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CTE in Athletes: Understanding the Long-Term Effects of Repetitive Head Injuries

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ABSTRACT
Introduction and Purpose
Chronic traumatic encephalopathy (CTE) is a progressive neurodegenerative disease linked to repeated head injuries such as concussions and sub-concussive impacts [19]. Initially observed in retired boxers with behavioral and cognitive symptoms, it was called “punch-drunk syndrome” [3,9,16]. CTE is mostly found in athletes from contact sports (e.g., football, boxing, hockey, rugby) and in military personnel [1,3,9,13,19]. While most reported cases involve male athletes, anyone exposed to repetitive head injury (RHI) is at risk [2,10,13]. Due to increasing awareness of its long-term effects on brain health, CTE is now a critical topic in sports medicine, neurology, and public health.
Material and Methods
A literature review was conducted using PubMed, focusing on studies published up to the end of 2023. Keywords included: “CTE in athletes,” “repetitive head impacts,” “tau protein,”

"encephalopathy," and "CTE." Studies were selected based on their focus on long-term effects of RHI, CTE pathology, and prevention strategies.
Results
The review highlights the connection between repeated brain trauma and CTE in athletes.
Ongoing research aims to uncover its biological mechanisms and develop early diagnostic tools and targeted treatments. Long-term studies tracking athletes are vital for identifying causes and modifiable risk factors.
Conclusions
CTE illustrates the complex link between repeated head trauma and brain health decline. While progress has been made, diagnosis, treatment, and prevention remain difficult. Continued research and collaboration among clinicians, researchers, sports organizations, and policymakers are essential to reduce risks and protect athlete well-being.

Keywords: CTE in athletes, repetitive head impacts, tau protein, encephalopathy, traumatic brain injury

Pathophysiology and mechanisms

CTE is characterized by the accumulation of abnormal tau protein within the brain, which forms neurofibrillary tangles (NFTs) and eventually causes widespread neurodegeneration. NFTs disrupt microtubule stability and impair intracellular transport, leading to neuronal dysfunction and death. [1, 13,21,22,23]

Repeated head trauma (RHI), which is the main factor responsible for the development of this condition, is believed to trigger inflammatory responses and axonal injury, leading to tauopathy. [15] Neuroinflammation, in which the key features are released cytokines, is closely linked to other neurodegenerative diseases and contributes to their pathophysiology and progression.

[5,15] RHI, apart from being responsible for a cascade of biochemical changes, leads to cellular and structural ones in the cerebrum as well. [17] The force of the potential blow doesn't have to be significant to cause serious damage to the brain since even mild, repeated impacts lead to collective damage to the brain. [7,22] Repeated trauma is responsible for alteration in the blood- brain barrier and its dysfunction, allowing entry of neurotoxic substances, e.g., plasma proteins and immune cells, into the brain parenchyma. [4,6, 13] This occurrence worsens inflammation and oxidative stress, creating a vicious cycle of neuronal injury; causing axonal shearing or tearing. [21] Additionally, mechanical forces may stretch and damage axons, disrupting the cytoskeleton and leading to impaired axonal transport and eventual degeneration. [1,21]

While studying the brain of the deceased athletes, we could observe pathological findings such as atrophy of the cerebral hemispheres, medial temporal lobe, thalamus, mammillary bodies, and brainstem, with ventricular dilatation and a fenestrated cavum septum pellucidum. [1,13,21,22,23] The previously characterized changes are the results of pathognomonic lesions caused by perivascular accumulation of neuronal phosphorylated tau (p-tau) in cortical and subcortical areas of the brain. [1, 13,21,22,23] These cerebral regions are responsible for

memory, emotion regulation, and executive function, which explains the diverse manifestations of the disease.

Clinical presentation

The overall clinical image of CTE manifests itself with a complex and broad spectrum of cognitive, behavioral, mood, and motor disturbances. [19] The symptoms vary depending on the stage of the disease. Mostly, the cognitive manifestation comes before the rest of the disturbances. Chronic traumatic encephalopathy progresses through distinct stages, initially presenting with subtle symptoms and advancing to severe neurodegeneration. In the tables below is a detailed look at the symptoms and staging of CTE.

