Concussive Brain Injury: A brief primer for community physicians

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Quick Facts on Concussion

- 4 to 5 million concussions occur annually
- 33% of all sports concussions happen at practice
• 39% -- the amount by which cumulative concussions are shown to increase the chance of catastrophic brain injury leading to permanent neurologic disability

• 90% of most diagnosed concussions do not involve a loss of consciousness

Introduction
Concussive brain injury (CBI), also known as mild traumatic brain injury (MTBI), although quite prevalent, is still poorly understood by many health care professionals. CBI incidence, which is likely underestimated, accounts for about 80 - 85 percent of all traumatic brain injuries that occur each year in the United States (many of which never receive hospital based care). Falls are the most frequent cause of CBI, followed by blunt trauma (as in sports injuries), vehicular accidents and assaults in that order. The typical persons sustaining such injuries are young males.

Post-concussional sequelae may impede cognitive, behavioral, physical, emotional, and social function and thereby create relational, vocational and avocational challenges. Awareness of community resources is a prerequisite to providing neuromedical, as well as, non-medical services to this special patient population. The Brain Injury Association of Virginia (BIAV) (804-355-5748) is an excellent resource for information on brain injury related issues for professionals, persons with brain injury and their families.

The term concussion has its origins in the Latin word “concussus” which means “to shake violently”. Interesting, there is no internationally agreed upon definition of concussion making both research and clinical care more challenging. Traditionally, CBI is defined by the initial, post-resuscitation Glasgow Coma Scale (GCS) score of 13 to 15 without subsequent decline below 13. CBI has been defined as a traumatically induced physiological disruption of cerebral function, as manifested by at least one of the following: 1) loss of consciousness of no longer than 30 minutes; 2) any loss of memory, either retrograde or
anterograde (with post-traumatic amnesia being no longer than 24 hours); 3) any alteration in mental status at the time of the accident, even in the absence of loss of consciousness or amnesia; 4) physical symptoms which are potentially brain related (e.g. nausea, headache, dizziness, tinnitus, visual aberrations, olfactory deficits, or extended periods of fatigue); 5) development of post-traumatic cognitive deficits which cannot be completely accounted for by emotional factors. Notably, individuals with intracranial lesions and those with lower GCS scores (e.g. 13) generally have poorer cognitive and neurobehavioral outcomes and some have advocated that such injuries actually be labeled as moderate traumatic brain injuries and not MTBIs. All clinicians should be aware that loss of consciousness is not a prerequisite for making a diagnosis of CBI.

**Presentation and Prognosis**

Symptoms following CBI typically start at the time of injury or soon thereafter. There is no true “syndrome” associated with this neurological injury and symptoms may be cognitive, behavioral and/or physical with type and number varying greatly across patients. Symptoms are typically short term and usually resolve spontaneously. Common physical symptoms following these types of injuries can include headaches, dizziness, tinnitus, visual alterations including blurry and double vision, as well as, photosensitivity, among others. Concussive convulsions, although rare, can be a scary phenomenon but the good news is that they are not associated with long term risk of epilepsy. Cognitive-behavioral symptoms will vary based on time post-injury with early potential symptoms including amnesia, confusion, dazing and disorientation. Later cognitive-behavioral symptoms may include problems with attention, short term memory, and executive skills such as multitasking, organization, prioritization and judgment. Psychobehavioral changes that may be seen following concussion include dysomnias, emotional lability, depression and a spectrum of anxiety related disorders (including PTSD) which may or may not be directly related to the CBI itself. Interestingly, the more amnestic a patient is for the events at hand the less likely it is that they will develop PTSD.
Most patients with CBI, assuming there are no other significant injuries, whether physical or psychological, do well within relatively short periods of time assuming they do not come to the injury with substantive risk factors for protracted recovery or have secondary gain incentives which may promulgate their symptom presentation. Most individuals, who are otherwise healthy and without risk factors for protracted recovery, do well within 3 to 6 months following CBI. Very few patients with concussion have neurogenic symptoms due to the concussion beyond one year (probably less than 5%).

