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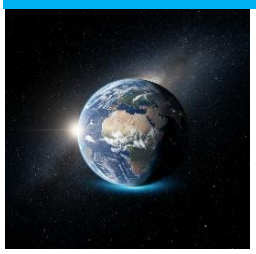

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Neuropathological Landscape of Repetitive Brain Trauma and CTE in Modern Sport

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ABSTRACT

Background: Repetitive brain trauma has become a significant problem in modern sport, particularly in athletes exposed to repeated head impacts. Such injuries have been associated with chronic traumatic encephalopathy (CTE), a progressive neurodegenerative disease characterized by abnormal accumulation of hyperphosphorylated tau protein and other neuropathological changes.

Aim: The aim of this study was to review current scientific knowledge on the neuropathological consequences of repetitive brain trauma in athletes, with particular emphasis on chronic traumatic encephalopathy.

Material and methods: This study was conducted as a literature review. Relevant research articles related to repetitive brain injury and chronic traumatic encephalopathy (CTE) were identified through a PubMed database search using the following keywords: chronic traumatic encephalopathy, repetitive brain trauma, traumatic brain injury, neuropathology, and contact sports. The selected publications were analyzed to summarize the current state of knowledge on the neuropathology of CTE.

Results: The reviewed studies indicate that repetitive head impacts can lead to progressive neurodegenerative changes, including abnormal tau protein deposition, neuroinflammation, neuronal loss, and axonal damage. These neuropathological alterations are considered key features of chronic traumatic encephalopathy.

Conclusions: Repetitive brain trauma may significantly contribute to the development of chronic traumatic encephalopathy. Further research is needed to improve early diagnosis, prevention strategies, and clinical treatment of athletes exposed to repeated head impacts.

Key words: chronic traumatic encephalopathy, repetitive brain trauma, traumatic brain injury, neuropathology, contact sports

Contents

1. Introduction

Repetitive brain trauma has become a significant problem in modern contact sports due to its potential long-term neurological effects. Athletes exposed to repeated head impacts are at increased risk of developing chronic traumatic encephalopathy (CTE), a progressive neurodegenerative disease associated with abnormal tau protein accumulation [17,13].

In recent years, increasing attention has been given to the long-term consequences of both concussive and subconcussive injuries. Even impacts that do not result in clinically diagnosed concussion may contribute to cumulative brain damage over time. As a result, concerns have grown regarding the neurological health of athletes participating in contact sports such as football, boxing, and hockey [17]. These concerns have led to increased interest in understanding the long-term neurological consequences of repeated head trauma in both professional and amateur athletes.

Repetitive brain trauma is associated with a range of pathological processes, including neuroinflammation, axonal damage, and gradual neuronal loss, which can contribute to cognitive and behavioral impairment over time [18,5]. These changes may develop silently and remain asymptomatic for many years, only becoming clinically apparent later in life.

Moreover, the complexity of these mechanisms suggests that CTE is not caused by a single factor, but rather by the interaction of multiple pathological processes. This highlights the importance of understanding both the biological and mechanical aspects of brain injury. Given the growing evidence on the long-term consequences of repetitive brain trauma, there is a need to systematically summarize current findings regarding its impact on neuropathology, which constitutes the aim of this study [13,14,17]. A comprehensive understanding of these mechanisms is essential for advancing research, improving diagnostic criteria, and developing effective preventive strategies in modern sport [14].

2. Materials and methods

This study was conducted as a narrative literature review focusing on the neuropathological consequences of repetitive brain trauma. A comprehensive search of the PubMed database was

performed to identify relevant peer-reviewed literature published between 2006 and 2026. This timeframe was selected to capture both foundational criteria for CTE and the most recent clinical advances. The following search terms and their combinations were utilized: "chronic traumatic encephalopathy", "repetitive brain trauma", "traumatic brain injury", "neuropathology", and "contact sports".

Inclusion and Exclusion Criteria:

Inclusion: Only original research articles, clinical case reports, and comprehensive reviews available as full-text were included.

Exclusion: Studies focusing on single-event traumatic brain injuries (TBI) without a history of repetitive impacts, as well as articles without accessible full-text or those published in languages other than English or Polish, were excluded.

Selected articles were analyzed to summarize data on tau pathology, neuroinflammation, and clinical disease progression.

3. Epidemiology and Risk Factors

Repetitive head impacts are a major risk factor for the development of chronic traumatic encephalopathy (CTE), particularly in contact sports. Athletes participating in sports such as American football, boxing, ice hockey, and soccer are at risk of both concussive and subconcussive injuries, which can accumulate over time and contribute to long-term neurological consequences [17]. The risk of such consequences depends not only on the severity of the individual injuries but also on the cumulative number of head impacts sustained during an athlete's career.

