercise-induced dizziness that may be used as an outcome measure to establish fitness for duty.

Finally, Akin and Murnane[23] published an overview of blast injury and chronic vestibular consequences. In addition to their summary of findings over the past several years, they also published preliminary data for 31 symptomatic patients with long-term blast mTBI and/or blunt-impact induced mTBI seen in the Mountain Home Veterans Affairs Medical Center Vestibular/Balance Laboratory. Otolith dysfunction, manifested by abnormal cervical vestibular evoked myogenic potentials and/or abnormal judgments of subjective visual vertical in static and/or dynamic conditions, was present in 26 of 31 (84%) patients. In addition, 29% had caloric weakness and/or abnormal rotational chair results. The long-term prominence of these signs is consistent with our earlier report[1] of increasingly severe impairment when the first presentation is more than 30 days after injury.

BALANCE DISORDERS AFTER BLUNT HEAD INJURY

Blunt head injury has a well-known association with balance disorders.[24–26] Suarez and colleagues[24] demonstrated that the dizziness was one of the most frequent symptoms seen after mild head injury in a civilian setting. Meanwhile, Grubenhoff[25] examined the relative frequency of specific types of dizziness as a function of age in an mTBI group and found that younger individuals were more susceptible to potentially treatable causes of balance disorders. Hoffer and colleagues[27] examined blunt head trauma in a military population and characterized the dizziness seen after this type of trauma as a common symptom with patterns of dizziness different from the pattern that occurs after primary blast injury.

DIAGNOSIS

Diagnosis of mTBI has always presented challenges. This fact is true in part because many of the symptoms are self-reported with variable intensity over time. As discussed earlier, balance disorders present a unique window into the brain for

diagnosing mTBI. One of the most interesting and promising areas includes examining optokinetic and vestibular reflexes in response to a variety of visual and vestibular challenges. Working with Neuro Kinetics, Inc (Pittsburgh, PA, USA), the authors have described that a combination of oculomotor, vestibular, and symptom measures can discriminate patients with mTBI from control subjects.[28–30] Patients with acute mTBI can be identified with greater than 90% selectivity and sensitivity with a test battery that includes saccade testing (saccades, antisaccades, and saccadic reaction times), smooth pursuit performance, vestibular performance (harmonic rotation, visual enhancement/suppression of the horizontal, and head impulse testing), optokinetic testing, as well as self-reports of posttraumatic migrainelike symptoms. Other groups have similarly reported consistent eye movement findings. In 60 individuals with chronic mTBI, oculomotor testing revealed position and velocity error as well as saccadic intrusions, as measured using a head-mounted tracker attached to binocular cameras to track eye movement in response to visual or motion stimuli.[31]

Although not specific to balance disorders, other recent and relevant work investigating mTBI diagnosis is enlightening. A study in vision performed by the Veterans Administration led to the development of a 17-item tool designed to work in concert with 7 specific eye tests.[32] Magnetoencephalography was used to examine a large series of patients with mTBI and reported a specificity of 87% for correctly identifying mTBI.[33] Emergency and point-of-service diagnostic techniques for mTBI include the King-Devick test, sports concussion assessment tool (SCAT-2) test, acute concussion evaluation tool, and immediate post- concussion assessment and cognitive testing (ImPACT) test.[34–37] Although none of these tests focus exclusively on balance, each of them relies on balance elements in the test. For an extended review of imaging technologies, beyond the scope of this article, the reader is referred to an excellent article that summarizes functional MRI findings.[33]

TREATMENT

Advances in treatment of mTBI have been relatively slow to develop. In general, treatment can be classified as pharmacotherapy and rehabilitation. To date, the only pharmacotherapy that has demonstrated effectiveness is *N*-acetyl cysteine (NAC). NAC was found to be an effective countermeasure for blast-induced mTBI.[38] Working in a combat environment, a double-blinded placebo-controlled study revealed that NAC was far more effective than a control medicine at reducing symptoms measured at 7 days postinjury. Other pharmacologic methods are in development, but none have gone to clinical studies as yet. Work must be continued in this area. At present, therefore, the mainstay of treatment remains physical therapy.

The type of physical therapy that has been shown to be most effective and most common in treating patients with mTBI is vestibular rehabilitation.[39] Vestibular physical therapy (VPT) is a subspecialization within physical therapy that requires patients with dizziness and balance disorders to perform challenging postural, gait, and gaze stability tasks. Most VPT programs prescribe exercises to be done multiple times at home, presuming the patient is compliant and can do the exercises safely. The VPT may also involve outpatient visits. While there exists a significant body of literature examining the benefit of VPT in vestibular pathology,[40] there is an ever-increasing amount of evidence to support its role in treating mTBI. Alsalaheen and colleagues[41] have shown that vestibular rehabilitation improves outcomes and shortens disability times in patients with mTBI that did not improve with rest alone. Gottshall has demonstrated that return to work rates in this population is dramatically improved by vestibular rehabilitation. In order to study recovery more closely, Gottshall[42] developed a

battery of vestibular-visual-cognitive tests for establishing initial vestibular function and tracking the effectiveness of the VPT. The battery included the sensory organization test and motor control test as part of computerized dynamic posturography, static visual acuity, perception time, target acquisition, target following, dynamic visual acuity, and gaze stabilization tests (as part of the vestibular-visual-cognitive function testing with the Neurocom inVision Tunnel [Nuerocom Inc, Clakmas, OR, USA]). Performance was assessed at 0, 4, and 8 weeks from injury. The results were compared with previously collected, unpublished normative data. After 8 weeks, target following and dynamic visual acuity normalized. Although gaze stabilization scores improved, they did not approach normative levels within the 8-week time frame.[42]

Others have focused on different types of therapy, which have also been shown to be productive. Many individuals will have difficulty when attempting to read near targets because of the required convergence of the eyes creating symptoms or difficulty to physically perform. Thiagarajan and colleagues[43] trained subjects to do ocular fixation, predictable saccades, and simulated reading exercises in patients with vergence deficiency. The investigators not only found improvement in symptoms but also found significant improvement in errors in horizontal vergence.[43] Cervical spine physical therapy was shown to resolve symptoms in 73% of 15 patients with mTBI compared with only 7% in a control group.[44]

SUMMARY

mTBI is an increasingly common public health issue. Most of the acute, subacute, and chronic symptoms are neurosensory in nature and most commonly cause dizziness. Recognizing dizziness in this population is important because it provides a starting point for management of a difficult clinical entity, which can be measured objectively and treated effectively. Vestibular rehabilitation remains the standard treatment of mTBI, although clinical trials in the effectiveness of both rehabilitation and pharmacologic management are lacking.

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