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REVIEW

Prevalence of Chronic Traumatic Encephalopathy in Athletes With Repetitive Head Impacts: A Systematic Review and Meta-Analysis

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ABSTRACT

Chronic traumatic encephalopathy (CTE) is a neurodegenerative disorder associated with repetitive head impacts (RHI), which is commonly observed in athletes participating in contact sports. This systematic review and meta-analysis aimed to provide a clearer elucidation of the prevalence, risk factors, and potential outcomes of CTE in athletes. Through a comprehensive search of PubMed, EMBASE, Web of Science, and Google Scholar (January 2015 to July 2024), we ultimately included eight eligible studies with a low risk of bias involving 1000 former contact sports athletes diagnosed by neuropathological methods. Data were analyzed in a random-effects meta-analysis. The results indicated that the pooled prevalence of CTE in contact sport athletes was 53.7% (95% CI: 37.6–69.5, $I^2 = 93.7\%$). Subgroup analyses revealed that rugby players exhibited the highest prevalence at 64.7% (95% CI: 48.5–79.5, $I^2 = 0\%$), followed by American football players at 53.0% (95% CI: 33.2–72.4, $I^2 = 92.9\%$). By participation level, the prevalence was estimated at 44.1% (95% CI: 29.6–59.1, $I^2 = 84.5\%$) in amateur athletes and 72.8% (95% CI: 50.9–90.5, $I^2 = 91.6\%$) in elite athletes. Notably, the suicide rate among individuals diagnosed with CTE was 39.0% (95% CI: 12.7–69.0, $I^2 = 94.4\%$), and the pooled prevalence of CTE in non-suicidal athletes was 30.1% (95% CI: 6.9–60.4, $I^2 = 97.8\%$). These findings reveal the high prevalence of CTE in contact sport athletes exposed to RHI, especially at elite-level competition, and emphasize suicide as a potential outcome. Further studies should incorporate more female athletes to comprehensively assess the risk factors, outcomes, and effective prevention strategies for CTE.

1 | Introduction

Chronic traumatic encephalopathy (CTE) is a neurodegenerative disorder that typically occurs in individuals exposed to repetitive head impacts (RHI) [1], particularly in high-risk populations such as military veterans, victims of physical violence, and athletes involved in contact and collision sports [2, 3]. Over 97% of CTE cases have been reported in individuals with known histories of RHI, most commonly from participating in contact sports [4]. Given the millions of active and former athletes in contact sports, CTE represents a major public health concern. Nevertheless, the diagnosis of CTE remains elusive during an individual's lifetime and can only be definitively confirmed post-mortem through neuropathological examination. McKee et al. [1] examined the immunohistochemical features of 68 autopsy-confirmed CTE cases, including 50 American football players, and proposed neuropathological diagnostic criteria for CTE. Further refinement

of CTE's diagnostic criteria was provided by the National Institute of Neurological Disorders and Stroke/National Institute of Biomedical Imaging and Bioengineering (NINDS/NIBIB) through two consensus conferences. They defined the characteristic neuropathological lesion of CTE as the accumulation of abnormal p-tau proteins in neurons and astrocytes, which distribute around small blood vessels at the depths of cortical sulci in an irregular manner [5, 6].

The clinical symptoms of CTE typically involve behavioral changes (e.g., irritability or aggression), mood disturbances (e.g., depression or hopelessness), and cognitive impairments (e.g., deficits in episodic memory, executive function, or attention), which emerge years or even decades after experiencing RHI [7]. As the disease progresses, individuals may develop more severe neurodegenerative symptoms such as dementia, gait and speech abnormalities, and Parkinsonism [8, 9]. In order to provide a more comprehensive description of the clinical presentation, Montenigro et al. [10] proposed traumatic encephalopathy syndrome (TES) along with its research diagnostic criteria. The diagnostic framework includes five general criteria, three core clinical features, and nine supporting features, which are used to define subtypes of TES, including TES behavioral/mood variant, TES cognitive variant, TES mixed variant, and TES dementia.

In recent years, multiple academic neuropathology centers and brain banks in Canada, Australia, the United Kingdom, and the United States have reported confirmed cases of former athletes utilizing CTE neuropathology criteria, including American football [11], rugby [12], ice hockey [13], and European football players [14]. Although previous studies provided critical epidemiological data, no consensus exists regarding the prevalence of CTE. For instance, through analyzing data from the Veterans Affairs-Boston University-Concussion Legacy Foundation (VA-BU-CLF) Brain Bank, Mez et al. [11] found that 235 out of 290 American football players were diagnosed with CTE, yielding a prevalence rate of 81%. Similarly, Daneshvar et al. [15] reported that 71% of former American football players (451 individuals) from the Understanding Neurologic Injury and Traumatic Encephalopathy and Framingham Heart Study Brain Banks suffered from CTE. Such inconsistencies complicate the accurate estimation of the true prevalence of CTE and limit the generalization of study findings.

More recent studies indicate that various types of contact sports and levels of competition (e.g., amateur and professional-level participation) are associated with the risk of CTE [11, 16]. Various prevalence rates of CTE have been reported in American football and ice hockey players, ranging from 15% [17] to 99% [16] in American football and 29% [17] to 83% [1] in ice hockey. In addition, according to the VA-BU-CLF Brain Bank, among 202 deceased former American football players, CTE was neuropathologically diagnosed in 51 of 67 amateur-level athletes (76%), and 117 of 119 professional league athletes were diagnosed with CTE, a prevalence of 98% [16]. The prevalence of CTE in professional athletes was generally higher than that in amateur athletes, which may be closely related to the long-term and high-intensity contact training and competition of professional athletes. It has been reported that there is a dose–response relationship between the severity of CTE pathology and the duration of exposure to contact sports [15, 18]. The longer an athlete participates in contact sports, the greater the frequency and the higher the risk of experiencing high-intensity head impacts leading to CTE. Additional risk factors include genetic predisposition (e.g., *APOE* ε4) [19, 20], age of first exposure to contact sports [21], and alcohol or drug abuse [22, 23].

Regarding the long-term outcomes for CTE patients, suicide has been recognized as a potential correlate. In 2010, Omalu et al. [24] identified that two out of three former National Football League (NFL) players with CTE died by suicide and first proposed suicide as a clinical feature of CTE. This result has sparked considerable interest in the relationship between CTE and suicide. Recent studies have further supported this association and documented cases of suicide among CTE patients [15, 25]. However, no studies to date have directly investigated the association between CTE and suicidal tendencies [26, 27].