Epidemiological studies based on brain bank analyses have shown that CTE occurs in people with a documented history of repetitive brain trauma, supporting a clear association between exposure to head impacts and the development of neurodegenerative changes [5]. Importantly, these findings suggest that even in the absence of clinically diagnosed concussions, repeated subconcussive impacts may still contribute to disease progression.

Recent research indicates that cumulative exposure is a key risk factor, with longer duration and higher intensity of sporting activity increasing the likelihood of developing CTE-related

pathology [14,4]. Additional factors, such as genetic susceptibility and inadequate recovery between injuries, may further influence the risk and progression of the disease.

Current evidence indicates that repetitive brain trauma is a significant and multifactorial risk factor for CTE, making it crucial to monitor exposure and implement preventive strategies in modern sport.

Although CTE is most commonly associated with contact sports, it has also been described in individuals with a history of repetitive head trauma outside of sports, such as military personnel or victims of repeated injuries. This suggests that the underlying risk is related to cumulative brain trauma rather than the specific type of activity [13,14].

3.1 Sports at Risk

Some sports are associated with a higher risk of repeated head impacts due to their intensity and the nature of play. Contact sports such as American football, boxing, and ice hockey involve frequent physical contact, which increases the likelihood of head injuries. In these sports, athletes are often exposed to repeated impacts over many years. Even when these impacts do not lead to a diagnosed concussion, they may still contribute to gradual changes in the brain.

Interestingly, concerns have also been raised in sports like soccer, where actions such as heading the ball are repeated regularly. Although each individual impact may seem minor, their cumulative effect over time may still be significant.

4. Neuropathology of CTE

Chronic traumatic encephalopathy (CTE) is a progressive neurodegenerative disease associated with repetitive brain trauma and characterized by distinct neuropathological features. The characteristic lesion in CTE is the abnormal accumulation of hyperphosphorylated tau protein in a specific pattern, which distinguishes it from other neurodegenerative diseases [13,14]. Recent criteria emphasize that the presence of perivascular tau aggregates, particularly deep in the cortical sulci, is essential for the neuropathological diagnosis of CTE [14].

In advanced stages of the disease, macroscopic changes such as cortical atrophy, ventricular enlargement, and reduced brain weight can also be observed [9,14]. These findings reflect the progressive nature of CTE and correspond to widespread structural degeneration.

4.1 Tau Pathology

Tau pathology is a central feature of CTE. The accumulation of hyperphosphorylated tau occurs in both neurons and astrocytes, leading to the formation of neurofibrillary tangles and astrocytic inclusions [13]. Unlike other tauopathies, tau distribution in CTE is irregular and focal, often occurring in a perivascular pattern. Different tau isoforms may also contribute to disease progression, suggesting a complex and evolving pathological process [8].

Additionally, abnormal tau may spread between connected brain regions, which can contribute to the gradual progression of the disease. This process may help explain why symptoms tend to worsen over time.

4.2 Localization of Neuropathological Changes

The distribution of pathological changes in CTE follows a characteristic pattern. Tau deposits are predominantly found in the superficial cortical layers and are concentrated at the depths of sulci, particularly in frontal and temporal regions [13,14].

This pattern is not random and is thought to be related to the mechanical forces acting on the brain during head impacts, which tend to affect these regions more strongly. As a result, these areas may be more vulnerable to the development of pathological changes.

In more advanced stages, pathology may extend to additional brain structures, including the hippocampus, amygdala, and brainstem. The involvement of these regions may help explain the progression of symptoms, particularly those related to memory, emotions, and behavior. This distinct topographical pattern is considered one of the defining features that differentiate CTE from other neurodegenerative diseases and plays an important role in its neuropathological diagnosis.

4.3 Disease Progression and Staging

CTE is a progressive disorder, with neuropathological changes evolving over time. Early stages are characterized by focal perivascular tau deposition, whereas later stages demonstrate widespread involvement of cortical and subcortical regions [9,14,13].

As the disease progresses, these pathological changes extend to larger areas of the brain, affecting regions responsible for cognition, behavior, and emotional regulation [3,14]. This gradual spread helps explain the increasing severity of clinical symptoms observed over time.

Neuronal loss, gliosis, and brain atrophy become increasingly pronounced in advanced stages of the disease [4,14]. These structural changes are associated with impaired brain function and reduced neural connectivity.

The staging system proposed in neuropathological studies reflects the extent and distribution of tau pathology and correlates with the severity of clinical symptoms [14,13]. It provides a useful framework for understanding disease progression and linking neuropathological findings with clinical presentation.

