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Advocacy! The next generation towards full recovery for TBI plaintiffs

A LOOK AT THE MYRIAD OF SYMPTOMS THAT CAN RESULT FROM TBI AND THE MEDICAL SPECIALISTS WHO TREAT THEM

Traumatic brain injury is now recognized as one of the leading causes of death and disability, not only in the United Sates, but globally. (Giner, *Traumatic brain injury in the new millenium: New population and new management* (2022) 37 Neurologia 5, pp. 383-389.) Medicine has advanced in documenting not only the event of TBI but the diagnosis and management.

In my experience, however, it is the rare doctor who takes a proper history, performs a proper physical examination, and makes a comprehensive diagnosis of TBI utilizing the latest science. Comprehensive in this setting means applying all present knowledge of TBI, to include the severity of the initial event, the initial symptoms and sequelae, nature of any recovery, and an in-depth inquiry into any further manifestations/ consequences of the TBI.

Medical science has taken significant leaps forward in discovering a host of post-TBI medical complications stemming from the original event. Inquiry into these complications, initially and on an on-going basis, is indispensable in the comprehensive diagnosis of TBI. There are excellent articles detailing the abundance of post-TBI complications and calling for TBI to be looked at, not only as an event, but an ongoing chronic disease process. (Masel, *Traumatic brain injury: a disease process, not an event* (2010) 27 Journal of Neurotrauma 8, pp. 1529-1540; Corrigan, *Traumatic brain injury as a chronic health condition* (2013) 94 Archives of Physical Medicine and Rehabilitation 6, pp. 1199-1201.)

Treatment is fragmented

It's safe to say the practice of medicine regarding TBI is fragmented into multiple specialties, with each specialty addressing a particular aspect of post-TBI complications. Take neurologists, for instance, who focus on seizures, or otolaryngologists, who investigate tinnitus/vestibular imbalance. But there is no treating medical doctor responsible for outlining the total complications that are present in a particular TBI patient and making the appropriate referrals to any and all specialists for accurate verification and proper treatment.

If we had TBI generalists advocating for all things medical with our clients, then our lives would be much easier. We lawyers would be easily advocating for recovery based on medical evidence of every complication of a particular TBI. But the world of TBI medicine is fragmented, our clients partially diagnosed and partially compensated. And certain treating doctors may have not yet reached the point of a comprehensive approach to TBI in their practice of medicine – same with some attorneys in our practice.

What to do? How can we advocate for the missing parts of the medical picture in our TBI clients? How do we build a next generation of advocacy for our TBI clients? The answer is twofold.

First, we have to acknowledge the diverse medical repercussions of a TBI. What follows is a review of the most cited and researched medical aftermaths of a diagnosed TBI. This review establishes that these consequences are now receiving mainstream medical attention and are slowly becoming hard driven spikes on which to hang an expanded advocacy for fuller recovery in our TBI cases.

Headaches

Post-traumatic headaches (PTH) are among the most common and persistent symptoms encountered in clients across the entire mild, moderate, and severe landscape of TBI and they can be challenging to treat. (Dwyer, *Post-traumatic cephalalgia* (2020) 47 NeuroRehabilitation 3 (2020), pp. 327-342.) Approximately 18-20% of PTH's lasted longer than one year across TBI severity. (Lew, *Characteristics and treatment of headache after traumatic brain injury* (2006) 85 American Journal of Physical Medicine & Rehabilitation 7, pp. 619-627.) The sobering truth is that more often than not, the TBI client walking into your office may be dealing with these headaches.

The productivity constraints on a client suffering from symptomatic headaches combined with a possibility of interference resulting from the need for TBI-related rehabilitation present a scenario that demands acute and chronic medical management and thus, demands acute and chronic compensation.

When you identify this disorder in your TBI client, either a referral to a regional headache clinic at a major university hospital system or to a private practitioner of neurology with a specialty in headaches is in order.

Psychological/emotional disturbances

Post-TBI emotional disturbance is as common and long-lasting as headaches – perhaps even more so. A majority of people experience psychiatric disturbances in the 12 months following a TBI, with affective disorders such as depression and anxiety being the most common. Other concurrent psychological and behavioral disorders can be present, including irritability, frustration intolerance, anger, aggression, impulsivity and apathy.