The available studies on the prevalence, primary risk factors, and potential outcomes of CTE are inconsistent and complex, and there is a lack of systematic reviews and meta-analyses on these topics. Accordingly, the present study aims to synthesize existing research findings and provide more reliable estimates. This will provide valuable insights for developing targeted interventions, advancing management strategies, and establishing evidence-based policies that will ultimately protect athletes' brain health and improve long-term outcomes.

2 | Materials and Methods

The systematic review and meta-analysis were conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [28]. The review protocol was registered with the PROSPERO international prospective register of systematic reviews [29] and is accessible online (Registration No. CRD42024576985). We were informed in an automated message from PROSPERO that due to their focus on systematic review registrations related to COVID-19, this submission was automatically published and not checked for eligibility. Patients and public partners were not involved in the design, implementation, or interpretation of this systematic review.

2.1 | Search Strategy

A comprehensive search spanning from January 2015 to July 2024 was conducted across electronic databases, including PubMed, EMBASE, and Web of Science. The search process used specific words around two areas: (1) “CTE” OR “Chronic traumatic encephalopathy”, (2) “player” OR “athlete”. The exact search strategy utilized in the present study is shown in Table S1. A secondary search of the reference lists of included articles and a Google Scholar search were also performed. This was done using backward citation tracking (to manually search the reference list of a journal article) and forward citation tracking (scanning a list of articles that had cited a given paper since it was published). Citations were tracked using Google Scholar

to make sure that studies were not missed inadvertently. When additional studies that met the inclusion criteria were identified, they were included in the final pool of studies.

2.2 | Selection Criteria

The inclusion criteria for retrieved studies were: (1) the study reported the prevalence of CTE in contact sport athletes, including but not limited to American football, boxing, hockey, rugby, and wrestling; (2) the study design was a retrospective cohort or cross-sectional study; (3) standardized neuropathological diagnostic methods were used to confirm the presence of CTE; (4) the study provided adequate data to estimate the prevalence or risk of CTE; (5) the study was published in English to ensure access and interpretation of the information. The exclusion criteria were as follows: (1) the study did not directly report case numbers and sample size used to calculate prevalence; (2) case reports and single case series; (3) the study reported data from the same cohort as another study; for multiple published studies using the same cohort, only the study with the most complete reporting of information was retained for analysis; (4) the study with patient number less than 10. The titles and abstracts of possible publications for inclusion were screened independently by authors D.F. and B.Q. using the selected inclusion/exclusion criteria. After this initial screening, the full texts of the articles meeting the inclusion criteria were retrieved and screened by the same two authors. Disagreements were resolved via discussion between the authors or via consultation with a third author (Y.Z.).

2.3 | Data Extraction

Data were extracted from the included studies by two investigators (B.Q. and J.T.) using a pre-established standardized data extraction form. The following data were extracted where available: study characteristics (i.e., main author, year of publication, country, brain bank, study design and sample size), population characteristics (i.e., type of sport, competitive level, and age at death), CTE diagnostic criteria, the number of individuals with CTE, or the prevalence of CTE. If the statistical data reported in a study were incomplete or insufficient to determine the prevalence in the study sample, the study was excluded.

2.4 | Risk of Bias Quality Assessment

The risk of bias of the selected studies was assessed by using the Joanna Briggs Institute (JBI) Critical Appraisal Checklist for Studies Reporting Prevalence Data [30, 31]. The specific JBI questions are presented in Table S2. The checklist contains nine questions that are categorized into three domains: participants (Questions 1, 2, 4, and 9), outcome measurement (Questions 6 and 7), and statistics (Questions 3, 5, and 8). Two reviewers (D.F. and B.Q.) independently scored each included study and cross-checked the information. In cases of disagreement, a third author (Y.Z.) was consulted. Each of the questions in this assessment tool was scored as "Yes," "No," "Unclear," or "Not Applicable". The risk of bias was categorized as "high" if the percentage of "yes" scores reached up to 49%; "moderate" for "yes" scores percentages between 50% and 69%; and "low" for "yes" scores percentages above 70% [32].

2.5 | Statistical Analysis

Meta-analysis was performed using the DerSimonian and Laird random-effects model to obtain the pooled prevalence of CTE due to expected heterogeneity among studies. The pooled effect size with a 95% confidence interval (CI) was generated and presented using a forest plot. Statistical heterogeneity was assessed using the I^2 statistic and defined as follows: $I^2 > 50\%$, substantial heterogeneity; $20\% < I^2 \leq 50\%$, moderate heterogeneity; and $I^2 < 20\%$, low heterogeneity [33]. Subsequently, the sources of heterogeneity were explored by subgroup analyses, including sex, type of sport, and level of competition [34]. Besides, we assessed the robustness of the CTE prevalence results using sensitivity analysis (leave-one-out method), in which the overall pooled estimates were calculated after deleting one study in each round [35]. Lastly, publication bias was assessed visually using a funnel plot and Egger's test for asymmetry if the number of included studies was more than 10. All statistical analyses were performed using Stata version 16.0 (StataCorp, College Station, TX, USA).

3 | Results

3.1 | Study Selection

The flowchart of literature selection is presented in Figure 1 in accordance with the latest PRISMA guidelines. The initial search yielded 1924 articles. After excluding 700 duplicates, we reviewed 1224 titles and abstracts for eligibility and excluded 1216 articles that did not meet the eligibility criteria. Eventually, there were a total of eight studies were used for the systematic review and meta-analysis.

3.2 | Characteristics of Included Studies

This systematic review included eight articles published between 2015 and 2024, involving 1000 retired athletes from four countries who participated in contact sports and experienced RHI. All studies performed a neuropathologic diagnosis of CTE according to the criteria proposed by McKee et al. [1] or the consensus criteria developed by the NINDS/NIBIB [5, 6]. The collected data included author, year of publication, brain bank source, sample size, sex distribution, age at death, main type of sport, and level of competition. Table 1 summarizes the main characteristics and primary outcomes of the included studies.

