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REVIEW ARTICLE

# CAN HEADING A BALL CAUSE DEMENTIA? THE RELATIONSHIP BETWEEN TBI AND NEURODEGENERATIVE DISEASES

## CZY GŁÓWKOWANIE MOŻE POWODOWAĆ DEMENCJĘ? ZWIĄZEK URAZOWEGO USZKODZENIA MÓZGU Z CHOROBAMI NEURODEGENERACYJNYMI

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ABSTRACT

**Introduction**

The link between contact sports and neurodegenerative diseases has become an increasingly prominent topic of scientific and public interest. Repetitive head impacts (RHIs) and traumatic brain injuries (TBIs) are associated with long-term neurological consequences. This has raised concerns among athletes, sports organizations, and medical professionals about the safety of contact sports.

**Aim**

This narrative review provides an overview of recent advances in understanding the relationship between TBI and neurodegenerative diseases in sports.

**Materials and methods**

A PubMed search was conducted for articles published within the last five years using the following keywords: football, soccer, heading, Alzheimer's disease, TBI, and RHI.

**Results**

Contact sports are linked to an increased risk of neurodegenerative diseases. Among former soccer players, the risk can be increased threefold. Each additional year of rugby play raises chronic traumatic encephalopathy (CTE) risk by approximately 14%. In American football, CTE can develop in players younger than 30. While CTE remains the most prevalent neurodegenerative outcome, other complications manifest as clinical depression, insomnia, and amyotrophic lateral sclerosis. Diagnostic methods involve biochemical analyses of serum and cerebrospinal fluid, histopathology, messenger RNA profiling, and neuroimaging techniques. Currently, no disease-modifying treatment for CTE exists. The literature identifies various therapeutic and supportive interventions, including the administration of neuroprotective agents, low-intensity pulsed ultrasound, and photobiomodulation, alongside the implementation of rehabilitation protocols.

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**Conclusions**
Repetitive TBI in athletes is associated with neurodegeneration. Key challenges include insufficient diagnostic standardization and lack of effective cytoprotective therapies, highlighting the need for further research.

**Keywords:** soccer, football, heading, TBI, RHI, neurodegenerative disease

STRESZCZENIE
**Wprowadzenie**
Związek między sportami kontaktowymi a chorobami neurodegeneracyjnymi staje się coraz bardziej popularnym tematem wśród naukowców i w debacie publicznej. Powtarzające się urazy głowy (RHI) oraz pourazowe uszkodzenia mózgu (TBI) wiążą się z długoterminowymi konsekwencjami neurologicznymi. Fakt ten budzi obawy wśród sportowców, organizacji sportowych oraz środowisk medycznych dotyczące bezpieczeństwa sportów kontaktowych.
**Cel**
Celem niniejszego przeglądu narracyjnego jest przedstawienie najnowszych osiągnięć w rozumieniu zależności między TBI a chorobami neurodegeneracyjnymi w sporcie.
**Materiały i metody**
Przeprowadzono przegląd piśmiennictwa w bazie danych PubMed, obejmujący artykuły opublikowane w ciągu ostatnich pięciu lat, z zastosowaniem następujących słów kluczowych: football, soccer, heading, Alzheimer's disease, TBI, RHI.
**Wyniki**
Sporty kontaktowe wiążą się ze zwiększonym ryzykiem chorób neurodegeneracyjnych. Wśród byłych piłkarzy ryzyko jest trzykrotnie wyższe. Każdy dodatkowy rok gry w rugby zwiększa ryzyko przewlekłej encefalopatii pourazowej (CTE) o około 14%. W futbolu amerykańskim CTE może rozwijać się u zawodników młodszych niż 30 lat. Podczas gdy CTE pozostaje najczęstszym skutkiem neurodegeneracyjnym, inne powikłania objawiają się jako depresja, bezsenność oraz stwardnienie zanikowe boczne. Metody diagnostyczne obejmują analizy biochemiczne surowicy i płynu mózgowo-rdzeniowego, histopatologię, profilowanie RNA informacyjnego oraz techniki neuroobrazowe. Obecnie nie istnieje leczenie modyfikujące przebieg CTE. Literatura wskazuje na różne interwencje terapeutyczne i wspomagające, w tym podawanie środków neuroprotekcyjnych, pulsacyjne ultradźwięki o niskiej intensywności, fotobiomodulację oraz wdrażanie protokołów rehabilitacyjnych.
**Wnioski**
Powtarzające się urazy u sportowców wiążą się z procesami neurodegeneracyjnymi. Do głównych wyzwań dotyczących tego zagadnienia należą brak standaryzacji diagnostycznej oraz brak skutecznych terapii cytoprotekcyjnych, co zaznacza potrzebę dalszych badań.

