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CTE Before the Grave

A game changer for concussion litigation

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To date, chronic traumatic encephalopathy (CTE), a degenerative brain disease associated with a history of repetitive brain injuries, has only been diagnosable after death. But medical researchers believe they are on the cusp of developing a means of diagnosing CTE in living patients.

The ability to determine when CTE develops will have a profound effect on concussion litigation and claims handling. Both plaintiffs and defendants have been grappling with proving or disproving that concussions that occurred decades in the past caused CTE. The causation conundrum has also left insurers struggling to sort out which policies are triggered by CTE claims.

On the one hand, it seems logical to trigger only the policies in effect at the time of a plaintiff's exposure to head trauma. On the other hand, courts have repeatedly acknowledged that degenerative diseases caused by exposure to toxic materials can trigger policies issued long after the exposure period ended. The ability to diagnose CTE in living patients would go a long way toward answering these questions and would provide the insurance industry with some much-needed clarity on a difficult issue.

Understanding CTE

CTE occurs when tau protein in the brain forms clumps that slowly spread over time, killing brain cells.

Tau protein forms to stabilize microtubules in the brain and has been associated with brain injuries, such as from concussions or sub-concussive hits. Doctors have diagnosed CTE in patients as young as 17, but symptoms do not generally begin appearing until years after the brain is exposed to the repeated shaking or impacts. Potential symptoms of CTE include behavioral problems, dementia, and depression. While CTE is most commonly associated with retired football players and other athletes, it has also been diagnosed in military veterans, victims of domestic abuse, seizure patients, and others who have incurred repetitive head trauma.

Currently, CTE can only be diagnosed through analysis of brain tissue after death. This limitation has significantly impacted concussion litigation of all kinds in recent years. For instance, despite being the disease most widely associated with repeated head trauma, the $1 billion+ NFL concussion settlement provides zero dollars in awards for CTE diagnoses made after April 22, 2015 (the date the settlement received final approval from the district court). This is largely because the parties did not want to incentivize troubled plaintiffs to commit suicide as a means of proving a CTE diagnosis that would entitle their survivors to compensation.

Additionally, plaintiffs concussed in auto accidents, bicycling crashes, and slip and falls have filed lawsuits seeking damages for potential future CTE diagnoses. These claims could result in medical monitoring awards or higher damages than would typically be awarded for concussion claims. A plaintiff who suffers from moodiness or depression following a concussion may have a colorable basis for making a CTE claim, leading to an expensive battle of experts.

The ability to diagnose CTE while a person is alive will, therefore, reshape the landscape of CTE litigation.

In late 2017, leading CTE researchers Ann McKee and Bennet Omalu each published articles on potential breakthroughs in diagnosing CTE in living patients. Their research revealed the possible presence of CTE “biomarkers.” A biomarker is a biologically-generated substance that identifies the presence of a disease. Biomarkers can be identified through imaging (PET/CT scans, X-rays, or MRI) or molecular analysis (plasma, serum, or biopsies), and are widely used to identify cancer and heart disease.

Dr. McKee’s team found an elevated level of a protein, CCL11, in the cerebrospinal fluid of former football players diagnosed with CTE. The study also found a positive correlation between the degree of CCL11 elevation and the number of years of football played. With further research, Dr. McKee and her team are hopeful that identifying the elevated presence of CCL11 may assist in the detection of CTE in living patients.

Dr. Omalu conducted PET scans of a living football player approximately 52 months before his death. The PET scans showed abnormal tau protein deposits and other potential signs of CTE, compared with healthy controls. After the patient’s death, Dr. Omalu was able to confirm that his antemortem CTE diagnosis was correct.

The Causation Conundrum

Proving causation remains a fundamental roadblock for plaintiffs litigating CTE claims. In order for former athletes to succeed on a liability claim, they must show that CTE would not have developed but for their exposure to brain injuries in whatever organization they are suing. Further, researchers believe that CTE may be caused not only by concussions, but also by repetitive minor collisions, such as those that occur on each play of every football game. For athletes suing professional sports organizations, this means proving that their injuries were not caused by youth, high school, or collegiate sports. Given that doctors have diagnosed CTE in the brains of athletes as young as 17, it is likely that some professional athletes enter their professional careers having already developed CTE.

Non-athletes face similar challenges. Plaintiffs in slip and fall or auto accident cases must prove that the underlying accident caused CTE instead of youth athletic participation, military service, or other prior concussive or sub-concussive events.

Identifying a biomarker that indicates CTE could go a long way to solving these causation problems. Athletes would be able to undergo testing at the beginning and end of each season, which would allow for a precise determination as to when CTE began to develop, providing empirical proof to plaintiffs and defendants of a definitive exposure period. Plaintiffs will be able to definitively show not only that they are suffering from CTE, but also that a certain event or series of events caused the damage. On the other hand, defendants will have diagnostic proof to support their causation and assumption of the risk defenses.