Table 1. Symptoms of CTE [1,3,4,6,7,11,13,14,15,18,19 21,22]

Cognitive Symptoms	**Behavioral Symptoms**	**Mood Symptoms**	**Motor Symptoms***
Memory impairment	npulsivity and disinhibition	Depression (often treatment-resistant)	Parkinsonism
Executive dysfunction	Increased aggression or violent tendencies	ety or panic attacks	Dysarthria
Impaired judgment and decision-making	ial withdrawal and apathy	idal ideation or behaviors	it instability and frequent falls
anguage difficulties in advanced stages		motional instability and mood swings	Spasticity or coordination issues

*Usually in advanced stages

Table 2. Staging of CTE* [11, 13, 14, 15, 22]

Stage I	**Stage II**	**Stage III**	**Stage IV**
Mild headaches	Persistent headaches	ificant memory impairment	Profund cognitive deficits, including dementia
	Early signs of depression, anxiety or mood wings	r executive function	notional lability or flattened affect
Difficulty concentrating or maintaining attention	Memory lapses and increased forgetfulness	Severe mood disorders, including severe depression and suicidal ideation	Severe behavioral changes (e.g. extreme aggression, paranoia)

Subtle mood changes, such as mild irritability or sadness	Behavioral changes, including impulsivity or mild aggression	ısed aggression or apathy	Severe motor symptoms: parkinsonism, gait abnormalities
		Early motor symptoms: tremors or unsteady gait	Complete dependence on caregivers

* Based on McKee et al., 2013.

CTE and its stages differ in the advancement of symptoms, severity, and pathology. The pathology of stage I is isolated foci of tau deposition in the depths of cortical sulci. [11, 13,15, 14, 22] Stage II develops due to more widespread tau accumulation, particularly in the frontal and emporal lobes. [11, 13, 14,15, 22] In stage III we can observe advanced tau deposition with neuronal loss and gliosis, additionally affecting the hippocampus and amygdala. [11, 13, 14,15, 22] Finally, stage IV is the most severe tauopathy with extensive brain atrophy, widespread gliosis, and loss of white matter. [11, 13, 14,15, 22] Symptoms typically develop years to decades after the initial head trauma, often in the 30s or 40s, although there is some variability as to when exactly it occurs. [9] Some individuals progress slowly over decades, while others' health declines rapidly. Understanding the staging and symptomatology of CTE is crucial for early identification, intervention, and management of this condition.

Diagnosis and detection

Currently, CTE cannot be definitively diagnosed during life; the diagnosis is made only through postmortem neuropathological examination, identifying characteristic tau protein deposits in the specific brain regions. In a 2013 study, on mostly male athletes with a history of RHI from contact sports, conducted by Mckee a distinctive pattern of p-tau pathology was recognized.

These findings were considered diagnostic for CTE, which led to proposing the McKee criteria for the pathological diagnosis of chronic traumatic encephalopathy. The diagnosis of CTE required (1) the presence of perivascular foci of p-tau NFTs and astrocytic tangles; (2) an irregular cortical distribution of p-tau NFTs and astrocytic tangles with a predilection for the depth of cerebral sulci; (3) clusters of subpial and perivascular astrocytic tangles in the cerebral cortex, diencephalon, basal ganglia, and brainstem; and (4) NFTs in the cerebral cortex located preferentially in the superficial layers. [13]

In living individuals, the condition remains a clinical challenge due to the lack of definitive diagnostic tests. The process of identifying CTE could also be handicapped by the overlap with other neurodegenerative diseases with potentially similar findings. Conditions that could overshadow the recognition of CTE are, e.g., Alzheimer’s disease, Parkinsonism, or dementia. [1,3,4, 19,23]

