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Chronic Traumatic Encephalopathy in Sports

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ABSTRACT

Chronic Traumatic Encephalopathy is an incurable neurodegenerative disease which is believed to be caused due to repetitive head injuries. In CTE, there is a buildup of Tau proteins in neurons which leads to the formation of Neurofibrillary tangles. These Neurofibrillary tangles prevent communication between the neurons. While the link between CTE and boxers was found years ago. In recent times, there have been links to the disease with sports such as American Football. The purpose of this paper to understand the clinical and pathological symptoms of CTE in sports players. The case studies of two players were reviewed and compared. It was found that both people suffered from, cognitive difficulties, a shift in personality and short temperedness. They also had a buildup of tau proteins in several parts of their brains, indicating that these are symptoms of CTE.

Keywords: ***Chronic Traumatic Encephalopathy, Tau Proteins, Neurofibrillary Tangles, Cognitive Difficulties***

INTRODUCTION

Over the past few years, more people are becoming aware of the long-term effects of repetitive brain injuries such as concussions, often experienced by sports players. Most head injuries experienced by athletes are minor and most athletes that have concussions recover in a few days. However, repeated head traumas can lead to players developing chronic and intensifying symptoms which are similar to dementia. For many decades, people believed that these symptoms were only found in boxers and the condition was known as "Punch Drunk Syndrome". Today, it is known that athletes such as wrestlers and football players also show similar symptoms.[1,9-10]. These players are likely to suffer from Chronic Traumatic Encephalopathy, a degenerative brain disease. Some of the signs of CTE in athletes are cognitive difficulties, depression, anxiety, and frequent mood swings as well as large accumulations of tau proteins in various parts of the brain along with brain degeneration.

Theoretical Information

Chronic traumatic encephalopathy or CTE is a terminal neurodegenerative disease caused by repetitive trauma to the head, due to the accumulation of tau proteins in neurons.[2-3] Tau proteins help assemble and maintain the structural stability of microtubules in the neurons. Repeated concussions, sub concussions or other head injuries can cause an abnormal build-up of tau proteins.[5] Tau proteins build up in neurons and astrocytes to form aggregates. These aggregates in the neurons are referred to as Neurofibrillary tangles. Neurofibrillary tangles prevent proper communication between neurons, damaging the synaptic communication between them.[6,14] These Neurofibrillary tangles can spread throughout the brain, first accumulating in the cortex before spreading to areas such as the amygdala and hippocampus.[7] Aggregates in the astrocytes are known as astrocytic tangles, are linked with damaging neurons. CTE, along with diseases such as dementia, are tauopathies. Tauopathies are neurodegenerative conditions in which there is an abnormal amount of the tau protein in the brain.[4,9-10] In CTE, the accumulation of tau proteins in brain cells has a unique pattern that separates CTE from other tauopathies.[8]

METHODS

By entering terms such as "Chronic Traumatic Encephalopathy", "CTE research papers", "CTE in NFL players", "CTE in boxers" into Google Scholar. Two papers with case studies were then selected. The papers were then checked to see if they were credible by checking the qualifications of the authors, publish dates and if the papers were reviewed. To check if they were peer reviewed, there was evidence that the articles were accepted or supported by organisations. Both the articles selected had case studies which talked about the clinical and pathological states of the patients, as

opposed to other papers which only had tables containing information or case studies which only mentioned the clinical state of the patient. These case studies are significant as they show the effects of CTE on an individual. It provides a more specific input on the life of a person with CTE, the symptoms they experienced while alive and the state of their brains. Out of the five case studies, two were regarding NFL players while 3 were regarding boxers. One of the two NFL case studies was selected due to the larger amount of information available. One of the three boxing case, a case which involved no serious accidents after their career, along with the length of their career, was selected to give more accurate findings. The reason one NFL player and one boxer were chosenwas to understand the connection of CTE in both sports.

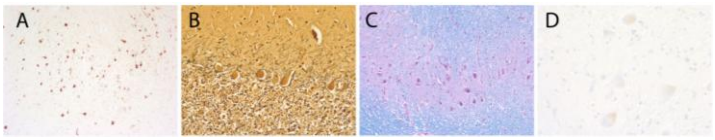
RESULTS

Case Study A

The first study follows a Caucasian male who died the age of 56 due to a myocardial infarction. The male partook in American football for a total of 17 years. He began playing American football at the age of 12 and played in the NFL for 7 years in the running and full back positions. He did not serve in the military. During his career, he was reported to suffer 20 concussions. 5 of them during his college career while 10 during his time playing in the NFL. Out of these, two concussions resulted in him losing consciousness. [12]

His family reported that he had a shift in mood in the middle of his 30s. He began to struggle with depression and sadness. He also had a prescription of Wellbutrin, a medication used to treat major depressive disorder. He suffered from a basal ganglia, an MRI report stated that he had a small lacunar infarct to the basal ganglia, a group of nuclei that ensure proper motor function. After the stroke, he suffered fatigue, slow movement, and a weakness in his leg. After 3 months, he was able to return to work as a vice president of a bank and performed well. One year before his death, he was reported to have developed feelings of hopelessness and worthlessness. He became easily frustrated and was emotionally explosive and very short tempered. He had also started frequently act out of character, become detached, listen to old music. After his stroke, he was reported to have shown an improvement in his motor symptoms. However, he was reported to suffer from mild to moderate cognitive difficulties in language, concentration, short term memory and attention. Many of these changes were reported before his stroke itself. In the functional activities' questionnaire, he did not show signs of dementia or Parkinson's. [12]