4.4 Differences Between CTE and Alzheimer's Disease

Although both CTE and Alzheimer's disease are associated with tau pathology, their patterns of distribution differ significantly. In CTE, tau deposition typically occurs in a perivascular pattern and is concentrated at the depths of cortical sulci, whereas in Alzheimer's disease it follows a more uniform and predictable distribution across cortical regions [13].

Additionally, beta-amyloid plaques, which are a hallmark of Alzheimer's disease, may also be present in some cases of CTE but are not required for diagnosis [19]. This highlights an important difference in the underlying pathology of the two conditions.

Another key distinction lies in their causes. CTE is strongly associated with a history of repetitive head trauma, while Alzheimer's disease is primarily linked to age-related neurodegenerative processes. These differences are also reflected in clinical presentation, as CTE more often involves early behavioral and mood changes, whereas Alzheimer's disease typically begins with memory impairment.

Table 1. Key Neuropathological and Clinical Differences Between CTE and Alzheimer's Disease

Feature	CTE	Alzheimer's Disease
Primary Etiology	History of repetitive head trauma (concussive and subconcussive).	Primarily linked to age-related neurodegenerative processes.
Tau Distribution	Focal, perivascular, and concentrated at the depths of cortical sulci.	Uniform and predictable distribution across cortical regions.
Beta-amyloid Plaques	May be present in some cases but are not required for diagnosis.	A hallmark feature essential for diagnosis.
Symptoms	Early behavioral and mood changes (e.g., irritability, impulsivity).	Typically begins with short-term memory impairment.
Pathological Focus	Predominantly superficial cortical layers in early stages.	Widespread involvement of cortical and subcortical structures.

5. Pathophysiological Mechanisms

Chronic traumatic encephalopathy (CTE) develops as a result of multiple pathological processes triggered by repetitive brain injury. Mechanical forces acting on the brain initiate a cascade of changes, including axonal damage, abnormal protein accumulation, and neuroinflammation.

In addition, repetitive trauma can disrupt the blood–brain barrier, allowing potentially harmful substances to enter brain tissue and further contribute to inflammation. Oxidative stress may also play a role by increasing cellular vulnerability and promoting neuronal damage.

5.1 Mechanical Injury and Axonal Damage

Repetitive head impacts expose the brain to mechanical forces that can lead to diffuse axonal injury, which is considered one of the earliest pathological events in CTE. These forces may stretch and shear neuronal axons, disrupting axonal transport and impairing communication between neurons [18].

As a result, neurons become more vulnerable to further damage and may gradually lose their functional integrity. Even relatively mild but repeated impacts can accumulate over time, leading to subtle but persistent structural changes in brain tissue.

In addition, repeated injury may impair the brain's ability to repair damaged axons, which further contributes to long-term dysfunction. Over time, these cumulative changes can disrupt neural networks and play an important role in the development and progression of neurodegeneration.

5.2 Tau Pathology and Protein Aggregation

Following mechanical injury, a range of abnormal biochemical processes may lead to the accumulation of hyperphosphorylated tau protein. This pathological tau can misfold and aggregate within neurons and glial cells, forming neurofibrillary tangles and contributing to cellular dysfunction [8,13].

These changes interfere with the normal function of neurons, particularly by disrupting intracellular transport and communication between cells. As a result, affected neurons gradually lose their ability to function properly, which contributes to progressive brain damage.

The spread of tau pathology is thought to occur through interconnected neural networks, suggesting a prion-like mechanism of propagation. Over time, this process may involve larger areas of the brain, which helps explain the gradual worsening of clinical symptoms.

5.3 Neuroinflammation

Neuroinflammation is another important mechanism involved in the development of CTE. Repetitive brain trauma can lead to chronic activation of microglia and astrocytes, resulting in the release of pro-inflammatory mediators and a sustained inflammatory response [6,4]. Although acute inflammation may initially have a protective role, prolonged activation of immune cells can lead to neuronal damage and further promote neurodegeneration.

Over time, this persistent inflammatory state may disrupt normal brain function and interfere with neuronal communication. Activated microglia can release substances that are harmful to surrounding neurons, which may further accelerate tissue damage. In addition, repetitive injury

may impair the brain's ability to regulate inflammation effectively, leading to a cycle of ongoing damage.

5.4 Disease Progression

The interaction between mechanical trauma, tau pathology, and neuroinflammation leads to progressive neurodegeneration over time. These processes contribute to neuronal loss, synaptic dysfunction, and brain atrophy, which are characteristic features of advanced CTE [4,14].

As these changes accumulate, they gradually affect different regions of the brain, leading to worsening cognitive and behavioral symptoms. The progressive nature of the disease reflects the ongoing interaction between structural damage and biological responses within the brain.