The identification of biomarkers may also spur professional and amateur sports leagues to require testing as a prerequisite for participation. Organizations could arguably have a legal duty to exclude those with signs of CTE from further activities linked with repetitive mild traumatic brain injuries (MTBI), or at least a duty to provide the testing so that the participant can give informed consent to further participation.

Ultimately, the direction of the research on CTE and biomarkers will dictate the outcome of these unresolved questions. The explosion of concussion litigation in the 2010s quickly outpaced the development of the medical science necessary to support those claims, but that gap is closing fast.

Cutting Into Continuous Trigger

The causation conundrum has necessarily impacted insurance companies' handling of CTE claims. If insurers cannot point to a single causative event, then how can they determine what policy or policies are triggered by a CTE claim?

The majority of CTE lawsuits are pled to resemble other long-term latent injuries. Common allegations reference the "slow buildup of tau protein" in the brain and allege "latent injuries sustained by [the plaintiff] developed over time and were manifest later in life." This has led policyholders to advocate for the application of a continuous trigger, where all policies in effect from the time of exposure to the head trauma through manifestation of CTE would be triggered.

While this issue has not been litigated to conclusion in the context of CTE, courts across the country have held that a continuous trigger applies to long-tail latent and deteriorating injuries that create new injuries in subsequent policy periods. For example, see Eli Lilly and Co. v. Home Ins. Co, which concluded that "each insurer on the risk between the ingestion of diethylstilbestrol (DES) and the manifestation of a DES-related illness is liable to insured for indemnification."

At the same time, apart from toxi torts, there are few examples of a continuous trigger being applied to bodily injuries, as demonstrated in Grimsley v. Mid-Century Ins. Co., which declined to apply a continuous trigger to injuries arising from an auto accident, finding that "virtually all" of the cases applying the continuous trigger approach to bodily injury claims "involve either long latency disease processes developing from exposure or exposure to toxic materials difficult to pinpoint, and resulting in increasing or cumulative damage over time."

Should a court ultimately hold that a continuous trigger applies to CTE claims, the trigger period would run from exposure through manifestation. In determining the endpoint of the continuous trigger, courts have generally looked at the date of diagnosis (or potential diagnosis) as the time that manifestation occurs, as noted in Thomson Inc. v. Ins. Co. of N. Am.: "[M]anifestation occurs when a latent disease 'becomes clinically evident, that is, when it becomes reasonably capable of medical diagnosis.'" This means that until a widely accepted method of diagnosing CTE in living patients is developed, manifestation of CTE arguably occurs only upon death of the claimant.

It's clear why the identification of a CTE biomarker would have a drastic effect on the insurance coverage available for CTE claims. Given that CTE diagnoses have been made in the brains of deceased football

players as young as 17, some NFL rookies may begin their careers this fall already suffering from CTE. Under the existing medical science, the trigger period for such a claimant would extend until the player's death, which could be decades into the future. A biomarker capable of establishing CTE diagnoses in otherwise healthy young people could significantly abbreviate the trigger period and remove millions of dollars in insurance coverage for CTE claims.

Further, CTE biomarkers could give insurers that wish to write policies for companies in concussion-prone businesses new underwriting options. Currently, since CTE can only be diagnosed after death, insurers that want to manage the risk of CTE claims have little choice other than to exclude coverage for CTE altogether. Otherwise, concussions or MTBIs that occur during the policy period could boomerang back as CTE claims decades later.

If a market emerges for CTE coverage, then an insurer could require biomarker examinations at the beginning and end of a policy period to determine if CTE has developed during the period. Any diagnoses made after the period could be excluded, giving the insurer certainty that it will have no further obligation for CTE claims for that policy in the future. Further, biomarker tests that either show evidence of CTE or a predisposition to develop it will provide clarity that will give underwriters better pricing ideals for policyholders at risk for CTE claims, and they would eventually have historical data to show the likelihood of a diagnosis.

A Future of Clarity

The development of medical science will drastically impact the way CTE claims are litigated. Dr. Omalu believes his successful diagnosis of CTE in a living patient will lead to increased funding for a phase three clinical trial to replicate the results on a broader scale. Once funding is secured, he believes a commercial test for CTE could be available within five years.

If Dr. Omalu's projections are correct, then we will see an increase in CTE diagnoses and, in turn, an increase in CTE litigants. With a means of accurately diagnosing CTE during the plaintiffs' lifetimes, these new claims will be litigated on a drastically different playing field than the surge of CTE claims in the early 2010s.

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