As of now, without postmortem evaluation, we can only diagnose athletes with traumatic encephalopathy syndrome (TES). TES is the clinical manifestation of chronic traumatic encephalopathy. [1,5] The primary characteristic of TES is neurobehavioral dysregulation (NBD), which involves difficulty in controlling emotions and behaviors, commonly seen in individuals who have experienced repeated head injuries (RHI).[1,5] Research to better understand biological correlates with manifestations of TES was carried out with a focus on the dependency of inflammatory biomarkers in CSF and symptoms of the condition in

athletes.[1,5] The results of the study showed associations between elevated levels of CSF IL-6 and plasma NfL with certain symptoms of neurobehavioral dysregulation in former American football players.[5] Elevated levels of CSF IL-6 were significantly associated with higher emotional dyscontrol, affective lability, impulsivity, and total NBD scores.[5] Plasma NfL was associated with higher emotional dyscontrol and impulsivity but also with worse executive function and processing speed. [5] Another independent study suggests that the selected biomarkers for CTE do not change significantly during a shorter period. [9] However, when observed consistently over an extended time, they might indicate the development of CTE. [9] The role and directionality of these findings must be further investigated to unravel the exact link between them. The promising way of diagnosing CTE in living individuals is emerging imaging techniques (e.g., PET scans targeting tau proteins). [7]

Risk factors and susceptibility

The development of CTE is influenced by a combination of external risk factors (e.g., repetitive head trauma) and intrinsic susceptibility factors (e.g. genetics, individual resilience). In 2022, there was the first ever diagnosis of chronic traumatic encephalopathy in female professional footballer Heather Anderson. [2] The increasing popularity of contact sports amongst women suggest that although Australian athlete was the primary case of female professional diagnosed with CTE, she likely will not be the last. The disease is spreading to not only greater but younger groups of people being potentially at risk. The young age of onset of participation in contact sports prolongates the time of exposure to repetitive head trauma. [20] Additionally, immaturity of brain in individuals increases their vulnerability due to ongoing brain development. The unfortune case of Heather Anderson shows how prior concussions, age at first exposure to contact sports and cumulative head impacts increases the CTE risks. Australian player was just in her late twenties at the time of her death, but she already had 18 years of experience as avid footballer. [20] She played simultaneously for both football and rugby league for approximately 2 years. She was forced to retire after one professional season due to musculoskeletal injury. She had suffered one diagnosed concussion, with four other possible concussions not formally diagnosed but suspected by family. The postmortem examination of her brain did in fact fulfill current diagnostic criteria for low-stage CTE. [2]

The intrinsic susceptibility factors may include genetic predispositions such as variants in the APOE Σ4 allele. The different variations of this gene may increase one's chances of greater tau accumulation and neurodegeneration.[8] APOEε4, which codes for the primary cholesterol transporter in the brain, confers the greatest genetic risk for sporadic Alzheimer disease (AD), and has been implicated as a risk factor for poor recovery after traumatic brain injury (TBI) and following exposure to contact sports.[8,19] Understanding genetic foundation of CTE may provide better insights into disease mechanism and offer ways to prevent or better handle the developed condition.

Studies have shown that there are significant differences in risk across sports with American football players having one of the highest likelihoods of developing CTE. In one of her newest research, Mckee discovered that among 63 brain donors diagnosed with chronic traumatic encephalopathy 48 (76.2%) of them played football both professionally and at amateur level. Participants of the study were also wrestlers, icehockey, soccer and rugby players. [6]

Most individuals who suffered from CTE consisting of 45 (71.4%) brain donors, were amateur athletes. [6] The gender role is hard to establish due to the fact of lower number of

female donors at the time of research, but we can definitively argue that females were younger age at death and had fewer years of contact sport exposure than the male donors.

Table 3. Summary of relative risks [6,19,21]

