Update on Mild Traumatic Brain Injury Claims

By

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This is an update on a number of developments in the world of medicine pertaining to mild traumatic brain injury (TBI). But first a word of caution. When I first started handling brain injury cases over 20 years ago, like many practitioners today, my knee jerk plan was to have my client examined by a non-treating neuropsychologist. Currently, however, I am reluctant to engage a non-treating neuropsychologist for a number of reasons. First, neuropsychological testing is subject to interpretation, which removes it from the realm of objective proof of injury. Inevitably, test results reveal some basis for the defense to argue that your client has treatable depression or is malingering. Do you really want to inject a discussion of the MMPI's poorly-defined Fake Bad Scales and somatization disorders into your litigation? Why invite this trouble? Second, a common defense practice is to reactively hire an expert to match your expert. So, if you hire a neuropsychologist, the defense counsel will hire one. This adds to litigation expense and complexity, neither of which is a friend to the plaintiff's attorney. Finally, no matter what, you will need a neurosurgeon, neurologist, physiatrist, psychiatrist, or, at the very least, an internist to address causation. Consider letting your treating M.D. or D.O. carry the ball the whole way, with no assistance from a PhD.

To date, there is still no imaging study that can be used to display mild TBI, though early CT and MRI imaging can show hemorrhage or brain swelling. Further, there are no EEG findings that are unique to TBI. However, recent advances in medicine may soon change this. Researchers at the University of Wisconsin are using an advanced MRI to map the complex pathways of white matter. Likewise, the ongoing Human Connectome Project seeks to map white matter connections within the brain. PET imaging is most promising though not routinely used. FDG PET imaging can measure changes in brain metabolism following brain injury. Even more interesting, PET "Tau" scans show tauopathy which is pathognomonic for chronic traumatic encephalopathy (CTE).

Because there is no objective test to prove the existence of organic brain damage, many cases of TBI go undiagnosed or misdiagnosed by medical professionals. One study shows that ER personnel miss the diagnosis over 50% of the time. You should consider TBI in any case involving a significant impact, explosion or electrical shock. We routinely screen such clients (TABLE 1) and make referrals to healthcare providers when appropriate.

Even when the symptoms of TBI are recognized, unsophisticated health care providers will commonly misdiagnose patients as having post-traumatic stress disorder (PTSD) or, worse, depression due to symptom overlap with TBI. These misdiagnoses are problematic. The

defense will try to discount PTSD by arguing that the individual lost consciousness or suffered retrograde amnesia, and thus, has no memory of the traumatic event. When your client is misdiagnosed with depression, the defense will argue that it was caused by other life stresses that commonly befall many individuals and they will argue that depression is treatable. Given a temporal relationship to a significant head trauma, any fairminded healthcare provider should opine that there is likely an organic component to a chronic TBI symptom complex. If the treating provider will not see reason on this point, schedule your own IME.

The Center for Disease Control and Prevention (CDC) estimates that about 1.7 million new cases of TBI occur each year in the United States. As a result, TBI is receiving increasing attention from the medical community. Recent developments include the following:

- Dickstein, et al., Cerebral [18 F]T807/AV1451 Retention Pattern in Clinically Probable CTE Resembles Pathognomonic Distribution of CTE Tauopathy, *Transl Psychiatry,* (2016), vol. 6, no. 9, reports that PET Tau scans can show tauopathy – the pathological scarring seen on autopsy in individuals with CTE. Tau scans will likely be at the center of future TBI litigation, once further research is done to correlate imaging findings to pathology findings and to measure the rate of disease progression.

- In Lee, et al., Increased Risk of Dementia in Patients with Mild TBI, http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0062422, it is reported that individuals with mild TBI are 3 times more likely to develop dementia. Currently, 1 in 9 individuals between age 65 and 85 (all comers) develop dementia. One-third of individuals over the age of 85 (again, all comers) develop dementia. Adding a TBI to the mix triples the odds of developing dementia. The case can be made that an individual suffering a mild TBI should be compensated for future costs of care associated with dementia. These costs are huge and should be included in your life care plan. Publicity about the progressive deterioration of NFL players with CTE should make it easier to convince jurors that brain injuries worsen over time.