Emotional disturbances have been a long-term disruptive factor in TBI patients returning to prior psychosocial function, including family, friends and work. Excessive levels of anxiety have been associated with significant

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impairment across several life spheres including occupational, social, and household/activities of daily living. In some cases, the level of impairment is greater than is seen in individuals with major depression.

And importantly, a high prevalence of PTSD, anxiety and depression has been documented following TBI, which contributes to poorer function, worsened outcomes, including increased risk of suicide. In a study examining the psychological and emotional outcomes and psychosocial functioning of patients in the 10 years following a TBI, it was found that higher levels of anxiety and depression were associated with poorer functioning across all domains of psychosocial functioning. (Draper, *Psychosocial and emotional outcomes 10 years following traumatic brain injury* (2007) 22 The Journal of Head Trauma Rehabilitation/Journal of Head Trauma Rehabilitation 5, pp. 278-287.)

There is also a strong association between a history of TBI and an elevated risk of suicide. This risk is present across all levels of TBI severity and does not decrease, even after many years post injury. (Bahraini, *Suicidal Ideation and Behaviours after Traumatic Brain Injury: A Systematic Review* (2013) 14 Brain Impairment 1, pp. 92-112.)

When you detect a disturbance in your client that approaches excessive anxiety or depression or there is PTSD, alcohol abuse, etc. a referral to a trusted psychiatrist is necessary. As your case progresses, all areas of ongoing and future compensation resulting from this disability must be documented including psychiatric/psychological consultation, medicines, home care, hospitalizations, and unemployment.

Sleep disorders

Disordered sleep is a common finding in the weeks after the acute phase of a TBI, and close to 50% of all TBI patients suffer from sleep-wake disturbances. (Mathias, *Prevalence of sleep disturbances, disorders, and problems following traumatic brain injury: A meta-analysis* (2012) 13 *Sleep Medicine* 7, pp. 898-905.) The most common sleep-related disorders following a TBI are insomnia, increased daytime sleepiness, increased need for sleep, and circadian rhythm disturbances (disturbances in the sleep/wake cycle or when you sleep and when you are awake vs. the norm). There are other manifestations of sleep disorders as well, including obstructive sleep apnea (OSA), narcolepsy, and excessive fatigue.

The prevalence of sleep disorders cannot be overstated. A study of 200,000 United States military veterans compared the incidence of sleep disorders over a 14-year period – half of the veterans had TBI and half did not. Those veterans who had a TBI were 41% more likely to develop any sleep disorders as compared to those veterans with no history of TBI. (Leng, *Traumatic brain injury and incidence risk of sleep disorders in nearly 200,000 US veterans* (2021) 96 Neurology 13, pp. 1792-1799.)

Identifying sleep disorders in your TBI clients is an essential component of any intake. Should these symptoms be present, it is appropriate to refer the client to a local medical specialist who specializes in sleep disorders or to a recognized sleep clinic.

Dizziness and imbalance

After headache, dizziness is the most reported symptom following TBI; it may or may not be associated with a sense of balance loss. In some TBI patients this will disappear with time and treatment; various studies have pointed out the nagging persistence of symptoms. (See, e.g., Kleffelgaard, *Associations among self-reported balance problems, post-concussion symptoms and performance-based tests: a longitudinal follow-up study* (2011) 34 *Disability and Rehabilitation* 9, pp. 788-794.) In these patients, it can obviously interfere with complete recovery and be associated with poor employment outcomes. The mechanisms for this symptom complex are multifactorial after a TBI but include peripheral vestibular injuries and central nervous system vestibular injuries.

The treatment and management of this symptom is complex. There is a plethora of tests designed to delineate the etiology of the dizziness and sense of imbalance. The usual medical specialists that handle this difficult problem are otolaryngologists and neurologists. Moreover, there are specialized standalone clinics that conduct this testing.

Tinnitus

Tinnitus is so frequently encountered in TBI patients that we – lawyers and medical professionals – tend to not fully appreciate its significance. "Tinnitus, the perception of sound in the absence of an external auditory stimulus, is perceived by about 1 in 10 adults and thus among the most prevalent symptoms of hearing disorders in industrialized countries. Although many people learn to ignore and compensate for the phantom sound, the quality of life is severely affected by tinnitus in about 1-3% of the population. Tinnitus can cause severe distress in individuals, and it has been shown to be associated with sleeping disorders, depression and anxiety. It may affect concentration and ability for attentional focusing and working memory and is very difficult to treat. Development of tinnitus may even end up in suicidal attempts." (Kreuzer, *Trauma-Associated tinnitus* (2014) 29 The Journal of Head Trauma Rehabilitation/Journal of Head Trauma Rehabilitation 5, pp. 432-442.)