3.3 | Quality of Included Studies and Publication Bias

According to the JBI Critical Appraisal Checklist for Studies Reporting Prevalence Data, the majority of the eight articles included in this review demonstrated high methodological quality. Specific grades (yes, no, unclear, or not applicable) were shown in Table S2, and no studies were excluded from our meta-analysis

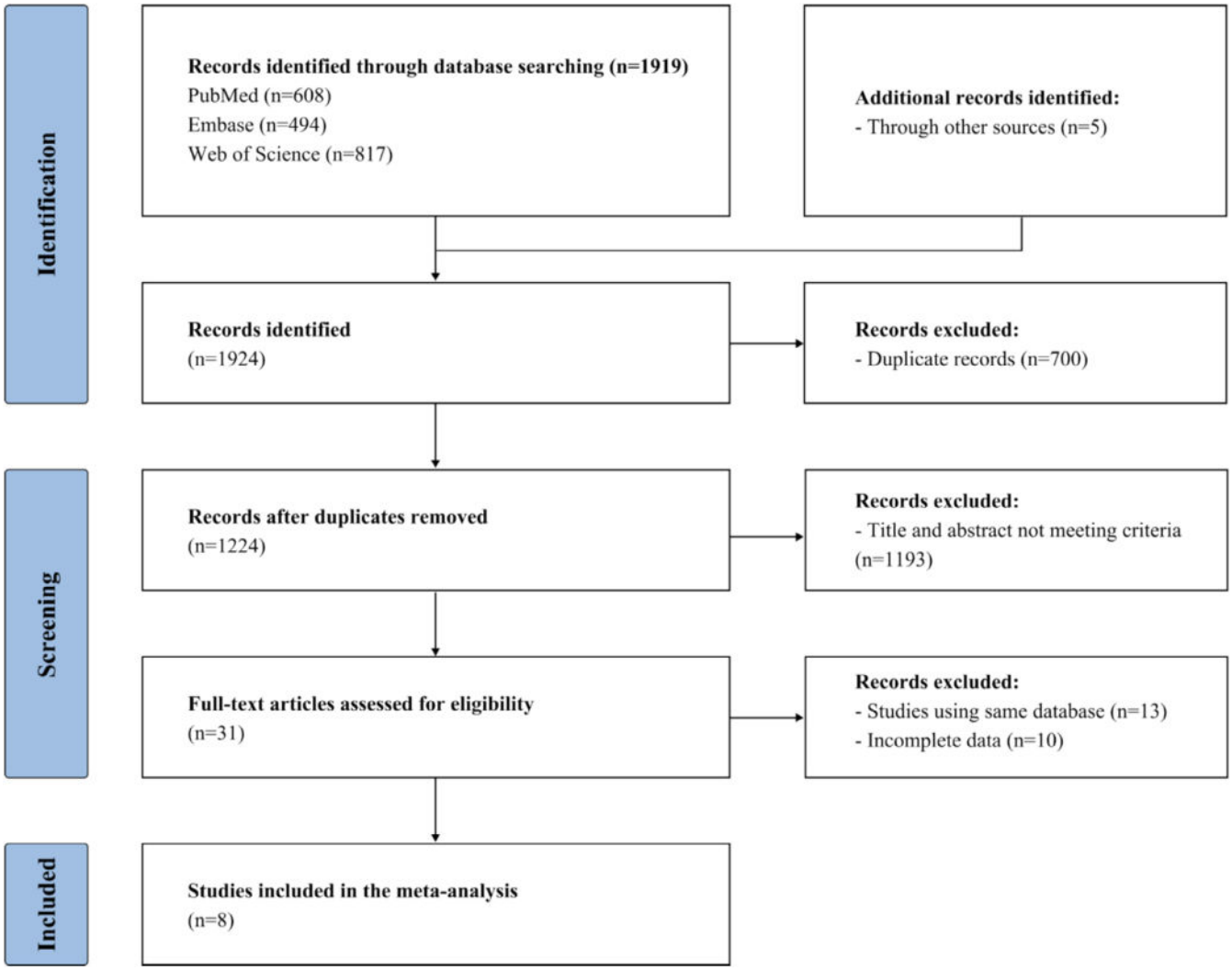

FIGURE 1 | PRISMA flow diagram of the literature search and study selection process.

due to methodological quality. Notably, insufficient sample size and inappropriate sampling methods were the main risks of bias of these studies. This study was unable to assess publication bias because less than 10 studies were original articles [39].

3.4 | Prevalence of Chronic Traumatic Encephalopathy in Athletes

The overall pooled prevalence of CTE among former contact sport athletes was 53.7% (95% CI: 37.6–69.5), with substantial heterogeneity ($I^2 = 93.7\%$, $p < 0.001$). Prevalence estimates from individual studies ranged from 26.9% to 91.7%, whereas the weight of individual studies ranged from 9.9% to 14.2%. Figure 2 presents the forest plot derived from the meta-analysis.

3.4.1 | Types of Sports

The subgroup analysis of different types of sports demonstrated that rugby had the highest prevalence of CTE with 64.7% (95% CI: 48.5–79.5, $I^2 = 0\%$), followed by American football with a prevalence of 53.0% (95% CI: 33.2–72.4, $I^2 = 92.9\%$), and the lowest prevalence was in hockey (42.0%) (95% CI: 26.2–58.5, $I^2 = 7.2\%$) (Figure 3). Due to the limited number of studies available for other sports (less than three), this study was unable to perform meta-analyses for these categories, including Australian rules football, European football, and wrestling.

3.4.2 | Sex of Athletes

This study included 14 female and 986 male participants for subgroup analysis. The results revealed a significantly higher prevalence of CTE in male athletes (55.0%, 95% CI: 39.1–70.4, $I^2 = 93.3\%$) compared to female athletes (0.7%, 95% CI: 0.0–24.4, $I^2 = 0$) (Figure 4).

3.4.3 | Levels of Competition

According to the level of competition in contact sports, the study included 531 amateur athletes and 455 elite athletes for analysis. The prevalence of CTE in former amateur athletes (including high school and collegiate level athletes) with a history of RHI was 44.1% (95% CI: 29.6–59.1, $I^2 = 84.5\%$), while the prevalence

TABLE 1 | Summary of the descriptive characteristics of included articles.