- In van der Naalt, et al., Early Predictors of Outcome After Mild Traumatic Brain Injury (UPFRONT), *Lancet* (2017), vol.16, no. 7, pp. 532-540, researchers showed that roughly half of patients with mild TBI will suffer long-term effects of the injury. This study corroborates findings in earlier studies by Selassie, et al., 2008,

Corrigan, et al., 2010, and Faul, et al., 2011, showing that 40% of TBI cases result in long-term disability. The UPFRONT study also identified the following risk factors for chronicity of symptoms: emotional distress and maladaptive coping early on after injury, pre-injury mental health disorders, less education, and older age at the time of injury – factors that are often used by the defense to try to minimize the effects of TBI.

- In Bavisetty, et al., Chronic Hypopituitarism After Traumatic Brain Injury, *Neurosurgery* (2008), vol. 62, no. 65, pp. 683-688, the authors showed that 20% of patients with TBI suffer a concurrent pituitary injury. Hypopituitarism can be tested for by an endocrinologist and, when present, provides objective evidence of pituitary injury following head trauma. *See also* Kelly, et al., Prevalence of Pituitary Hormone Dysfunction, Metabolic Syndrome, and Impaired Quality of Life in Retired Professional Football Players, *J Neurotrauma* (2014), vol. 31, no. 13, pp. 1161–1171.

- In Rea, et al., Pupillary Dysfunction in Post Traumatic Headache, (2016) *ANA Journal*, Abstract M307, the authors reported that dysfunction of the sympathetic nervous system following trauma may play a role in post-traumatic headaches. Sympathetic nerve dysfunction can be assessed by measuring pupillary dilation. Post-traumatic headaches, unlike migraines, are notoriously persistent and refractory to treatment.

- In August, 2016, the FDA approved the ImPact and ImPact Pediatric tests to assess cognitive function following a concussion. The test assesses memory and reaction time, and compares them with an age-matched control database. The FDA specified that the test is "not intended to diagnose concussions or determine appropriate treatments."

- In Carney, et al., Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition, *Neurosurgery* (2017), Vol. 80, no. 1, pp. 6-15, newly updated guidelines for treatment of *severe* TBI underscore the debilitating nature of severe TBI and the fact that once brain tissue is destroyed, it cannot be recovered.

Public awareness of TBI has skyrocketed with the widely publicized lawsuit against the NFL over sports-related CTE. Public awareness of PTSD has also increased with the return of many military personnel carrying that diagnosis. Likewise, it seems that everyone is being treated for depression these days. Naturally, defense attorneys will inquire at deposition about your client's history of concussions, their exposure to other traumatic events and stressors, and their mental health history. But also be aware that many jurors will now have pre-formed, often erroneous ideas about TBI, CTE, PTSD and depression. These issues must be addressed at trial, beginning with jury selection.

TABLE 1: TBI SCREENER

Were you diagnosed with a concussion?
Have you had any head imaging?
Do you remember the accident?
Did you lose consciousness?
Describe the impact.
Did you hit your head/face?
Are there photos depicting head/face injury?
Did the airbag deploy?
Do you have headaches?
Tinnitus?
Dizziness?
Chronic fatigue?
Difficulty with memory or concentration?
Confusion or slow thinking?
Visual disturbance?
Seizures?
Anger outbursts or unusual irritability?
Nausea?
Loss of Smell?
Light or sound sensitivity?
When was the onset of these symptoms?
Did you report these symptoms to providers?
Have you had any of these symptoms before?
Have you had other concussions/head trauma?
Have you ever been treated for depression?
Other mental health history?
List other emotional trauma/life stressors.
List all pre- and post-injury medications.