Individuals report several types of tinnitus sensations including ringing, buzzing or a rushing sound and the auditory perception varies across individuals in terms of intensity, frequency and duration.

In one study of adults aged 21-45 years, all recovering from TBI that had previously occurred (range 19 months-27 years), the prevalence of tinnitus was reported to be 53%. (Nölle, *Pathophysiological Changes of the Central Auditory Pathway after Blunt Trauma of the Head* (2004) 21 Journal of Neurotrauma 3, pp. 251-258.) Thirty-three percent also had sensorineural hearing impairment

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(a degree of hearing loss). Auditory dysfunction can be a side effect of blunt head trauma.

Referral to an otolaryngologist for tinnitus is appropriate to begin the period of diagnosis and treatment and to establish whether treatment is successful, partially successful or has failed. Compensation for all variables must be determined for the future to include medical and psychiatric consultations, medicines, appliances, surgery and negative psychosocial outcomes. Not to be overlooked is the common presence of hearing loss in your clients with tinnitus and any disabilities that result from this impediment. A referral to an audiologist is required.

Temporomandibular disorders and bruxism

The term temporomandibular (TM) disorders consist of disordered conditions of the temporomandibular joint (TMJ), the masseter and temporalis muscles, and the trigeminal nerve (5th cranial nerve) that serves them. Associated signs and symptoms include regional pain, clicking of the joint, limited mouth opening, deviation and deflection during opening and closing of the mouth, tenderness in the masseter and masseter muscles, headache, earache and malocclusion that affects the TMJ and the muscle, bone and soft tissues components with which it is associated.

Recognition of TMJ-associated problems in patients with TBI has come late to the party. (Babiloni, *Temporomandibular disorders in traumatic brain injury patients: a chronic pain condition requiring further attention* (2020) 21 Pain Medicine 12, pp. 3260-3262.) The problem of TMJ is often blurred between the professions of dentistry and medicine. In my experience involving clients with TMJ disorders, they have been treated with biofeedback, dental consults and bite splints. One doctor that I've worked with was emphatic that, in head and neck injuries especially, it is routine for the patients to clench their jaws at the time of impact to such an extent as to cause inflammation in the masseter and temporalis muscles. This inflammation would continue unless treated in a proportion of patients and cause TMJ problems for many years with headaches and jaw pain. Of note here is that TBI is frequently associated with facial trauma – thus further showing the connection between TBI and TMJ.

In recent years there has been a growing interest in bruxism, which is excessive activation of the masseter and temporalis muscles resulting in involuntary, repetitive, jaw muscle activity resulting in rhythmic or spasmodic gnashing, grinding or clenching of teeth and/or bracing of the mandible, mainly at night. Bruxism can occur outside the context of TBI, but it is common in TBI patients. This results in dental wear, loss of teeth, jaw pain, headaches and can lead to TMJ disorders.

In your patients who identify bruxism, jaw pain, temporal headaches and/or inability to open their mouth completely, have your client consult with a dentist who specializes in TMJ. Anti-inflammatory medication, bites splints and physical therapy are mainstays of treatment. This referral should occur soon after discovery of bruxism/TMJ Disorder to avoid chronicity.

Post-traumatic epilepsy

Post-traumatic epilepsy (PTE) is recognized as a significant and potentially drastic consequence of a TBI at all levels of severity. PTE is divided into three different categories. First, is concussive seizures, which often occur almost instantaneously at the time of the occurrence of the TBI. These are often self-limited and are not frequently associated with the development of epilepsy in the future. Second, is early posttraumatic seizures, which typically occur within seven days of the TBI. Third, is late epileptic seizures, which occur beyond seven days after injury.

Early posttraumatic seizures are a predictor of late traumatic seizures and epilepsy. (Majidi, *Prevalence and Risk Factors for Early Seizure in Patients with Traumatic Brain Injury: Analysis from National Trauma Data Bank* (2016) 27 Neurocritical Care 1, pp. 90-95.) And individuals with late traumatic seizures carry a higher risk of developing epilepsy than the general population. (Ritter, *Incidence and risk factors of posttraumatic seizures following traumatic brain injury: A Traumatic Brain Injury Model Systems Study* (2016) 57 Epilepsia 12, pp. 1968-1977.) This is true across mild, moderate and severe TBI.