**Słowa kluczowe:** piłka nożna, football, główkowanie, TBI, RHI, choroby neurodegeneracyjne

## Introduction

Traumatic brain injury (TBI), also known as concussion, is a common neurological injury, with incidence in Europe ranging from 47.3 to 694 cases per 100,000 population per year [1,2]. The U.S. Department of Veterans Affairs and U.S. Department of Defense define mild TBI (mTBI) as a traumatically induced structural injury and/or physiological disruption of brain function resulting from an external force [2]. While diagnostic criteria were recently updated by the American Congress of Rehabilitation Medicine in 2023 to include mechanism of injury and laboratory findings [3], a significant knowledge gap persists regarding the cumulative effects of head impacts. Specifically, the relationship between repetitive head impacts (RHIs) and long-term neurodegeneration has become a prominent topic of scientific interest, as athletes and medical professionals question the safety of contact sports.

Chronic traumatic encephalopathy (CTE) is a progressive neurodegenerative disorder marked by abnormal hyperphosphorylated tau accumulation. The Second National Institute of Neurological Disorders and Stroke/National Institute of Biomedical Imaging and Bioengineering (NINDS/NIBIB) Consensus established current *post-mortem* neuropathological criteria [4], diagnosing CTE based on at least one pathognomonic lesion: perivascular neuronal tau aggregates, with or without glial tau, at cortical sulcal depths. The clinical manifestation of CTE is traumatic encephalopathy syndrome (TES). However, while the link between contact sports and CTE is increasingly established, the association with other diseases such as Alzheimer's disease (AD) remains debated [5,6].

While existing literature has examined various facets of sport-related neurodegeneration, there is a scarcity of comprehensive reviews that integrate these diverse topics into a unified framework. This review provides a multi-dimensional overview of initial risk factors, pathomorphological changes, complications, and the latest diagnostic and therapeutic developments. In addition to this synthesis, this work identifies key future research priorities, including the establishment of validated in vivo diagnostic markers and the expansion of study demographics to improve the generalizability of findings.

## Aim

this narrative review provides an overview of recent advances in understanding the relationship between TBI and neurodegenerative diseases in sports.

## Material and methods

A literature search was conducted via PubMed/MEDLINE for English-language studies published between January 2020 and October 2025. Search terms included: "football," "soccer," "heading," "Alzheimer's disease," "TBI," and "CTE." Inclusion was limited to open-access articles. A total of 82 studies were included in this review, comprising recent papers from the database search and older landmark studies identified through manual citation searching. Studies were selected based on their methodological quality and clinical relevance to the field.

## Results

### Risk factors

The prevalence of CTE varies by sport and exposure: 68% of retired rugby players were diagnosed with CTE, with each additional year of play increasing the risk by 14% [7]. Similarly, CTE was found in 54.5% of exposed former hockey players, with longer playing history increasing the risk [8]. In football, the odds of CTE correlate with duration of play and lifetime TBI exposure [9]. Some studies question the TBI–CTE association, though TBI histories were informant-reported, thereby reducing accuracy [10].

Position-dependent risk of neurodegenerative disease is also documented. In soccer, heading frequency influences cognitive decline: defenders have 3.16-fold higher odds

of neurodegenerative disease compared with goalkeepers, although this difference is not statistically significant ($p = 0.09$) [11]. Studies report an increased risk of neurodegenerative disease in professional soccer players compared to controls in Sweden (hazard ratio [HR] 1.46) and in Scotland (HR = 3.66) [12,13]. In the Scottish cohort, the risk varied by position, reaching an HR of 4.98 in defenders. Notably, while the HR for goalkeepers was 1.83, this elevation was not statistically significant ($p = 0.08$). In the Swedish cohort, players had a higher risk of AD and other dementias (HR = 1.62), but a lower risk of Parkinson's disease (HR = 0.68) [13].