His brain weighed 1550g. The corpus callosum was thinned throughout its extent. Ventricular enlargement was also present. The substantia nigra and the locus coeruleus were pale. The microscopic findings shown in Figures A-D showed several tau-immunoreactive neurofibrillary tangles and astrocytic tangles in the perivascular, sulcal, depth, glial, cortical and subpial distribution. The man was diagnosed with stage III/IV CTE. [12]



AT8 immunostaining[11] shows neurofibrillary tangles in the sulcal depths in image A. In image B, by using Wielechowski silver stain it is shown that the cerebellum was not damaged, and the Purkinje cell layer was well populated. By using Luxol hematoxyl in and eosin in image C, it is visible that the nucleus is dentate. In image D, there are no signs of phosphorylated tau build-up in the dentate nucleus.[12]

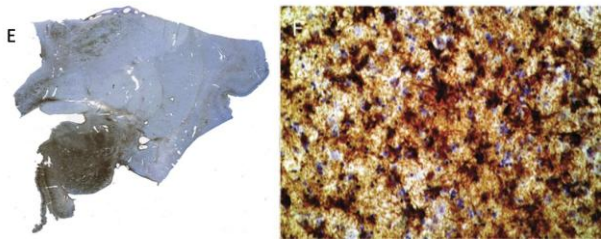
Case Study B

The second case study follows a Caucasian male boxer who died at the age of 73 due to pneumonia. He started boxing as an amateur for 9 years before becoming a professional boxer for 13 years. He took part in 48 professional matches and even obtained 2 world championships. He retired at the age of 33. [9]

It was reported that he started to become agitated, have mood swings and often forget things when he reached his late 50s. His personality shifted from being an easy going individual to a paranoid, introverted, agitated person with a lack of sympathy. In the next 2 years, he became more aggressive, easily agitated, and had developed anxiety. He had become verbally abusive to his own wife and even attempted to attack her. He also had trouble distinguishing relatives from each other. He needed neuroleptics, to prevent his short temperedness. In the next year, he experienced dizziness often and was admitted to the hospital after believing it was vertigo. When a neurological examination was conducted, it concluded that he had poor immediate and remote memory skills, was very inattentive and had poor visuospatial skills. Neuropsychological tests showed that he had cognitive difficulties of all types including language, attention, and

visuospatial skills as well as lack of learning and memory abilities. His symptoms continued to worsen over the next two years, and he began to fall often and had developed a tremor on his left hand. Tests when he was 67 showed a further decline in his cognitive abilities. By 70, he had difficulty swallowing and had a jumbled speech. His first cousin had dementia which had developed in her early 50s. [9]

His brain weighed 1220g. The frontal, temporal and parietal lobes showed signs of moderate atrophy. The corpus callosum and floor of the hypothalamus were thinned. A cavum septum that was 0.8cm in size was present. The entire hippocampus, amygdala and medial cortex were atrophic. The medial thalamus was also atrophic. The temporal and frontal white matter had prominent perivascular spaces. The substantia nigra and locus caeruleus were noticeably pale and the frontopontine fibres in the cerebral peduncle were discoloured. The microscopic findings show that tau-immunoreactive astrocytic tangles, neurofibrillary tangles, were densely accumulated in the subcallosal, dorsolateral frontal, insular, superior parietal cortices. However, they were most prominent in the medial temporal lobe. The amygdala and hippocampus had highly dense neurofibrillary tangles as well as the olfactory bulb, hypothalamus, and red nucleus. [9]



The coronal sections were immunostained (AT8) for the presence of Tau. Cresyl violet counterstains show dense deposits of the tau in the amygdala. There are extremely marked neurofibrillary tangles and astrocytic tangles that are present in image F.[13]

DISCUSSION

Both case studies A and B showed changes in their personality and behaviour. They both became more violent, aggressive, and short tempered and progressively developed feelings of hopelessness. While case A suffered greatly from depression and case B faced serious anxiety, both cases needed to be prescribed medication to help treat their mental health conditions. Both men also had cognitive difficulties for example, they had trouble remembering things, difficulties in language as well as attentiveness. However, it is important to note that the change in mood in case A was significantly earlier (In his mid-30s) as compared to case B (In his late 50s). This suggests that clinical symptoms of CTE can develop well after athletes retire or suffer from a head injury. Additionally, the fact that case study A was able to work at a bank and perform well is a sign that one can still work jobs with CTE.

The case studies both stated that parts of the brain became thinned. There were also accumulations of tau-immunoreactive astrocytic and neurofibrillary tangles. This shows that the build-up of neurofibrillary and astrocytic tangles, along with the thinning of parts of the brain, are signs of CTE. Moreover, the accumulation of these tangles is not local to one part of the brain, but to various areas. This indicates that these tangles spread and are not localised to a specific region in the brain. Additionally, both the case studies mention parts of the brain being pallor. In case study B, there are also mentions areas of the brain being atrophic, indicating that it is a sign of CTE. To conclude, the findings match the thesis statement.

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