Most patients remain at risk for epilepsy after mild TBI for up to five years, after moderate TBI for up to 10 years, and after severe TBI for 20 years or more. (Annegers, *A Population-Based Study of Seizures after Traumatic Brain Injuries* (1998) 338 New England Journal of Medicine 1, pp. 20-24.)

Sadly, this last statistic has been replicated by numerous studies. Across all age groups, standardized mortality ratios range from two to three, indicating that the observed numbers of deaths in people with epilepsy are two- to threefold higher than in reference populations without epilepsy. What is important is that people also died from epilepsy-related causes and not epilepsy per se.

Thus, one needs to be vigilant with cases of TBI for any evidence of PTE. Seizure activity takes several different forms, beyond the typical grand mal seizures that most people are familiar with. In-depth screening for the various types of seizures within your TBI clients should be routine practice at your firm, because often these episodes are misunderstood and underreported. This disorder can be destructive of quality of life and daily functioning on many levels. Once diagnosed with PTE, planning for lifetime compensation needs is often in order. This needs to include compensation for PTE, the increased risk for psychiatric consequences, including suicide and the prospect of premature death and advocating for funding for proper medical care to help counter such prospects.

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The neurodegenerative disorders

A neurodegenerative disease is one caused by the progressive loss of structure or function of neurons – i.e., a process known as "neurodegeneration" that can ultimately involve cell death. These diseases include amyotrophic lateral sclerosis, multiple sclerosis, Parkinson's disease (PD), Alzheimer's disease (AD), Huntington's disease, multiple system atrophy, tauopathies, and prion diseases. Neurodegeneration can range from molecular to systemic. These conditions are irreversible, incurable, and they all eventually lead to death.

The neurodegenerative diseases that will be our main focus here will be Alzheimer's Disease (AD) and Parkinson's Disease (PD). Because it is the most common neurodegenerative disorder, I will begin with a discussion of Alzheimer's and its relationship to TBI.

Dementia is a state of an individual associated with an impaired ability to remember, think or make decisions that interferes with doing everyday activities. AD dementia is a disease process associated with specific criteria, which have been promulgated by the by the National Institute on Aging and the Alzheimer's Association (NIA-AA).

The NIA-AA criteria for probable AD dementia require the presence of dementia and the following characteristics:

1. Interference with ability to function at work or at usual activities
2. A decline from a previous level of functioning and performing
3. Not explained by delirium or major psychiatric disorder
4. Cognitive impairment established by history-taking from the patient and a knowledgeable informant; and objective bedside mental status examination or neuropsychologic testing
5. Cognitive impairment involving a minimum of two of the following domains:
   - Impaired ability to acquire and remember new information
   - Impaired reasoning and handing of complex tasks, poor judgment
   - Impaired visuospatial abilities
   - Impaired language functions
   - Changes in personality, behavior, or comportment
6. Other clinical criteria include:
   - Insidious onset
   - Clear-cut history of worsening
   - No evidence of another concurrent, active neurologic or non-neurologic disease or use of medication that could have a substantial effect on cognition.

Many consider AD to be an old person's disease – because it is unusual to diagnose AD in patients under 60. Of the small percentage of persons with documented AD before the age of 60, TBI has been found to be an acquired factor in the early onset of AD. In recent years, medical literature has demonstrated that across all levels of severity, TBI is associated with an earlier onset of AD.

In a study of 100,000 patients, with almost a third of that population being identified as having an mTBI, the diagnosis of mTBI carried a significant independent hazard factor of 3.26 for developing dementia. (Lee, Increased Risk of Dementia in Patients with Mild Traumatic Brain Injury: A Nationwide Cohort Study" (2013) 8 *PloS One* 5, p. 62422.)

There is a wealth of material documenting the relationship between TBI and the development of early dementia/AD. These reports cover all levels of TBI and many durations of time between the actual event and the onset of dementia/AD. What they all emphasize is the early onset of dementia/AD.

Parkinson's Disease (PD) is the second most common neurodegenerative disorder. It typically manifests as bradykinesia (slow gait), rigidity, resting tremor and posture instability. PD is primarily characterized by death of neurons in the substantia nigra, a region of the midbrain. The cause of this selective cell death is widely unknown.