While most children and adolescents recover from concussion within 4 weeks, 10–30% experience longer post-concussion symptoms [14–16]. Post-traumatic headache (PTH) is the most common persistent symptom associated with prolonged recovery [16,17]. Patients with any type of PTH following a concussion experienced significantly longer recovery times compared to those without PTH (log-rank $p < 0.001$). Furthermore, the phenotype matters: patients in the PTH migraine-type (PTH-M) group took significantly longer to recover than the PTH non-migraine-type group (median [interquartile range], 95 [54–195] days vs. 70 [46–119] days; log-rank $p = 0.01$) [18]. Although female sex is frequently identified as a predictor of prolonged recovery [15,19,20], this association appears to be mediated by headache phenotype. When data were stratified by PTH type, no significant differences in recovery time or PTH prevalence at 3 months were observed between sexes. This suggests that the prolonged recovery often attributed to female sex is driven by the higher frequency of PTH-M in females [18].

Patients with a history of emotional distress (i.e., anxiety or depression) reported a higher prevalence of persistent symptoms compared with those without ($p = 0.009$). This disparity was evident throughout the recovery trajectory: at week 4 (80.9% vs. 75.6%), week 8 (57.8% vs. 50.5%), and week 12 (48.0% vs. 33.3%). A similar pattern was observed in patients with a history of migraine ($p = 0.001$), in whom symptom persistence remained consistently elevated compared to controls at week 4 (87.3% vs. 73.9%), week 8 (67.7% vs. 49.0%), and week 12 (55.7% vs. 33.2%) [20]. Additional factors affecting symptom intensity and duration include pre-existing ADHD, learning difficulties, stress exposure, and sleep disorders [19].

Complicated mTBI is strongly linked to impaired recovery, though specific imaging phenotypes reveal distinct recovery trajectories. A cluster including cerebral contusion, subarachnoid hemorrhage, and/or subdural hematoma was associated with significantly higher odds of both incomplete recovery and unfavorable outcomes at all time points, from 2 weeks to 1 year (odds ratios [ORs] ranging from 1.67 to 3.23) [21].

### Pathomorphology

Evidence indicates that gray and white matter (WM) alterations following TBI are significantly driven by traumatic cerebrovascular injury, a major contributor to long-term neurodegeneration, including CTE. Primary mechanical disruption of cerebral endothelium after TBI breaches the blood–brain barrier (BBB), causing petechial hemorrhages, microthrombi, pericyte migration, neuroinflammation, and oxidative stress. Resulting hypoperfusion and maladaptive angiogenesis lead to chronic microbleeds, iron deposition, WM loss, and progressive neuronal degeneration [22].

Despite established *post-mortem* neuropathological criteria for CTE using the McKee Staging Schema [23], *in vivo* diagnosis remains impossible, as morphological parameters are insufficiently defined, prompting ongoing searches for *ante-mortem* markers.

A key abnormality is *cavum septum pellucidum* (CSP), presenting with septal separation, cavity enlargement, lamina fenestration, and fornix thinning or atrophy [24]. CSP appears in 65% of confirmed CTE cases.

Its prevalence is higher in former American football players (77.7%) than unexposed asymptomatic controls (47%) [25], and similarly higher in retired elite rugby athletes (61.0%) than unexposed asymptomatic controls (41.5%) [26]. Statistically significant difference in CSP length was observed between football players and low-contact sport (volleyball) controls ($p = 0.02$) [27]. Former American football players also exhibit a higher CSP ratio (CSP length/septal length) than both unexposed asymptomatic controls and former college players (25). CSP is more common in RHI-exposed individuals (44.6%) compared with AD (13.3%) and frontotemporal dementia (16.7%) [28]. Higher odds of CSP grade 2+ were observed in TES (OR = 10.9), especially with ≥11 years of exposure (OR = 3.34); combined RHI and symptomatic TBI are associated with the highest odds of having CSP [29]. Enlarged CSP associates with poorer verbal learning and memory [28,29]. Although not diagnostic, CSP may serve as a useful RHI-assessment parameter [25].