No.	Author (Year)	Brain Bank; Country	Sample size	Male	Age at death or age range	Number of CTE (%)	Main types of sports (CTE number, Total number)	Level of participants (CTE number, Total number)	Number of suicides in CTE (%)	Diagnostic criteria
1	Bieniek et al. (2015) [36]	Mayo Clinic Brain Bank; US	66	66	73.4 ± 11.2	21 (31.8)	AF (16, 43), Bo (2, 8)	Amateur (12, 44), Elite (6, 8)	/	McKee criteria
2	Schwab et al. (2021) [13]	Canadian Concussion Centre's; Canada	35	35	62.6 ± 17.5	17 (48.6)	AF (11, 24), H (6, 11)	Amateur (3, 6), Elite (14, 29)	/	NINDS/NIBIB criteria
3	Suter et al. (2022) [25]	ASBB; Australia	21	19	59.6 ± 23.0	12 (57.1)	ARF (6, 8), Ru (5, 9)	Amateur (4, 10), Elite (8, 11)	6 (50.0)	NINDS/NIBIB criteria
4	Daneshvar et al. (2023) [15]	VA-BU-CLF and FHS Brain Bank; US	631	631	59.7 ± 20.1	451 (71.5)	AF (451, 631)	Amateur (189, 320), Elite (262, 311)	62 (13.7)	NINDS/NIBIB criteria
5	McKee et al. (2023) [37]	UNITE Brain Bank; US	152	141	23.0 ± 4.3	63 (41.4)	AF (48, 92), H (6, 16), S (4, 23), W (2, 9), Ru (2, 4)	Amateur (45, 128), Elite (18, 24)	33 (52.4)	NINDS/NIBIB criteria
6	Stewart et al. (2023) [18]	UNITE Brain Bank, GTBI Archive, ASBB; US, UK, Australia	31	30	60.4 ± 21.7	21 (67.7)	Ru (21, 31)	Amateur (13, 23), Elite (8, 8)	/	NINDS/NIBIB criteria
7	Taskina et al. (2024) [17]	None; Canada	52	52	60.4 ± 19.1	14 (26.9)	AF (4, 26), H (4, 14)	Elite (14, 52)	7 (36.8)	NINDS/NIBIB criteria
8	Taghdiri et al. (2024) [38]	None; Canada	12	12	71.7 ± 14.0	11 (91.7)	AF (7, 7), H (3, 4)	Elite (11, 12)	/	NINDS/NIBIB criteria

Abbreviations: AF, American football; Amateur, not semiprofessional or professional athletes; ARF, Australian rules football; ASBB, the Australian Sports Brain Bank (Royal Prince Alfred Hospital and University of Sydney, Australia); Bo, boxing; FHS, Framingham Heart Study; GTBI, the Glasgow TBI Archive (University of Glasgow, United Kingdom); H, hockey; Ru, rugby; S, European football; UNITE, Understanding Neurologic Injury and Traumatic Encephalopathy Brain Bank (Boston University School of Medicine, United States); VA-BU-CLF, Veterans Affairs-Boston University-Concussion Legacy Foundation; W, wrestling.

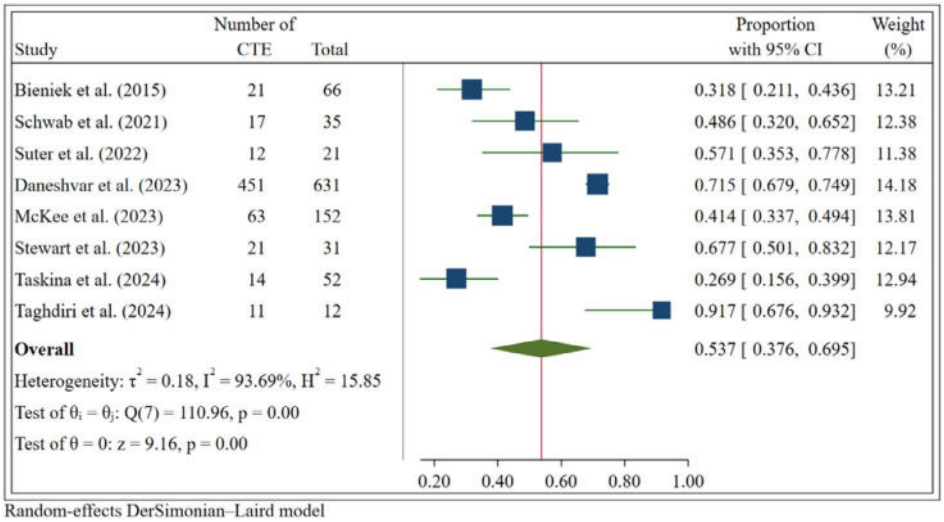

FIGURE 2 | Forest plot of meta-analysis for the pooled prevalence of CTE in former contact sport athletes. Blue square: Point estimate; Green diamond: Combined point estimate and 95% CI.

among former elite athletes (encompassing professional and semi-professional level athletes) was 72.8% (95% CI: 50.9–90.5, $I^2 = 91.6\%$) (Figure 5).

3.5 | Suicide Rates in Athletes With Chronic Traumatic Encephalopathy

The meta-analysis revealed a pooled suicide rate of 39.0% (95% CI: 12.7–69.0, $I^2 = 94.4\%$) among contact sport athletes diagnosed with CTE. Four various studies reported suicide prevalence estimates ranging from 13.7% to 52.4% (Figure 6).

3.6 | Prevalence of Chronic Traumatic Encephalopathy in Non-Suicidal Athletes

The study included 432 athletes with CTE who died from non-suicidal causes for analysis. The meta-analysis revealed that the pooled prevalence of CTE in non-suicidal athletes was 30.1% (95% CI: 6.9–60.4, $I^2 = 97.8\%$). Four studies reported CTE prevalence estimates ranging from 13.5% to 61.6% (Figure 7).

3.7 | Sensitivity Analysis

The sensitivity analysis was performed to assess the effect of individual studies on the pooled estimates of CTE prevalence. The results showed that when individual studies were ignored, the pooled prevalence obtained was within the 95% CI of the overall pooled prevalence. This substantiated that no single study biased the overall effect size (Figure S1).

