

'We need to protect the brain'

Addressing the growing problem of chronic traumatic encephalopathy

Football is a multi-billion dollar industry in the United States. Scholarships to play football allow many young men to pursue an education they otherwise might not be able to afford. In states where no professional teams are based, collegiate football is king.

The National Football League (NFL) had its highest concussion tally last year: 182 such injuries reported¹ in the 2014-2015 regular season. The true rate of concussion in the NFL is likely higher, as a result of multiple factors (fear of "letting the team [or the coach] down," fear of retaliation from team owners,² etc.).

To simply call a head injury a "concussion" is a disservice to players and their family: Any blow to the head, severe or otherwise, has the potential to cause microvascular disruption in the brain; repeated blows to the head undoubtedly cause further damage.

In reality, a "concussion" is a mild traumatic brain injury (mTBI). With repeated blows, an mTBI can lead to *chronic traumatic encephalopathy* (CTE). In 2015, eighty-seven of 91 brains from autopsied former NFL players displayed some stage of CTE.³

Pathophysiology and presentation

CTE comprises 4 histological stages; Stage 4 is the most advanced. Alzheimer's disease (AD) and CTE display similarities, which suggests a separate classification of CTE-AD; the presence of amyloid β plaques correlates with (1) more severe hyperphosphorylated tau (pTau) pathology and (2) advanced stages of the disease and clinical presentations. Death tends to occur 10 years earlier in CTE-AD than in AD, suggesting that repetitive mTBI might change the deposition and accumulation of amyloid β plaques, and even accelerate the aging process in the brain.⁴

Symptoms. The case series by Omalu et al⁴ (which inspired the 2015 motion picture *Concussion*) and the case series presented by McKee et al⁵ described severe psychiatric symptoms associated with CTE:

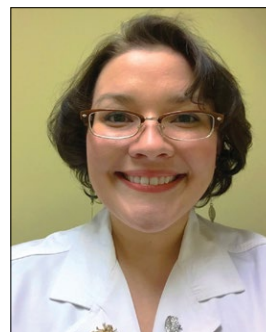
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- decreased speed of information processing
- increase in religiosity
- lack of insight
- poor judgment
- involvement in illegal activities
- substance abuse
- indiscretion
- verbal and physical abuse
- problems with interpersonal relationships
- isolation
- restlessness and hyperactivity
- somatic complaints.

The 2 groups of researchers also noted hopelessness, social phobia, anxiety, agitation, mania, labile mood, insomnia, explosivity, and suicidal ideation, attempt, and completion.^{4,5}

By Stage 4, all affected patients are symptomatic. Cognitive impairment is severe; many are described as having “severe memory loss with dementia,”⁵ “profound” inattention and loss of concentration,⁵ and dysarthria. Paranoia may develop. Mood symptoms can be severe: Approximately 31% of subjects studied have contemplated suicide; of those, 26% had “suicidal tendencies” and 14% completed suicide.⁵

Two distinct types of CTE progression are apparent:

- patients who display cognitive deficits first; they progress to dementia but live longer
- patients who display mood and behavioral symptoms first; they tend to be younger, more violent, depressed, and explosive.⁶

CTE cannot be diagnosed with imaging. There are, however, a few positron emission tomography (PET) ligands for pTau that show promise:

- **[F-18]FDDNP**, which consistently identifies pTau deposits in brains in which CTE is clinically suspected, in

the same distribution of pTau neurofibrillary tangles on autopsy.

- **[11C]DPA-713**, which detected TBI-related inflammation of neurons in 9 former NFL players in whom CTE was suspected based on the clinical presentation.

- **PiB amyloid ligand**, under investigation for use in PET neuroimaging.⁷

Casualties

In January 2016 alone, at least 3 former NFL players were found to have CTE posthumously.

Earl Morrall. Former quarterback who had a 21-year NFL career. Official cause of death in 2014 at age 79 was recorded as “complications of Parkinson’s disease.” In 2016, Stage-4 CTE was discovered on autopsy.⁸

Ken Stabler. Former quarterback for several NFL teams over 15 seasons. Died of colon cancer at age 69 in 2015. On autopsy, was found to have Stage-3 CTE.⁹

Tyler Sash. Former University of Iowa and New York Giants football player. Died in September 2015 at age 27 of an apparent drug overdose; posthumously, determined to have Stage-2 CTE. His family reported memory loss, minor fits of rage, confusion, inattention, lack of focus, and chronic pain.

Sash’s mother said, “My son knew something was wrong, but he couldn’t express it. He was such a good person, and it’s sad that he struggled so with this—not knowing where to go with it. Now it makes sense.”¹⁰ Sash played 16 years of football in all, sustaining at least 5 concussions. (“If you’ve played football, you know there are often other incidents [of head trauma],” Sash’s father said.¹⁰)

Cultural and medical mindsets about contact sports

In the United States, children as young as age 5, with a low weight limit of 35 pounds, routinely are introduced to football.¹¹ Reports of 5 high school players dying from football-related injury in the 2014 season, and 3 deaths in the 2015 season, led a St. Louis, Missouri, area school district to defund their football program entirely. The district's 2015 homecoming game was a soccer match; students and parents seemed to embrace the change.¹²

On its face, soccer seems a good alternative to football. When children are instructed to "head" the ball, however, concern arises about CTE: Mild CTE changes have been reported in 2 young soccer players, and late-stage CTE changes were seen in a retired soccer player with dementia.¹³

Perhaps most disturbing is that players who develop symptoms of CTE, or are at risk, are unlikely to seek psychiatric help. We, as psychiatric clinicians, must be diligent about questioning young patients about their extracurricular activities. It is not enough to simply ask about a history of head trauma: Ask patients about *any* blow to the head, and don't limit your questioning to whether they sustained a "concussion" during practice or play.

When speaking with adult and geriatric patients, ask about a history of playing interscholastic or collegiate contact sports, such as football, hockey, and soccer.

Is the solution to better shield the head?

That is *not* a solution: Helmets and other protective headgear appear to be insufficient to protect the brain from

traumatic injury. Perhaps keeping children from engaging in violent sports that put them at high risk of CTE later is the preventive approach that merits the most attention.

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Perhaps keeping children from engaging in violent sports that put them at high risk of CTE in adulthood merits the most attention as a preventive measure