Longitudinal T1-weighted volumetric magnetic resonance imaging (MRI) differentiates CTE-typical atrophy – orbitofrontal, dorsolateral frontal, and anterior/medial temporal regions with sulcal widening – from aging or AD [30]. Sulcal widening is associated with early RHI exposure ($p = 0.03$) and cumulative football years ($p = 0.047$) [31]. Former football players exhibited significant cortical thinning ($p = 0.01$) and reduced cortical volume ($p < 0.01$) in CTE-typical areas compared to unexposed asymptomatic controls [32]. Tau positron emission tomography (PET) with 18F-flortaucipir detects frontal and temporal tau accumulation related to gray matter loss ($p = 0.006$) and memory deficits ($p = 0.27$) [33].

Neurodegeneration also includes anterior horn ventricle enlargement and thalamic notching [31,34,35]. WM and gray matter loss with increased cerebrospinal fluid (CSF) volume exceeds controls ($p < 0.001$) but remains less severe than AD ($p < 0.001$) [30].

WM abnormalities – rarefaction, loss of oligodendrocytes and myelin proteins, reactive astrocytes, and pigment-laden macrophages – are more common in RHI-exposed individuals with CTE, although no statistically significant association was found. Perivascular pigment-laden macrophages were found significantly more often in those with CTE (94.5%) vs. without (70.3%) [35,36]. WM atrophy affects precentral gyri, corticospinal tracts, superior longitudinal fasciculi, and corpus callosum [30]. Rugby players exhibited a higher prevalence of fractional anisotropy (FA) abnormalities (17%) and WM loss (50%) compared to controls (3%, 25%) [37]. *Corpus callosum* microstructure associates with RHI exposure in former National Football League (NFL) players ($p < 0.05$) [38]. No significant associations were found between WM organization and cognition up to 6 months post-mTBI, however, more than 3 years post-injury, higher FA with lower mean and radial diffusivity relates to better cognition [39].

Susceptibility-weighted imaging reveals diffuse vascular injury that potentially impacts cognition impairment [37]. Enlarged perivascular spaces, markers of impaired perivascular transport, are increased in former football players and correlate with RHI exposure ($p = 0.05$), cognitive deficits ($p = 0.04$), and executive dysfunction ($p = 0.04$) [40].

Despite extensive research, no morphological abnormality has been unequivocally linked to CTE. Current studies are limited by small sample sizes, selection bias in brain banks, and potential co-pathologies. Furthermore, interpretation is hindered by unreliable medical histories, TBI misdiagnosis, and measurement inaccuracies.

## Complications

TBI is associated with multiple chronic neurological complications. Its link with CTE is well established, while associations with AD and amyotrophic lateral sclerosis (ALS) remain under investigation. CTE manifests

clinically as TES, whose core features include cognitive impairment and/or neurobehavioral dysregulation (NBD) [41].

Cognitive impairment most commonly involves episodic memory and executive functioning deficits, typically in older individuals [42], with patients reporting memory or concentration problems and word-finding difficulty [24]. Symptom profiles vary by position and career length: longer football careers predict greater executive deficits, especially in verbal fluency (42.0 [40.7–52.7] in the group with career ≤ 7 years vs. 46.0 [40.0–51.0] in the group with career > 7 years, $p = 0.057$), while line-field players show poorer visuoperceptive performance. Although , it should be emphasized that all neuropsychological test performances of the athletes were within normal range and this study included only 20 football players aged 20–30 years [43]. In rugby, cognitive deficits occur in players aged 80+ with ≥ 3 concussions ($\beta = -1.04$; 95% CI [–1.62, –0.47]) [44]. Episodic memory – particularly for unstructured verbal stimuli – is most frequently impaired, affecting 21.2% of former football players, while 51.8% performed within the impaired range on at least one episodic memory test [45]. Severe semantic memory deficits correlate with p-tau in frontal and temporal regions ($r = -0.38$, $r = -0.48$), while episodic deficits relate to parahippocampal p-tau ($r = -0.38$, $r = -0.40$) [46]. Moreover, Alosco *et al.* [47] established that greater global p-tau burden is associated with cognitive and functional deficits (standardized $\beta \geq 0.02$, 95% confidence interval, CI > 0). Notably, p-tau severity explains 13–49% of the variance in standardized clinical measures, suggesting a robust link to reported symptoms.