Age is the most important risk factor for PD. The incidence and prevalence rise steadily in adults beginning in the fifth decade. Nonetheless, PD is not solely a disease of older adults. Approximately 25 percent of people with PD are diagnosed before the age of 65 years. Multiple studies have reported a strong correlation between TBI and the onset of PD.

Pituitary hormone dysfunction

The pituitary gland is located at the base of the brain, protruding off the bottom of the hypothalamus; it's home to the production of eight hormones. Like other components of the brain structure, the pituitary gland can be impacted by physical forces that potentially lead to a primary brain injury TBI. Even in the absence of a direct impact, significant acceleration or deceleration of the head can cause a TBI; however, in most cases, a combination of impact and acceleration is probably to blame. Then the pituitary gland, like the brain in general, must deal with the consequences of a possible secondary brain injury mediated by brain transmitter chemicals excitotoxicity, ischemia from blood vessel spasm and inflammatory responses.

For our discussion, I want to focus on Growth Hormone (GH), which is one of the common pituitary hormones found to be compromised in production following a TBI. Pituitary dysfunction with reduced GH production is considered a common consequence of TBI. (Gasco, *Traumatic Brain injury as frequent cause of hypopituitarism and growth hormone deficiency: Epidemiology, diagnosis, and treatment* (2021) 12 Frontiers in Endocrinology, p. 634415.)

This deficiency of GH (GHD) may be temporary or permanent. When permanent, the neurological and functional sequelae can lead to significant impairment, and this demands that we remain aware of its possible presence in order to facilitate diagnoses, treatment and future compensation. Unfortunately, we cannot always rely on our usual medical providers to identify this diagnosis. So we, as a new generation of advocates, have to be aware of its potential manifestation in our TBI clients. Logically, that leads us to the signs or symptoms that indicate that there is a GH deficiency after a TBI.

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During the acute stage, which is considered the first two weeks after trauma, GHD is a common shift. The chronic phase of TBI begins at three months, and GHD remains one of the more common pituitary abnormalities. However, separating GHD from other post-traumatic consequences of a TBI can be challenging.

The most common symptoms of GHD are fatigue, anxiety, depression, diminished cognition with memory impairment and lack of concentration, irritability and loss of libido. Other quality-of-life-related symptoms that may be present are anxiety, sleep disturbance and other cognitive impairments. As is obvious, there is overlap with ongoing symptom complexes related to other injury processes at play after a TBI. As your TBI client progresses through the post-TBI incident phase and these symptoms remain in evidence, consider having them evaluated for pituitary dysfunction; and certainly so if these symptoms persist into the 6-12 months post-TBI phase.

Besides the quality-of-life factors, muscle loss, fat gain and the possibility of fractures, there are other problems that may arise from unrecognized and untreated GHD. There is a significant increase in cardiovascular events, possibly due to atherosclerotic changes in the blood vessels. And even more pressing is the evidence that TBI GHD in adult life results in a reduced life expectancy.

With this knowledge in hand, it is hard not to be cognizant of GHD in your TBI client, who faces you across your conference table at 6-12 months into their recovery period. You should know that the diagnosis and treatment of GHD is a difficult one to achieve and you must contact an endocrine specialist who is well versed in this pursuit. Know as well that the treatment in the form of an injection is going to be lifelong. Resistance to this pursuit from the usual cast of characters is to be expected, as it can become quite expensive.

Life expectancy after TBI

There is growing medical literature that confirms that among patients that survive, there is a lower life expectancy for TBI patients than for the general population. This should not be surprising as it is becoming increasingly accepted that TBI is a chronic disease process, and many survivors have resulting and longstanding disease problems that are susceptible to worsening over time. As compared to those in the general population, those who suffer a TBI experience a reduction in life expectancy of six to seven years. (See, e.g., Harrison-Felix, *Mortality after surviving traumatic brain injury* (2012) 27 The Journal of Head Trauma Rehabilitation 5, p. E45-E56.)

Individuals with moderate to severe TBI who survive at least one year post-injury are 37 times more likely to die of seizures, 12 times more likely to die of septicemia and four times more likely to die of pneumonia as compared with individuals in the general population of matched age, gender and race. (Harrison-Felix, *Causes of death following 1 year postinjury among individuals with traumatic brain injury* (2006) 21 The Journal of Head Trauma Rehabilitation 1, pp. 22-33.) There are studies showing a diminished life expectancy in mTBI as well. (See, e.g., McMillan, *Mortality and morbidity 15 years after hospital admission with mild head injury: a prospective case-controlled population study* (2014) 85 Journal of Neurology, Neurosurgery and Psychiatry 11, pp. 1214-1220.)