In those patients with more persistent symptoms, it is important to keep in mind that undue sensitization to distress from mild, negligible or benign symptoms can lead to a spectrum of abnormal illness behaviors and response bias in patient self-reports. Anxiety can augment symptom perception and health concerns. Sensitization may be especially relevant for post-concussive symptoms that often appear with similar frequency in the general population. Neurobiological theories of persistent symptomatology have been posited but there are likely multiple mechanisms responsible, many of which are probably non-neurogenic. Symptom magnification of post-concussive impairment can reflect multiple factors, including financial reward and psychological needs, including: garnering attention that would otherwise not be forthcoming; resolving pre-existing life conflicts; retaliating against employer or spouse; finding more socially acceptable attribution for psychological disorders; reducing anxiety and exerting a "plea for help" or soliciting acknowledgment of perceived difficulties. Depression, post-traumatic stress disorder and other anxiety conditions in which there can be sensitization or magnification of symptoms can represent important imitators of bona fide physical and neurologic impairment, as can symptoms generated by cranial/head trauma, as well as cervical acceleration deceleration injury (i.e. whiplash).

The following variables have all been found to contribute to poorer outcome and higher levels of functional impairment and disability: a) injury context variables including collateral non-cerebral traumatic injuries, especially cranial and cervical injuries, multiple injuries, greater motor impairment, presence
of chronic pain; b) premorbid biologic variables such as previous brain injury and older age; c) pre- and post-injury psychosocial variables including lower intelligence, history of alcohol or substance abuse, psychiatric history, poor school achievement, lack of social supports; d) personality and coping variables such as perceptions of victimization, over-achievement, dependency, grandiosity and borderline personality traits, along with childhood sexual abuse, post-traumatic stress and post-traumatic depression; e) environmental variables including litigation related financial incentives.

**Assessment**

The clinician should include a thorough medical history (including mental health), results of clinical evaluations, assessment of current clinical status, plans for future treatment, including rehabilitation and re-evaluation, diagnosis and clinical impressions, and an estimate of time for full or partial recovery as well as return to activities (i.e. school, sports, work, recreation). Any restrictions or accommodations should be stipulated. A good exam of a patient with CBI should include an elemental neurological exam including chemosensory assessment, a relevant musculoskeletal exam typically focused on the head, neck and upper shoulder girdle, and appropriate cognitive behavioral assessments as clinically indicated. There are now a number of on field, as well as office based assessments that can be used for general concussion, as well as cognitive screening, the latter which can be done serially to monitor recovery.

Importantly, the diagnosis of CBI should be based on: patient and chart history, temporal relationship of symptoms to injury in question, nature of post-concussive complaints and "fit" with expected symptomatology, corroboration by others including "non-invested" individuals, and degree to which symptom progression fits with the expected natural history of neurologic recovery. Adequate consideration must be given to alternative explanations for each impairment. Clinicians need to remember that symptoms associated with concussion can be seen in other conditions including affective disorders, chronic pain, and insomnia and are not, in and of themselves, indicative of concussion.
Assessment should also include testing to determine validity of symptom complaints (e.g. response bias testing).

Symptom descriptors used as diagnoses, such as "post-traumatic headache" and "post-traumatic dizziness," are not only incomplete but provide no pathoetiologic information which can translate to appropriate diagnostic and treatment strategies. Each condition/impairment should be listed with a presumptive pathoetiology, such that "post-traumatic headache" might be "post-traumatic headache secondary to right greater occipital neuralgia, right-sided referred cervical myofascial pain and migraine without aura". Objectification of symptoms should be sought when feasible and when doing so may alter clinical management.

Neuropsychological assessment is a much more sensitive measure than bedside examination and currently is considered an important tool in assessment of cognitive dysfunction in CBI; although it has its limitations. There is no consensus on when to send someone for this type of testing nor is there any agreed upon battery of tests for assessing CBI related impairments. Generally, if patients remain symptomatic at 3 months it is probably advisable to have such an evaluation performed. Due to the nature of assessing cognitive functions in analogue situations, it too often falls short of predicting real world work performance.

Because assessments include interviews about self-reported symptoms and rely heavily on measures of performance on standardized tests, valid results require patient veracity, cooperation and motivation; however, patients seen for presumptive brain injury related impairments often over-report pre-injury functional status in regard to post-concussive symptoms that often appear with similar frequency in the general population. Given the frequent highly desirable incentives to distort performance, particularly when there are secondary gain incentives, examinee motivation to provide truthful report and full effort is an extremely important prerequisite to valid assessment. Valid assessment is required for provision of: a) accurate diagnosis; b) appropriate and timely treatment to promote optimal recovery; c) prevention of iatrogenic impairment
and disability reinforcement, and promulgation of unnecessary health care costs; and d) appropriate legal compensation decisions based on causality and level of damages suffered. Certain populations such as athletes, as well as police, fire and military personnel will often tend to minimize rather than magnify symptom complaints to enable them to continue to “serve”. This type of response bias must also be assessed for and recognized when present.