NBD is characterized by explosiveness, impulsivity, and emotional dyscontrol [36], more common in younger individuals [42]. Although global p-tau is not linked to NBD, frontal cortex p-tau burden was associated with higher score in the standardized scale of NBD ($\beta$ standardized = 0.21, $p < 0.01$) [47,48]. Recent studies suggest that NBD in CTE may be driven by mechanisms other than p-tau deposition [45,47]. Alosco *et al.* [49] proposed myelin degeneration as a potential mechanism. They established greater degeneration is reflected by lower levels of myelin-associated glycoprotein, while this decreased level can be correlated with higher Barratt Impulsiveness Scale-11 scores ($\beta = -0.02$; 95% CI [–0.04, –0.0003]). Another study found that CSF interleukin-6 (IL-6) was associated with NBD total score ($\beta = 0.276$; 95% CI [0.107–0.446], false discovery rate [FDR] *p*-value = 0.012), impulsivity ($\beta = 0.294$; 95% CI [0.128–0.460], FDR *p*-value = 0.004) and affective lability ($\beta = 0.286$; 95% CI [0.120–0.452], FDR *p*-value = 0.005). In addition, researchers found that higher CSF norepinephrine correlated with greater emotional dysregulation ($\beta = 0.446$; 95% CI [0.081–0.811]; $p = 0.018$) and several other NBD components [50]. Former NFL players with ≥ 4 adverse childhood experiences (ACEs) show a 48% increased dementia risk (relative risk = 1.48; 95% CI [1.22–1.79]); since ACEs are linked to aggression and poor emotional regulation [51], NBD in some players may reflect ACE-related effects rather than direct CTE manifestations [52].

The relationship between RHI and ALS is still being explored [36]. Coexisting ALS and CTE has been associated with prior TBI [53]. Former NFL players exhibit higher ALS incidence and mortality, with longer careers increasing risk [54].

The link between AD and TBI remains debated. In a study of 2,005 retired amateur athletes, former boxers exhibited the highest risk of AD (HR 4.10; 95% CI [2.55–6.61]), followed by wrestlers (HR 2.11; 95% CI [1.28–3.48]) and soccer players (HR 2.07; 95% CI [1.23–3.46]) [5]. Former professional football players also show higher dementia/AD rates at younger ages [55]. However, other studies dispute this association: Stern *et al.* [56] found no differences in neuritic amyloid-beta (Aβ) plaque burden, and most

players lacked amyloid PET evidence of AD pathology. Early post-traumatic neurodegeneration also appears distinct from AD, characterized by central WM atrophy absent in AD [30]. Clinically, some former contact-sport athletes present with AD-like symptoms without amyloid pathology, with several cases confirmed postmortem as CTE [57]. A detailed case report similarly showed suspected CTE underlying an AD clinical diagnosis [58].

**Diagnostics**

The current diagnostic criteria for CTE were established during the Second NINDS/NIBIB Consensus Meeting [4]. At present, this tauopathy can only be diagnosed *postmortem*, although efforts to develop *in vivo* diagnostic criteria are ongoing [24].

Some studies suggest that flortaucipir (FTP) PET may detect CTE in former football players [59]. However, results remain inconclusive [33]. Elevated FTP uptake has been reported in prespecified regions, but correlations with cumulative head-impact exposure appeared only in the superior frontal region [60]. Off-target binding in the hippocampus and thalamus limits specificity to CTE p-tau [45]. Another study found minimal FTP differences between symptomatic players and controls, with only marginal associations with career duration in the entorhinal cortex, questioning its clinical utility [61]. In retired athletes negative for AD biomarkers, tau PET–positive regions showed lower gray matter volume and frontal/temporal patterns resembling CTE MRI, while higher tau burden correlated with worse memory. However, low specificity for 4R tau constrains clinical application [33]. Florbetapir PET showed no diagnostic differences in its uptake among professionals, college players and control. Florbetapir standardized uptake value ratios were not associated with football exposure years, cognition, or functioning [56].