4 | Discussion

To the author's best knowledge, this study is the first systematic review and meta-analysis to comprehensively evaluate the prevalence and risk factors of CTE in contact sport athletes with a history of RHI, as well as the prevalence of necropsies with suicide and CTE-compatible pathology. The primary finding of this study suggested that the overall prevalence of CTE among contact sport athletes exposed to RHI was as high as 53.7%, highlighting a significant health risk for this population. In comparison, previous studies have reported CTE prevalence rates ranging from 26.9% to 91.7% in this high-risk population [13, 15, 17, 18, 25, 36–38]. These discrepancies may stem from selection bias, recall bias, and complexity of diagnostic criteria. Specifically, the present study involved samples of CTE patients from four countries and seven brain banks, including the Mayo Clinic Brain Bank, Canadian Concussion Centre, Australian Sports Brain Bank, VA-BU-CLF and FHS Brain Bank, Understanding Neurologic Injury and Traumatic Encephalopathy Brain Bank, and the Glasgow TBI Archive. Multiple studies have indicated that athletes with clinical symptoms of CTE are more likely to donate their brains for research [40, 41]. This may contribute to a sample that is skewed toward individuals with more severe brain injuries or more pronounced symptoms while overlooking those who are asymptomatic or have milder injuries. This sample selection bias could result in an overestimation of CTE prevalence. Furthermore, the clinical information about donated brains typically relies on retrospective interviews with close relatives, which are susceptible to recall bias (including the accuracy of historical event or symptom information), thus affecting the accuracy of the data [41, 42]. Notably, some of the pathologic features listed in the diagnostic criteria for CTE, particularly p-tau deposition, are also observed in other neurodegenerative diseases, such as Alzheimer's disease (AD), frontotemporal dementia, and Lewy body disease [5, 13]. Iverson et al. [43] suggested that some cases of CTE may be misdiagnosed as aging-related tau astrogliopathy (ARTAG), emphasizing the need for improved neuropathological diagnostic criteria to better distinguish CTE from other neurodegenerative diseases. Additionally, Schwab et al. [13] conducted a retrospective cohort study and found that 17 of 35 former American football or ice hockey players showed pathologic evidence of CTE, while 24 players exhibited other neuropathologies, including 15 cases of AD and five cases of ARTAG, with 13 cases coexisting with CTE. Similarly, Suter et al. [25] revealed the presence of other pathological features in 12 patients with CTE, including

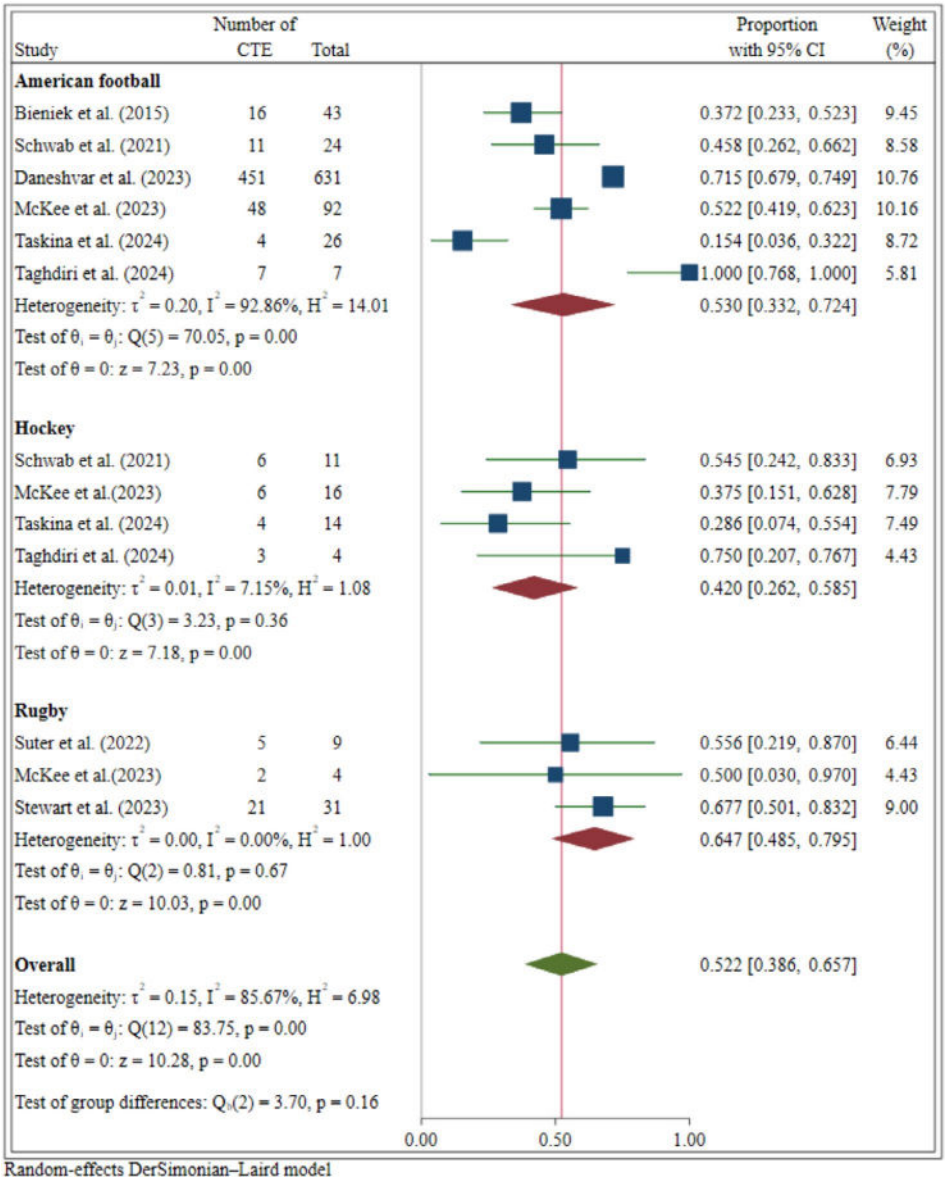

FIGURE 3 | Forest plot of meta-analysis on the prevalence of CTE in athletes of different types of contact sports. Blue square: Point estimate; Green diamond: Combined point estimate and 95% CI.

three cases of AD and three cases of ARTAG. These findings suggest that CTE may commonly co-occur with other neurodegenerative diseases, further increasing the complexity of pathological diagnosis and potentially affecting the accuracy of CTE prevalence estimates.

