ANSWERING QUESTIONS ABOUT

Chronic Traumatic Encephalopathy (CTE)

INFORMATION FOR HEALTHCARE PROVIDERS

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This handout presents information based on the latest science about CTE, including clinical presentation, risk factors, diagnosis, and strategies for discussing CTE with patients. While knowledge of the neuropathology of CTE has grown and media attention on CTE has increased, the scientific understanding of CTE is still in its infancy.[1] More studies are needed to fully answer questions about CTE. CDC will update this handout as more information on CTE becomes available.

Understanding CTE

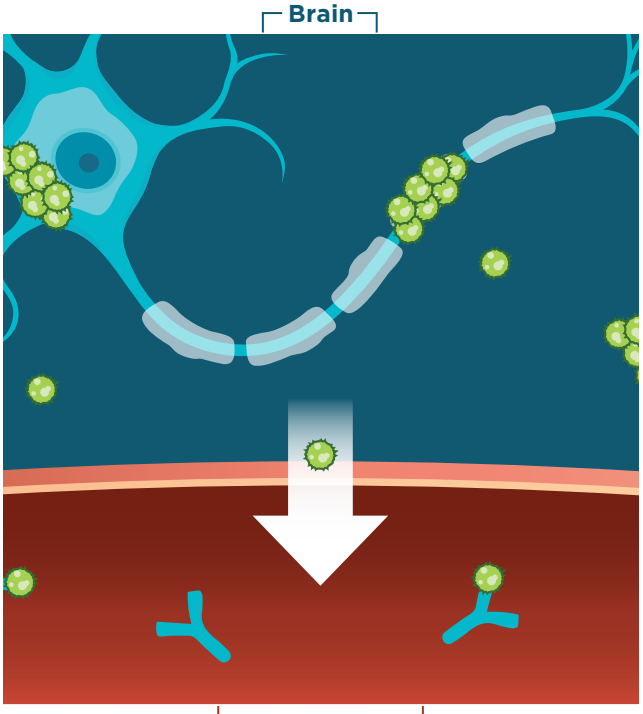
Evidence of CTE was first described almost 90 years ago when its symptoms were observed among boxers. However, the neuropathology and clinical presentation differ somewhat from how CTE is characterized today.

The etiology of CTE has not been definitively established. Most research suggests that CTE is caused in part by exposure to repeated traumatic brain injuries, including concussions, and repeated hits to the head, called subconcussive head impacts (i.e., head impacts that do not cause symptoms of concussion).[2] Research suggests that repetitive head impacts set in motion a complex set of events in the brain that may lead to CTE. The primary neuropathological changes are observed in the white matter and in the accumulation of an abnormal, hyperphosphorylated form of tau protein (p-tau). Some scientists think that p-tau may spread to other parts of the brain.[3] More studies are needed to provide definitive information on the cause(s) and the long-term effects of CTE.

Clinical Presentation

Descriptions of the clinical features of CTE are based almost entirely on interviews with family members of deceased individuals who were diagnosed with CTE after death.[4-6] Family members have reported noticing changes in thinking, feeling, behavior, and movement among people who were later diagnosed with CTE after death.[4-6] Some people diagnosed with CTE were reported to first have had problems with depression or anxiety. Some later developed memory and other thinking problems. Over time, some of these people were reported to have had mood or personality changes. Family members have

Mechanism of Tau Protein Capture



The protein tau (green) aggregates abnormally in a brain cell (blue). Tau spills out of an injured cell and enters the bloodstream (red). Research shows that antibodies (blue) can capture tau in the blood that reflect its levels in the brain.