This condition is multifactorial and influenced not only by mechanical and biological factors, but also by individual susceptibility, including genetic predisposition and differences in recovery [4]. The extent of exposure to repetitive head impacts, as well as the time allowed for recovery between injuries, may further influence the severity and progression of the disease.

Not all individuals exposed to repetitive head trauma develop CTE, which suggests that additional factors may influence disease susceptibility [3,4,14].

6. Clinical Manifestations and Diagnosis

The clinical manifestations of CTE are varied and often develop gradually over time. In the early stages, patients may experience subtle changes in mood and behavior, including irritability, anxiety, and depressive symptoms [3,13]. These early symptoms are often nonspecific and may be overlooked or attributed to other conditions, making early diagnosis challenging. A characteristic feature of CTE is the long latency period between exposure to head trauma and the onset of clinical symptoms, which may span several years or even decades [13,14]. As the disease progresses, behavioral and emotional symptoms become more noticeable. Individuals may show increased impulsivity, aggression, and emotional instability [3,5]. Depression is common, and in some cases may be linked to suicidal thoughts [5]. These changes can significantly affect daily functioning and social relationships.

Cognitive decline is another important feature of CTE. Patients may experience problems with memory, attention, and executive functions such as planning and decision-making [13,14]. These difficulties tend to worsen over time and may resemble those seen in other neurodegenerative conditions [14]. In advanced stages, CTE is associated with severe cognitive impairment, including dementia, along with persistent behavioral symptoms [14]. This leads to a significant decline in daily functioning and may require long-term care.

Diagnosis of CTE remains a major challenge in clinical practice. At present, a definitive diagnosis can only be made postmortem through neuropathological examination of brain tissue [13,14].

Ongoing research focuses on the development of reliable diagnostic tools, including biomarkers and advanced imaging techniques [14]. Positron emission tomography (PET) targeting tau protein has shown potential in detecting pathological changes, although it is not yet widely used in clinical practice.

Identifying biomarkers in blood or cerebrospinal fluid may also improve early detection in the future [14]. Despite these advances, there is currently no universally accepted method for diagnosing CTE during life.

Currently, there is no specific treatment for CTE, and management is limited to symptomatic care. This further emphasizes the importance of prevention and early risk reduction strategies.

7. Prevention

Prevention of CTE mainly focuses on reducing repeated head impacts, especially in contact sports. This can be achieved by improving safety measures, such as using protective equipment, introducing rule changes, and promoting safer playing techniques.

Increasing awareness among athletes, coaches, and medical staff is also essential for early recognition and proper management of head injuries. Allowing enough time for recovery after concussions and limiting further exposure to head impacts may help reduce the risk of long-term neurological damage.

In recent years, increasing emphasis has also been placed on the implementation of return-to-play protocols, which aim to ensure that athletes do not resume activity before full recovery. These guidelines are particularly important in preventing repeated injuries within a short period of time.

Special attention should also be given to youth sports, where developing brains may be more vulnerable to injury. Early education and proper coaching strategies may play a key role in reducing long-term risk.

8. Conclusion

Chronic traumatic encephalopathy (CTE) is a progressive neurodegenerative condition closely linked to repetitive head trauma, particularly in contact sports. In this review, we summarized the current knowledge on its neuropathology, clinical presentation, and underlying mechanisms.

The development of CTE is associated with processes such as tau pathology, neuroinflammation, and ongoing neurodegeneration, which together contribute to structural and functional changes in the brain. These mechanisms interact and evolve over time, leading to gradual damage of neural networks. Clinically, the disease may present with a wide range of symptoms, including cognitive decline, behavioral changes, and mood disturbances that tend to worsen as the disease progresses.

Despite growing awareness, CTE remains difficult to diagnose during life, as a definitive diagnosis is still based on postmortem neuropathological examination. This highlights an important gap in current clinical practice and underlines the need for more reliable diagnostic tools that could be used earlier in the disease course.

Reducing exposure to repetitive head trauma and improving awareness of head injuries are essential steps in prevention. Proper management of concussions, including sufficient recovery time, may also play an important role in limiting long-term neurological consequences.

Further research is needed to better understand the mechanisms underlying CTE, as well as the factors that influence its progression and clinical variability. In particular, the development of

biomarkers and advanced imaging methods may improve early detection and support more effective prevention strategies in the future.

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Author's contribution

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Methodology: AP, MO;

Software: n/a;

Validation: JK, MS, AW;

Formal analysis: KCG, AK;

Investigation: AP, AŻ, MO, KT;

Resources: AP, MS;

Data curation: AP, MO, KCG, AK, JF, AK1, KT;

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