The prevalence of CTE varies among athletes involved in different types of contact sports. Specifically, rugby players exhibit the highest prevalence (64.7%), followed by American football players (53.0%) and ice hockey players (42.0%). This variation may be attributed to multiple factors, including the characteristics of each sport and the use of protective equipment. The longer duration of rugby matches compared to other contact sports (American football, ice hockey) is more likely to increase the cumulative risk of head impacts in athletes, including concussions and sub-concussive impacts. A retrospective study by Van Pelt et al. [44] found that rugby had the highest risk of concussion, followed by American football, ice hockey, and wrestling. Hollis et al. [45] reported the time to sustain a mild TBI among a cohort of community rugby union athletes, and 7% of the cohort sustained a mild TBI within 10 h of game time, increasing to 14% within 20 h. Moreover, differences in the use of protective equipment may influence the risk of developing CTE. Rugby provides minimal head protection, and its rules only permit players to wear scrum caps made of soft, thin materials. In contrast, American football regulations, such as the National Football League and the National Collegiate Athletic Association, require players to use hard football helmets during play. Similarly, in ice hockey, organizations like the International Ice Hockey Federation and the National Hockey League require athletes to wear helmets and mouthguards. These devices are designed to reduce the risk of head injuries by absorbing and dissipating the mechanical forces generated by external impacts [46]. Although there is no direct research demonstrating that the aforementioned factors could influence the prevalence of CTE, given

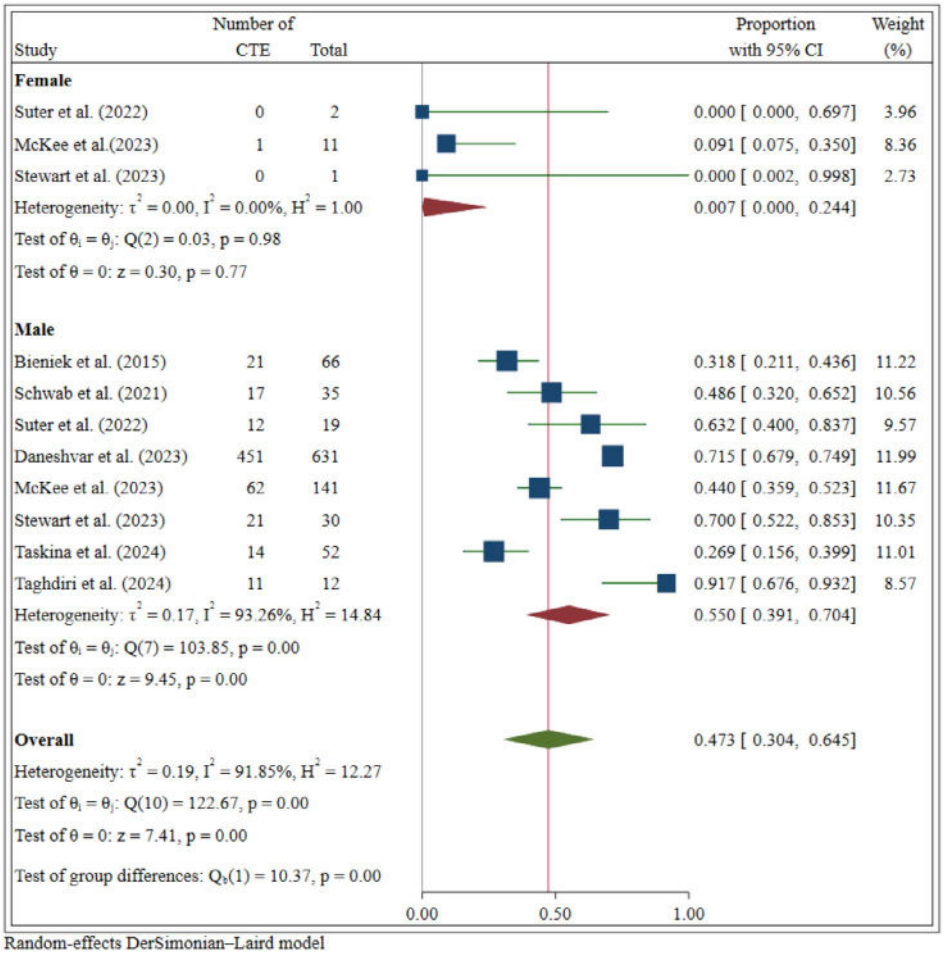

FIGURE 4 | Forest plot of meta-analysis for prevalence of CTE in contact athletes by sex. Blue square: Point estimate; Green diamond: Combined point estimate and 95% CI.

their crucial role in reducing the risk of concussions and/or sub-concussive head impacts, they may still have a potential effect in reducing the occurrence of CTE.

Sex may be a contributing risk factor for CTE. Subgroup analysis results showed that male athletes (55.0%) had a significantly higher prevalence of CTE compared to female athletes (0.7%). It is important to note that 98.6% of the participants in the current study were male ($n = 986$), while only 1.4% were female ($n = 14$), and this disproportionate sex distribution may limit the generalization of the findings to female athletes. Previous studies of CTE have predominantly focused on the male population, with relatively limited research involving female athletes [13, 15]. This discrepancy may be attributed to the higher participation rates of males in most contact sports. Although the participation of females in contact sports has increased over the past two decades [47] and the reported head injury rate has risen significantly [48, 49], the number of donated brains available for post-mortem pathological examination of CTE remains limited, restricting accurate estimates of CTE prevalence in female athletes. Existing evidence suggests that females are more susceptible to sports-related concussions than males, even after controlling for participation rates [50]. Furthermore, females experience a greater number, severity, and/or duration of post-concussive symptoms, such as headaches, irritability, aggressiveness, anxiousness, depression, or short-term memory impairment [51, 52]. Given these adverse factors, future research should emphasize the study of female athletes to facilitate the elucidation of CTE prevalence in female athletes, thereby informing more targeted prevention and intervention strategies.