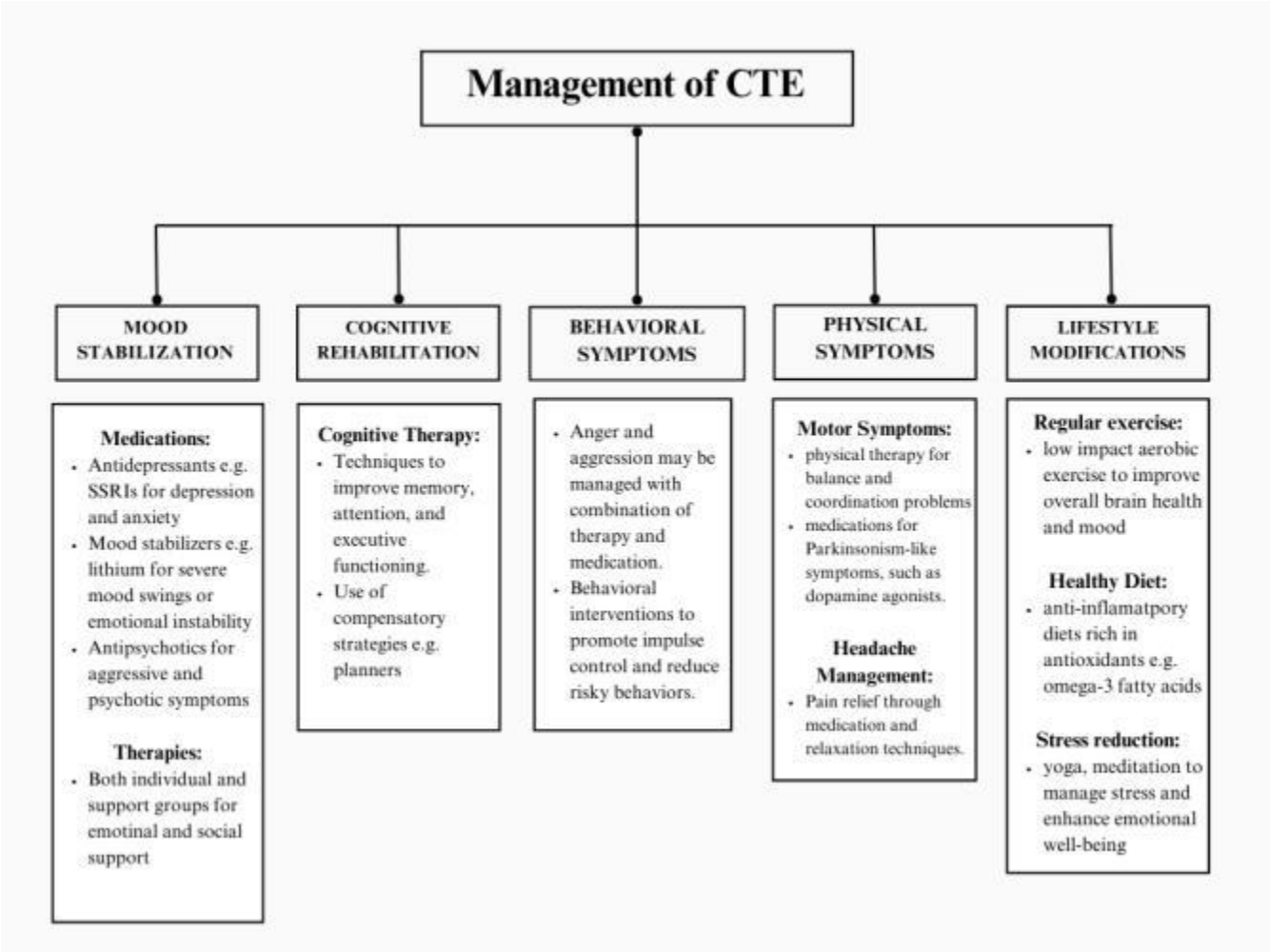
Sport	**Risk of CTE**	**Key Contributing Factors**
American football	High	t impacts, career duration
Boxing	High	Direct head blows, knockouts
Icehockey	High	ions, fights, fast gameplay
Rugby	High	g, scrums, lack of headgear
Soccer	Moderate	Heading, aerial collisions
Wrestling	Moderate	Head slams, repeated trauma
Lacrosse	Moderate	Body checks, stick impacts
Baseball/Softball	Low	al collisions, hit-by- ball injuries
Non-Contact Sports	Very Low	Minimal or no head impacts

Prevention and management

Preventing and managing CTE involves a multidimensional approach that first and foremost focuses on raising public awareness of this condition. [18] Providing education on how to reduce exposure to head trauma by safe practice techniques like e.g. safe tackling and hitting could have measurable benefits. [11] Additionally ensuring training programs on recognizing