MicroRNAs (miRNAs) show promise as concussion biomarkers, distinguishing concussed from non-concussed players with good sensitivity/specificity. Specific miRNAs correspond to acute and chronic phases of post-concussion recovery [62], and elevated miRNAs correlate with reduced dorsal cervical spinal cord WM integrity in collegiate players [63].

Neuroinflammation plays a central role in CTE, potentially making inflammatory biomarkers crucial for *in vivo* detection [64]. C-C motif chemokine ligand 2 (CCL2) recruits microglia to perivascular tau pathology in the frontal cortex, correlating with exposure years, microglial density, and CTE severity, more strongly than in AD. CCL may represent a future therapeutic target [65]. CSF IL-6 is elevated in players with NBD, linking to emotional dyscontrol and impulsivity, while plasma neurofilament light chain (NfL) associates with executive dysfunction [66]. Other markers, such as CCL11, CCL21, and glial fibrillary acidic protein (GFAP) show specificity limitations, supporting multimodal biomarker panels that combine clinical and imaging data for accurate CTE detection [64].

Blood biomarkers – including p-tau181, p-tau217, NfL, and GFAP – show potential for detecting CTE and TBI in athletes with RHI. Elevated p-tau217, p-tau181, and reduced GFAP, synaptosomal-associated protein 25, kallikrein-related peptidase 6, and beta-site amyloid precursor protein cleaving enzyme 1 have been reported in ex-rugby players [67,68], and acute increases in total tau and p-tau181 occur after soccer heading [69]. However, other studies report limited utility: NfL and GFAP were not associated with physical activity or number of headers [70], and t-tau, p-tau, NfL, and GFAP did not change during an American football season [71]. A scoping review found evidence weak and heterogeneous, deeming clinical application premature [72].

**Treatments**

Over the years, various therapeutic directions for CTE have emerged. A key approach

targets the primary cause (RHI) by modulating neuronal function at cellular and molecular levels. Rehabilitation, supported by modern technologies, aims to restore lost functions through neuroplasticity, with early initiation being crucial, as delayed admission to rehabilitation is associated with poorer outcomes, independent of initial injury severity [73].

Low-intensity pulsed ultrasound (LIPUS) delivers thermal and mechanical energy in pulses, exploiting phenomena such as acoustic radiation and streaming, particle displacement, and cavitation to temporarily open BBB for better drug delivery. Its thermal effects enhance neuronal excitability and synaptic transmission [74]. LIPUS reduces pro-inflammatory cytokines interleukin-1β [IL-1β], IL-6, interleukin-8 [IL-8], and tumor necrosis factor-α) and excess nitric oxide synthesis implicated in CTE pathogenesis [75]. Animal studies show reduced cerebral edema, improved BBB function, and lowered oxidative stress [76,77]. Numerous positive results from LIPUS studies conducted in animals indicate a potentially promising therapeutic strategy. However, the limitations of animal studies preclude the direct translation of results to human care. Furthermore, the dense structure of the skull and intermediate brain areas hinder the ultrasound waves from reaching the target structures. The safety of this method should also be validated, considering possible microhemorrhages, the negative impact of scattered intracranial ultrasound signals, and significant brain tissue damage induced by thermal energy [74].

Photobiomodulation (PBM) uses red (600–770 nm) and near-infrared light (800–1100 nm) to repair damaged cells, dilate lymphatic and blood vessels, reduce oxidative stress, and promote brain-derived neurotrophic factor [78,79]. Transcranial PBM improved verbal learning and memory in former football players with CTE after 18 transcranial light-emitting diode sessions [78]. Further research is necessary to validate this method and assess the potential introduction of self-administered PBM therapy at home.

Rehabilitation addresses common TBI-related cognitive deficits – attention, memory, and executive dysfunctions. Cognitive rehabilitation identifies, compensates for, and restores impaired cognitive and behavioral functions. Mood, depression, aggression, and anxiety improve with psychotherapy, including cognitive-behavioral therapy, music therapy, and mindfulness [73]. Traditional training combined with virtual reality enhances balance and mobility more than traditional methods alone [80]. Robotics-assisted therapies – self-feeding robots, wheelchair assistants, gait devices – improve quality of life [73].