The different levels of competition can significantly affect the prevalence of CTE in athletes [16]. The present study demonstrated that elite athletes exhibited a higher prevalence of CTE (72.8%) compared to amateur athletes (44.1%), indicating that the prevalence of CTE increased with the level of competition. The elevated prevalence of CTE among elite athletes may be attributed to their prolonged exposure to high-frequency, high-intensity mechanical head impacts during professional competition. Comparative rugby injury studies have demonstrated that professional athletes experience 22.2 concussions per 1000 player hours [53], which is 5.4 times the rate of concussions at the amateur community level (4.1/1000 player hours) [54]. Sub-concussive events may occur more frequently. Bailes et al. [55] have utilized helmet accelerometer data to estimate that high school American football players sustain approximately 600 sub-concussive impacts per season, collegiate athletes exceed 1000, and professional athletes have more than 1200 impacts. This disparity in the frequency of impacts influences the risk of developing CTE. LeClair et al. [11] found that college-level and professional American football players had 2.4 and 2.5 times the risk of being diagnosed with CTE compared to high school-level

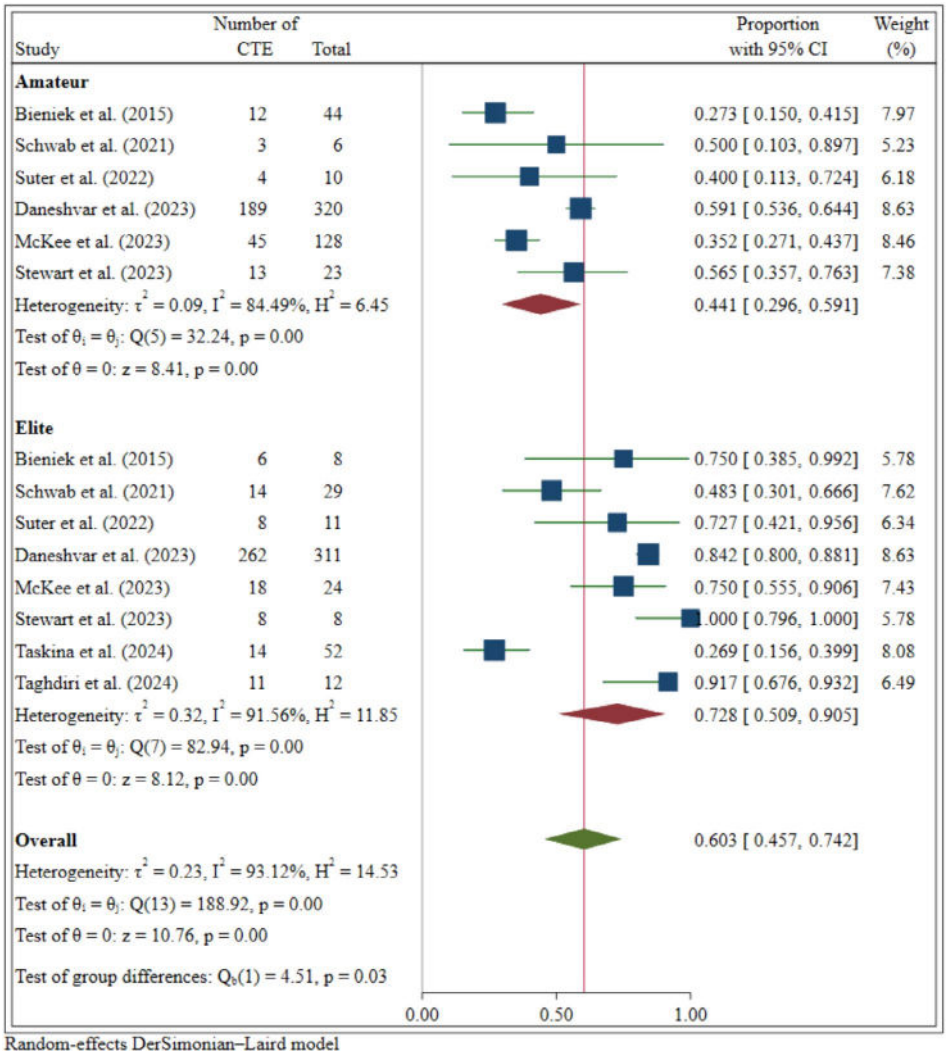

FIGURE 5 | Forest plot of a meta-analysis on the prevalence of CTE in contact sports athletes at different levels of competition. Blue square: Point estimate; Green diamond: Combined point estimate and 95% CI.

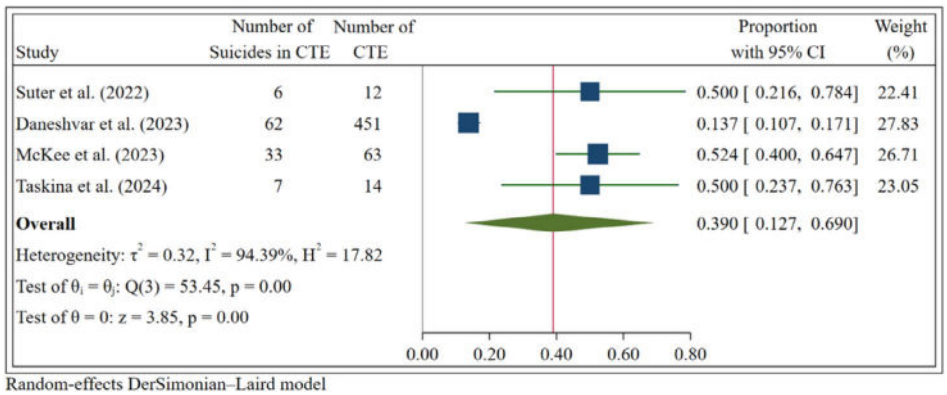

FIGURE 6 | Forest plot of a meta-analysis of suicide rates in athletes with CTE. Blue square: Point estimate; Green diamond: Combined point estimate and 95% CI.

players, respectively. They estimated the cumulative incidence of CTE among high school, college, and professional American football players in their target population as at least 5.1, 376, and 10 703 cases per 100 000 deaths, respectively. To further quantify the consequences of RHI exposure and CTE, Daneshvar et al. [15] developed the Positional Exposure Matrix model, which confirmed that for approximately every additional 1000 head impacts increased the odds of being diagnosed with CTE increased by 21%, and among those already diagnosed with CTE, the odds of being diagnosed with severe CTE increased by 13%.