The role of diagnostic testing remains controversial. In the acute setting, cerebral CT is indicated when there is a loss of consciousness, focal neurological deficit, abnormal GCS score, persistent altered mental status, suspected skull fracture or dural penetration, and/or clinical evidence of neurological deterioration. MRI may be useful when the exam remains abnormal and the CT is unrevealing. In the post-acute setting, imaging generally does not change clinical management but may be relevant in clinicolegal contexts as well as prognostication. There is currently insufficient evidence based medicine to recommend functional imaging studies, or volumetric brain imaging (singly or serially) as these tests cannot date injuries, nor are abnormalities on them necessarily pathognomonic of CBI as other conditions can present with similar findings. Most importantly there is insufficient data showing a correlation to clinical symptoms and/or prognosis, never mind an absence of evidence indicating that such abnormalities drive treatment decisions in any way whatsoever.

Treatment

There are no agreed upon treatment protocols for post-concussion symptomatology. Clinical practice guidelines have been proposed based on relatively scant evidence based literature most of which is not of a prospective, controlled and blinded nature with large sample sizes. Appropriate and timely referral to other specialists including psychologists, neuropsychologists, physical and occupational therapists, speech language pathologists, vocational rehabilitation specialists may all be relevant in the context of addressing impairment and disability in those with more persistent problems. That being
said there are general principles that are seemingly agreed to by those who treat this patient population including:

- Modulating pain as early as possible including pain symptom generators in the head/scalp and neck, the latter which can refer into the head and cause headaches
- Facilitating restorative sleep as expediently as possible
- Treating associated affective disorders and/or instability
- Limited down time with early progressive aerobic exercise program
- Providing cognitive compensatory techniques
- Early provision of focused education on concussion and prognosis for both patient and patient’s family
- Cognitive behavioral therapy as relevant to facilitate adjustment and minimize secondary psychoemotional impairments

**Chronic Traumatic Encephalopathy**

Chronic Traumatic Encephalopathy (CTE) is a progressive degenerative disease of the brain associated with repetitive brain trauma, including symptomatic concussions as well as asymptomatic sub-concussive injuries. Trauma triggers progressive degeneration of the brain tissue, including the build-up of an abnormal protein called tau. These changes in the brain can begin months, years, or even decades after the last brain trauma. The brain degeneration is associated with memory loss, confusion, headache, impaired judgment, impulse control problems, aggression, depression, and, eventually, progressive dementia. The issue of CTE and its causal relationship to recurrent concussion remains a hot topic of debate on several levels.

**Return to Work**

Employment determinations should be made based on the ability of the injured person to work, with or without accommodation, potentially with persistent objectively defined impairment(s) and meet job demands and other conditions of employment, including travel to and from work. Risks and restrictions need to be specifically defined for all involved parties. Getting people back to work at least
on a part time, decreased work capacity basis facilitates earlier work reentry and minimizes risks of longer term absenteeism as well as increases potential for earlier return to full work duty. Many employers, however, take the position that the injured worker can only return to their prior position at normal hours and normal work duty assignment….this approach is shortsighted to say the least and results in less likelihood of earlier return to work or return to work at all.

Return to Sports

Based on current evidence based guidelines, athletes should not return to play until their symptoms have abated, their neurological exam “normalized” including neurocognitive testing and they are off medications. Return to play should be graded with ongoing monitoring and if post-concussive symptoms recur then progression should cease. There has been a burgeoning of work looking at the role of subsymptom exacerbation aerobic training to facilitate recovery following concussion. Formal neuropsychological testing would be indicated in players with histories of multiple concussions and along with a focused neurological exam may be used to make decisions about retirement from contact sports.

Conclusions

The majority of individuals who sustain single concussive brain injuries do well, particularly if their injuries are not complicated by secondary issues such as PTSD, depression, chronic sensory disorders such as vestibulopathy, chronic pain, and/or other issues. Early education, appropriate rest with graduated return to aerobic exercise, referral to community resources and expedient modulation of early symptoms facilitates earlier recovery and minimizes risks for symptom persistence.

Recommended Reading


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