In a quirky double-edged-sword situation, both plaintiff and defense counsel tend to steer away from these statistics.

Visual defects after TBI

In my experience, this aftereffect of a TBI on vison has been sorely overlooked. Until recently, it was not even high on my own radar, despite my studies in this very area. And that is why I have saved this consequence until last, trusting that your last impression of our discussion will remain with you as you visit with your TBI clients.

Visual impairments are a commonplace problem following a TBI. They are often serious and can lead to ongoing functional and quality-of-life problems as your clients recover and face their future.

The majority of these problems will fall into several categories. The first is damage to the globe of the eye, the retina, the choroidal structures, the optic nerve, and the associated areas of the visual cortex of the brain.

The second is ocular motor dysfunction or the ability of the eyes to move in concert. This can result from damage to cranial nerves 3, 4 or 6, which serve to move the eyes in all directions and in concert. This damage can be localized in these nerves as they exit the brainstem and run their course to their respective eye muscles, or the damage can be to the areas of the brainstem themselves where these cranial nerves arise and have connections to other brainstem pathways that function in unison with vision such as the vestibular system.

The third is defective accommodation and impaired visual perception. With imperfect accommodation, there is a problem with the correct functioning of the crystalline lens of the eye, which is responsible for focusing our vision on near or far objects and to quickly and smoothly adjust our vision as we move from near to far objects. Visual processing or perceptual disorder refers to a hindered ability to make sense of information taken in through the eyes. Difficulties with visual perception result from defective visual processing of visual information by various areas of the brain.

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As you can see, defects in vision can be multiple and arise from multiple areas of the brain, brainstem, nerve pathways entering and exiting from the brain/brainstem to the eyes and the eye structures themselves. There are a myriad of symptoms that arise from these injuries to include total or partial loss of vision, restricted peripheral vision, double vision, blurred vision, difficulty reading with loss of place, increased photosensitivity, altered color perception and many others. The cost to your visually compromised TBI client in functional deficiencies and disabilities can be considerable.

One can imagine no driving, limited occupational opportunities, family life adjustments, anxiety and depression. The list is formidable and there are several articles to assist you in this complicated endeavor. (See, e.g., Armstrong, *Visual problems associated with traumatic brain injury* (2018) 101 Clinical and Experimental Optometry 6, pp. 716-726; Jacobs, *Neuro-Ophthalmic Deficits after Head Trauma* (2013) 13 Current Neurology and Neuroscience Reports 11, p. 389.)

When reading these articles, you will notice that symptoms are sometimes clothed in ophthalmological/optometric terms that can be daunting to understand. Likewise, there are discussions of vision problems arising from TBI that only a trained person can hope to understand without sustained effort.

From my experience and related literature, the various examinations that must be performed on TBI patients with visual complaints are not routine. There are some examinations that are regularly done by ophthalmologists/optometrists such as visual acuity, lens and retina examinations and peripheral field exams. However, these are only a small part of the examination package. Fairly sophisticated testing needs to be performed by specialists uniquely trained in the diagnosis of TBI visual defects that goes beyond what the regular ophthalmologists/optometrists are accustomed to executing.

Once you have detected vision complaints in your TBI clients a referral for diagnosis and treatment needs to follow. With the expert opinions and advice, you can advocate for a reasonable plan for future compensation.

Conclusion

The aftermath of a TBI is of titanic proportions and we have just begun to plumb its depths. What has been holding us back in our pursuit of just compensation for our clients is often the absence of a medical advocate who takes all possibilities into consideration when assessing a TBI patient/client. Just remember what it is like to advocate for a client's full compensation when many of their physical and mental complaints are missing from the medical records.

It is time for a new generation of advocacy for our clients; one in which we accept that we can and should deliver the big picture. This begins with the first visit of our TBI clients to our offices and extends to the day of final resolution (and, sometimes, even after that).

Maxey Scherr was first admitted to the Texas Bar in 2009, and she founded Scherr Law Firm *in El Paso in 2022. She focuses her trial practice on catastrophic-injury cases. With so many of her cases involving TBI, Maxey has done postgraduate work in Neuroscience at Harvard University. She is licensed in Texas, New Mexico and Illinois.*