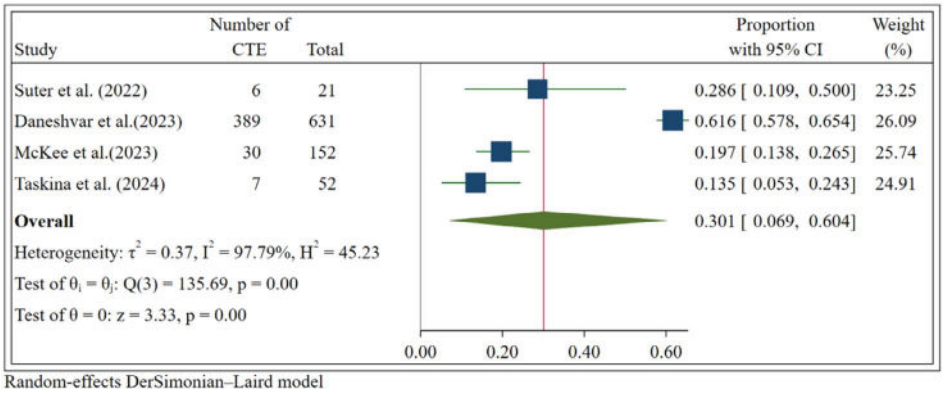

FIGURE 7 | Forest plot of meta-analysis for the pooled prevalence of CTE in non-suicidal athletes. Blue square: Point estimate; Green diamond: Combined point estimate and 95% CI.

This study provided evidence of potential long-term outcomes associated with CTE. The meta-analysis results revealed that 39% of CTE patients died by suicide, which indicates that CTE may be a potential factor in increased suicide risk. Mez et al. [16] previously reported that the most common cause of death for American football players with mild CTE pathology was suicide (27%), further emphasizing the profound impact of CTE on individual mental health and behavior. The relationship between CTE and suicide risk is complex and multifactorial. In some cases, co-occurring mental health conditions (e.g., depression, anxiety) may independently contribute to suicide risk, even in individuals diagnosed with CTE post-mortem. For instance, Grashow et al. [56] conducted a cross-sectional study within the Football Players Health Study at Harvard University and discovered that suicidality was reported by 171 of 681 former professional American football players with perceived CTE (25.4%) and 64 of 1299 without perceived CTE (5.0%). Importantly, even after adjusting for established predictors of suicidality (e.g., depression, anxiety), individuals with perceived CTE remained twice as likely to report suicidality. Furthermore, the association between CTE and suicide risk may involve indirect pathways mediated by comorbid factors. Chronic pain is a common health issue among CTE patients [26] and is often caused by brain injuries, joint, or other musculoskeletal injuries sustained during an athlete's career [57]. The existing evidence has demonstrated that patients suffering from chronic pain are more susceptible to suicide and suicidality [58]. Chronic pain patients frequently rely on opioid medications for relief, which may lead to substance abuse [59]. Cottler et al. [60] surveyed 344 retired NFL players regarding their opioid use throughout their careers and found that over half (52%) of individuals used opioids during their NFL career, with 71% reporting misuse. The excessive use of opioids can depress the central nervous system, leading to exacerbating emotional instability and worsening depressive symptoms. This creates a vicious cycle between chronic pain and mental health problems that further increases the risk of suicidal thoughts and behaviors [61]. It is worth noting that not all studies have found a high risk of suicide in athletes participating in contact sports, and the results of multiple studies have even challenged the proposed link between CTE and suicide [27, 62, 63]. To clarify this controversy, Pichler et al. [63] suggested that future research should adopt longitudinal designs or large multicenter cohort studies. These studies could help explore the relationship between the development of CTE and its potential clinical manifestations, particularly mental health conditions that may contribute to suicide.

The mandatory autopsy procedures in suicide cases may introduce selection bias into CTE research. Therefore, this study conducted a secondary analysis of CTE prevalence exclusively in non-suicide cases. The meta-analysis revealed that the prevalence of CTE among non-suicidal athletes was 30.1%, which was lower than the overall prevalence including suicidal athletes (53.7%). These findings suggested that CTE prevalence may be overestimated in existing epidemiological studies. Nonetheless, the risk of CTE in athletes who have suffered RHI remains a major concern. Previous studies have demonstrated that athletes exposed to RHI in various contact sports may experience varying degrees of neurocognitive impairment [64, 65]. To mitigate CTE risks, evidence-based prevention strategies targeting head injury reduction in contact sports are crucial. Contact training is a common approach to preventing concussions and head impacts in American football [66, 67]. The USA Football and NFL's Heads Up Football program, which emphasizes coach certification and standardized contact training protocols, has reduced concussion incidence by 32% and head impact frequency by 38% in high school American football athletes [68]. Some evidence also supported that implementing a neuromuscular training warm-up program during rugby matches reduced the incidence of concussion [69, 70]. Additionally, policies and rules that limit the number and duration of contact practices, the intensity of contact in practice, and strategies to restrict the duration of collisions in American football practices at all levels have also been shown to reduce the rate of head impacts [70, 71].

4.1 | Limitations of the Study

This study has several limitations that should be considered. Primarily, there was significant heterogeneity among the included studies, which is a common challenge in epidemiological meta-analyses but does not invalidate our findings [72, 73]. In this study, we attempted to identify and account for sources of heterogeneity through random-effects modeling and subgroup analyses. However, some potential sources of heterogeneity may remain unaddressed, and future studies are encouraged to explore these factors to enhance the understanding and interpretation of the findings. Secondly, this review was limited to articles

published in English, omitting studies in other languages, as well as reports. Additionally, the inclusion of studies with small sample sizes could have affected the estimated pooled prevalence. Publication bias is also a concern, as studies reporting positive associations between RHI and CTE are more likely to be published.

5 | Conclusions

In conclusion, this systematic review and meta-analysis revealed a high prevalence of CTE among former contact sports athletes with a history of RHI, particularly in high-intensity sports and elite-level competition. Furthermore, the identification of suicide as a potential outcome of CTE underscores the severe mental health implications associated with this disease. Future research should further investigate the pathogenesis and potential risk factors of CTE, providing a scientific basis for optimizing preventive strategies and improving long-term health outcomes.

6 | Perspective

This study is the first meta-analysis to reveal the high prevalence of CTE in contact sport athletes, particularly in rugby and American football players, as well as those participating in high-intensity elite competitions. Future research should prioritize more specific assessments of CTE pathology at autopsy, particularly targeting younger individuals who are less likely to be confounded by age-related tau deposits. In addition, it is essential to systematically investigate the prevalence of CTE among female athletes, an area that remains significantly underexplored. The development of early diagnostic tools for CTE also represents a critical direction for future research, such as the use of positron emission tomography. These findings provide valuable evidence to guide the design of effective protective measures and treatment strategies, which may ultimately contribute to improving long-term neurological outcomes in athletes.

Acknowledgments

The authors have nothing to report.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The authors have nothing to report.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.