CREDIT: Sara Moser/Washington University School of Medicine

SOURCE: National Institutes of Health Director's Blog, "Antibody Makes Alzheimer's Protein Detectable in Blood," May 2017

reported that some people who were later diagnosed with CTE had problems that became serious enough to get in the way of normal daily activities (such as social or work-related activities).[4-6]

Researchers are not certain which symptoms are directly linked to CTE. The symptoms that have been associated with CTE can also occur in individuals without CTE. Alternatively, some individuals without any known symptoms during their life have been found to have neuropathology indicative of CTE upon death. Studies that diagnosed CTE based on postmortem determination included individuals ranging from 17 to over 80 years of age, and found variations in clinical symptoms, as well as the age of symptom onset.[4-6] More research, including longitudinal studies, is needed to understand the clinical presentation of CTE, and to explore the pathological correlation with particular symptoms.[1]

Risk Factors

Early evidence suggests that individuals may have a higher risk of developing CTE if they engage in activities that increase their chances of sustaining repetitive hits to the head.[6] Researchers currently do not know the incidence and prevalence of CTE, but they do know that CTE does not occur only in athletes. Most people with head impacts or brain injuries will not get CTE, and there is no evidence to suggest that one concussion leads to CTE. CTE has also been diagnosed in individuals after death without a known history of head trauma.[7,8] Research on the role of genetics, comorbid medical conditions, sex, and other factors (such as environmental or lifestyle factors) is needed to better understand the risk factors for CTE.

Diagnosis

Patients should not be informed that they likely have CTE based on the results of experimental procedures that are not yet approved by the Food and Drug Administration. At this time, a diagnosis of CTE can only be confirmed through postmortem neuropathological examination. The pathognomonic lesion of CTE involves an irregular deposition of p-tau around small blood vessels at the base of the cortical sulci.[6] At the NIH Consensus Workshop to Define the Neuropathological Criteria for the Diagnosis of Chronic Traumatic Encephalopathy,[9] this pattern of p-tau was agreed to be distinct from other neurodegenerative tauopathies, including Alzheimer's disease and frontotemporal lobar degeneration.

Provisional research diagnostic criteria for the potential clinical manifestation of CTE, referred to as traumatic encephalopathy syndrome (TES), have been proposed.[10] However, these symptoms overlap with many other conditions, and at this time, no validated biomarkers exist for CTE. Additionally, the reliability and validity of the provisional research diagnostic criteria for TES (or similar proposed "clinical criteria"[11]) have not been established. Therefore, it is premature for practicing healthcare providers to implement TES as a diagnostic entity.

Advancing Research on CTE

In 2013, NIH launched a major program to advance research to better understand CTE, its causes, and how to diagnose it while a person is alive.

To learn more, visit the National Institute of Neurological Disorders and Stroke (NINDS) Traumatic Brain Injury Information page.

To read a report from the Consensus Meeting on CTE Neuropathology hosted by NIH, go to the Report from the First NIH Consensus Workshop to Define the Neuropathological Criteria for the Diagnosis of Chronic Traumatic Encephalopathy.

Improving Diagnosis of CTE

To fill current gaps in knowledge about the disease, researchers are studying methods to diagnose CTE during life, including through the use of experimental PET scans, MRI scans, tests of cerebrospinal fluid, and even blood tests. As part of this effort, large research studies funded by NIH are currently underway. In 2015, NINDS funded a $16M, 7-year, multi-center grant known as DIAGNOSE CTE (Diagnostics, Imaging, and Genetics Network for the Objective Study and Evaluation of Chronic Traumatic Encephalopathy).[12] Additionally, NIH has funded research to evaluate the application of the NIH consensus-based diagnostic criteria and examine clinical-pathological associations in the Late Effects of TBI (LETBI) study.[13]

Talking With Patients About CTE

Increased awareness of CTE has led to growing concerns about it among patients and caregivers. Without available methods to diagnose CTE during life, healthcare providers can educate patients about CTE (see CDC's "Answering Questions About Chronic Traumatic Encephalopathy" handout).

Patients should be reminded that the kinds of symptoms currently suspected of being caused by CTE can be experienced by people who do not have CTE, and treatments are available to help with many of these symptoms. Additionally, most adults who sustain a concussion are symptom-free within a couple of weeks, with 70% returning to baseline within 3 months.[14-17] Researchers have found that educating patients about having positive expectations for a good recovery following a concussion is associated with improved patient outcomes.[18]

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