Chronic traumatic encephalopathy in American football players

There is tremendous media attention regarding chronic traumatic encephalopathy (CTE), primarily because of the deaths of high profile American football players who were found to have CTE upon neuropathology [1].

Physicians in clinical practice are likely to face an increasing number of retired football players seeking evaluation for chronic neurobehavioral symptoms. Guidelines for the evaluation and treatment of these patients are sparse. Clinical criteria for a diagnosis of CTE are under development. The contribution of CTE vs other neuropathologies to neurobehavioral symptoms in these players remains unclear.

Gardner et al. describe the experience in evaluating and treating a series of 14 self-referred symptomatic players. The aim is to raise awareness in the neurology community regarding the different clinical phenotypes, idiosyncratic but potentially treatable symptoms, and the spectrum of underlying neuropathologies in these players [2].

Altered Corpus Callosum White Matter Microstructure

Forty retired National Football League (NFL) players, ages 40-65, were matched by age and divided into two groups based on their age of first exposure (AFE) to tackle football: before age 12 or at age 12 or older. Participants underwent DTI on a 3 Tesla Siemens (TIM-Verio) magnet. The whole CC and five subregions were defined and seeded using deterministic tractography. Dependent measures were fractional anisotropy (FA), trace, axial diffusivity and radial diffusivity. Results showed that former NFL players in the AFE <12 group had significantly lower FA in anterior three CC regions and higher radial diffusivity in the most anterior CC region than those in the AFE ≥12 group. [3].

Prevention

Findings suggest that regulation of practice equipment could be a fair and effective way to substantially reduce subconcussive head impact in thousands of collegiate football players [4].

Case series

2017

Case series of 202 football players whose brains were donated for research. Neuropathological evaluations and retrospective telephone clinical assessments (including head trauma history) with informants were performed blinded. Online questionnaires ascertained athletic and military history.

Neuropathological diagnoses of neurodegenerative diseases, including CTE, based on defined diagnostic criteria; CTE neuropathological severity (stages I to IV or dichotomized into mild [stages I and II] and severe [stages III and IV]); informant-reported athletic history and, for players who died in

2014 or later, clinical presentation, including behavior, mood, and cognitive symptoms and dementia.

Among 202 deceased former football players (median age at death, 66 years [interquartile range, 47-76 years]), CTE was neuropathologically diagnosed in 177 players (87%; median age at death, 67 years [interquartile range, 52-77 years]; mean years of football participation, 15.1 [SD, 5.2]), including 0 of 2 pre-high school, 3 of 14 high school (21%), 48 of 53 college (91%), 9 of 14 semiprofessional (64%), 7 of 8 Canadian Football League (88%), and 110 of 111 National Football League (99%) players. Neuropathological severity of CTE was distributed across the highest level of play, with all 3 former high school players having mild pathology and the majority of former college (27 [56%]), semiprofessional (5 [56%]), and professional (101 [86%]) players having severe pathology. Among 27 participants with mild CTE pathology, 26 (96%) had behavioral or mood symptoms or both, 23 (85%) had cognitive symptoms, and 9 (33%) had signs of dementia. Among 84 participants with severe CTE pathology, 75 (89%) had behavioral or mood symptoms or both, 80 (95%) had cognitive symptoms, and 71 (85%) had signs of dementia.

In a convenience sample of deceased football players who donated their brains for research, a high proportion had neuropathological evidence of CTE, suggesting that CTE may be related to prior participation in football [5)].

1)
Riley DO, Robbins CA, Cantu RC, Stern RA. Chronic traumatic encephalopathy: Contributions from the Boston University Center for the Study of Traumatic Encephalopathy. Brain Inj. 2015;29(2):154-63. doi: 10.3109/02699052.2014.965215. PubMed PMID: 25587744.
2)
Gardner RC, Possin KL, Hess CP, Huang EJ, Grinberg LT, Nolan AL, Cohn-Sheehy BI, Ghosh PM, Lanata S, Merrilees J, Kramer JH, Berger MS, Miller BL, Yaffe K, Rabinovici GD. Evaluating and treating neurobehavioral symptoms in professional American football players: Lessons from a case series. Neurol Clin Pract. 2015 Aug;5(4):285-295. PubMed PMID: 26336629.
3)
Stamm JM, Koerte IK, Muehlmann M, Pasternak O, Bourlas AP, Baugh CM, Giwerc MY, Zhu A, Coleman MJ, Fritts NG, Martin B, Chaisson C, McClean MD, Lin AP, Cantu RC, Tripodis Y, Stern R, Shenton ME. Age at First Exposure to Football is Associated with Altered Corpus Callosum White Matter Microstructure in Former Professional Football Players. J Neurotrauma. 2015 Jul 22. [Epub ahead of print] PubMed PMID: 26200068.
4)
Reynolds BB, Patrie J, Henry EJ, Goodkin HP, Broshek DK, Wintermark M, Druzgal TJ. Practice type effects on head impact in collegiate football. J Neurosurg. 2015 Aug 4:1-10. [Epub ahead of print] PubMed PMID: 26238972.
5)
Mez J, Daneshvar DH, Kiernan PT, Abdolmohammadi B, Alvarez VE, Huber BR, Alosco ML, Solomon TM, Nowinski CJ, McHale L, Cormier KA, Kubilus CA, Martin BM, Murphy L, Baugh CM, Montenigro PH, Chaisson CE, Tripodis Y, Kowall NW, Weuve J, McClean MD, Cantu RC, Goldstein LE, Katz DI, Stern RA, Stein TD, McKee AC. Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football. JAMA. 2017 Jul 25;318(4):360-370. doi: 10.1001/jama.2017.8334. PubMed PMID: 28742910.