Implanfig brain-computer interfaces decode neural signals; based on this information, functional electrical stimulation devices stimulate muscles directly, bypassing damaged neural pathways [73]. Neuromodulation techniques aim to reverse or prevent adverse neuroplastic changes. Non-invasive methods, such as transcranial magnetic stimulation and transcranial direct current stimulation, are safe and effective for motor and mood disorders. Invasive methods, such as deep brain stimulation and epidural motor cortex stimulation, are currently less favored due to their invasiveness [73].

Pharmacotherapy remains limited, but research on neuromodulatory agents (progesterone, astaxanthin, acetylcholinesterase inhibitors, enzogenol, amantadine, methylphenidate) continues. Erythropoietin reduces mortality, while botulinum toxin decreases muscle spasticity, and mirogabalin decreases neuropathic pain [73].

## Discussion

Current evidence indicates a strong correlation between contact sports participation and neurodegenerative diseases, such as CTE and ALS, though the association with AD remains a subject of ongoing debate [6,7].

A primary obstacle in this field is that a definitive CTE diagnosis can only be established post-mortem [4]. *In vivo* diagnostic attempts, particularly tau-PET imaging, continue to yield inconclusive or low-specificity results, failing to provide a clear correlation between imaging markers and specific proteinopathies [31,33]. This is further complicated by the overlapping clinical features of CTE and AD, which often lead to misdiagnosis [57]. Subjective symptoms of TES frequently emerge decades after the initial TBI, significantly delaying clinical recognition [81].

Morphological markers, while promising, offer limited diagnostic utility in isolation. For instance, while an elongated CSP demonstrates increased specificity for RHI exposure, its presence in the broader aging population diminishes its predictive value [29]. Furthermore, post-traumatic atrophy often lacks the specificity required to distinguish between Wallerian degeneration triggered by initial axonal injury and progressive neurodegeneration [30].

The reliability of current findings is constrained by significant selection and measurement biases. Most data originate from brain bank samples, which inherently favor symptomatic donors, potentially inflating the reported prevalence of CTE compared to the general athletic population [9,10]. This ascertainment bias is compounded by a reliance on retrospective informant reports to reconstruct exposure history, a method susceptible to recall bias, especially when informants are aware of the donor's symptomatic status [8,11]. Precision is further reduced by the use of "duration of play" as a proxy for cumulative head impact. This metric fails to account for heterogeneous play styles, evolving safety protocols, and transitions in sports equipment, such as the shift from leather to synthetic balls in soccer [8,13]. Furthermore, the demographic homogeneity of study cohorts, which are predominantly white and male, limits the generalizability of results to female and amateur athletes [35,37]. Finally, the superior overall health and lower all-cause mortality observed in elite athletes compared to population controls act as a significant confounding variable that may attenuate the observed risk of neurodegenerative outcomes [12,13].

The TBI rehabilitation sector suffers from systemic fragmentation and significant technical barriers. For example, in the United States of America, only 13–25% of survivors receive specialized interdisciplinary care, with access further restricted by rural-urban disparities and insurance limitations [73]. Emerging therapies, such as LIPUS and PBM, show promise in maintaining BBB integrity and addressing protein deposits [76,78]. However, these technologies face physical hurdles, such as the dense cranial barrier which attenuates wave penetration and requires precise parameter optimization [74]. Current clinical research into these modalities is often limited by small pilot samples and assessment tools that may be insensitive to functional progress in severely impaired patients [80]. Preventive strategies seem therefore critical. Examples include banning heading in children < 12 years and limiting high-force headers in adults in English Football Association. Another example of a potential rule change in soccer is allowing temporary player substitutions during suspected concussions, giving medical staff sufficient time for proper neurological assessment and reducing the risk of misdiagnosis [82].

**Conclusions**

Sport-related head impacts represent a critical risk factor for long-term neurodegeneration. CTE remains diagnosable only post-mortem, with *in vivo* methods insufficiently validated. Overlapping symptoms with other neurodegenerative diseases and delayed CTE onset hinder clinical recognition. Given the absence of effective treatments, preventive measures such as sport regulations seem essential to reduce long-

term risks. Future research must prioritize the validation of multimodal biomarker panels and the diversification of study populations to effectively address the diagnostic and therapeutic challenges associated with CTE.

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