concussion symptoms for both parents and coaches could speedup the proper management of injured
players. [18,21] The rule changes such as penalizing hits to the head, applying age limit to full- contact practices or reducing fight allowances in hockey could reduce the risk of developing CTE. Another method of prevention could be introducing players to advanced equipment including helmets with improved shock absorption materials, such as multi-layered foam or gel inserts. Protective headgear should be optimized for the unique demands of each sport like football, hockey, or cycling to minimize sport-specific injuries. Unfortunately, helmets have their limitations as they can reduce skull fractures and severe head trauma but may not prevent sub-concussive impacts or the rotational forces that contribute to CTE. For individuals with suspected or early CTE, management focuses on alleviating symptoms and improving quality of life, as there is no cure for the condition. Depending on the degree of advancement in manifestation of chronic traumatic encephalopathy and the area it affects we can focus on either mood stabilization, cognitive rehabilitation or management of behavioral and physical symptoms. The diagram below shows the possible ways of dealing with CTE.

Diagram 1. Management of symptoms in chronic traumatic encephalopathy [25]



In summary, it's important to note that prevention is the key to reducing the incidence of CTE, with rule changes, advanced protective equipment, and education playing critical roles. For at-risk individuals, early detection and comprehensive management strategies, including medication, therapy, and lifestyle changes, are essential to reduce the impact of the disease and improve quality of life.

Ethical and social implications

Chronic traumatic encephalopathy presents profound ethical and social challenges, affecting not only people diagnosed with the condition but also their families, caregivers, sports organizations, and society at large. The impact on the close ones of individuals suffering from CTE is multidimensional and creates both financial and physiological difficulties. Families experience the unpredictability of symptoms and witness drastic changes in their loved ones, including aggression, mood swings, depression and memory loss leading to significant emotional distress. For late-stage CTE patients, the progressive decline can feel like a prolonged grieving process as families lose the personality of the person they used to know. Long term care, including therapy medication and assisted living can impose a heavy financial burden becoming a challenge on caregivers who often end up sacrificing careers and personal well-being. Additionally, the stigma associated with the behavioral symptoms of CTE may cause families to feel socially isolated.

The lack of understanding or support networks for dealing with CTE-specific challenges can further alienate them in society. Consequently, organizations such as the Concussion Legacy Foundation (CFL) and Boston University's CTE Center play crucial roles in promoting awareness about CTE and risks as well as helping families affected by this condition through resources and peer networks. Public messaging despite having the best interest at heart makes balance between awareness and fear difficult. Efforts to draw attention to the issue of CTE may inadvertently discourage sports participation, leading to reduced physical activity among youth. Parental concerns and heightened anxiety are raising questions whether the risks outweigh the benefits of sports for their children. Another matter is the ethical responsibility of sports organizations such as e.g. The National Football League (NFL). NFL is a highly popular sports league in the United States that attracts great number of athletes due to its promise of fame and well-paid position. [12] Perusing a career in the NFL comes with significant health risks due to exposure to RHI and its long-term effects. Despite clear scientific evidence, NFL and other sport organizations have historically downplayed or ignored risks associated with head trauma, delaying crucial safety reforms.[12] Ethical concerns arise when the financial interests of professional leagues appear to conflict with prioritizing players' health. Moving forward, the NFL must recognize the connection between concussions and CTE, invest in new diagnostic technologies for the early detection of CTE in active players, and consider possible treatment options. [12] By taking a unified and open approach, the NFL can work towards protecting the health and futures of its athletes while tackling the serious impacts of head injuries and promoting a safer playing environment. [12]

Future directions in CTE research

Future research directions in CTE aim to fill critical gaps in knowledge about disease onset, progression, and management. Longitudinal studies, coupled with advancements in neuroimaging and biomarkers, are expected to revolutionize diagnostic capabilities. [19,23,24] Simultaneously, emerging therapeutic strategies targeting tau pathology, neuroinflammation, and neuroprotection offer hope for effective treatments. Collaboration across disciplines will

be essential to realize these advancements and reduce the burden of CTE on athletes and society.

Summary
Understanding the pathology of chronic traumatic encephalopathy and its long-term effects is crucial to finding ways to prevent its development and reduce the negative impact of the disease on individuals who suffer from it. The review explores the mechanisms behind CTE, its relationship with RHI, risk factors, as well as the challenges of diagnosing the disease, as it can
only be confirmed post-mortem. Extensive clinicopathological studies involving both current and former athletes are essential to gain a clearer understanding of CTE and to determine its true prevalence and associated risk factors. [4,19]

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