SHORT COMMUNICATION

Chronic Traumatic Encephalopathy

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Jones K. A Brief Behavioral Activation Treatment for Depression. J Food Drug Res. 2021; 4(6):1-2.

ABSTRACT

CTE (Chronic Traumatic Encephalopathy) is a progressive neurodegenerative condition induced by single, episodic, or recurrent blunt force hits to the head and the transfer of vibration forces to the brain. Clinically, CTE manifests as a composite syndrome of psychiatric conditions, behavioral and cognitive loss, and sensory impairment, just without sensorimotor dysfunction. CTE signs can start with persistent onset of acute traumatic brain injury (TBI) after a confirmed episode of brain trauma or after a hidden period of days, months, months, and years, up to 40 years after a confirmed episode of neurological damage or the cessation of recurrent TBI.

Key Words: *Chronic Traumatic, Behavioral Treatment, Psychiatric conditions*

INTRODUCTION

Posttraumatic encephalopathy is a clinicopathologic illness caused by focal and/or diffuse, gross and/or microscopic loss of brain material after brain trauma. It can be associated to CTE. The brain of a CTE patient may appear ordinary on the surface, but microscopic signs of primary and secondary proteinopathies can be seen. Tauopathy is the predominant proteinopathy in CTE, but secondary proteinopathies include amyloidopathy and TDP proteinopathy, among others. CTE prevalence estimates in groups subject to TBI have been reported to vary from 3 to 80% throughout age categories.

On the surface, a CTE patient's brains may appear normal, but microscopic evidence of primary and secondary proteinopathies can be found. The most common proteinopathy in CTE is tauopathy, however there are also secondary proteinopathies such amyloidopathy and TDP proteinopathy. CTE help estimate in people who have had a TBI range from 3 to 80 percent, depending on their age group. While CTE normally manifests clinically over a long period of time, certain patients with CTE may not have the typical extended lag phase before developing clinical symptoms. Microscopic tissue evaluation with histochemical and immunological tissue analysis remains the final and conclusive diagnosis of CTE. Furthermore, employing 2-(1-[(2-[F-18]fluoroethyl)(methyl)amino]- 2-naphthylethylidene)malononitrile-positron emission tomography (FDDNP-PET), in vivo premortem presumptive diagnosis of CTE has emerged recently. In live rains, FDDNP-PET detects simultaneous tau tangle and amyloid plaque formation. Premortem clinical diagnosis of CTE in living individuals utilising radioligands such as FDDNP is a positive message for the management of CTE in the nearish term.

CTE is an unique neurodegenerative disorder with distinct histomorphologic and proteinopathic phenotypes that differ from those of the most common neurodegeneration such as Alzheimer's disease (AD), Lewy body cancer (LBD), temporal lobe lobar deterioration, vasculature brain injury, and cerebrovascular disease [10, 11]. CTE, like other degenerative illnesses, can happen at the same time as one or more other forms of neurodegenerative disorders in the same person. CTE is a disease that progresses over time, resulting in chronic neuropathologic and proteinopathic alterations. Early or end-stage CTE in senior patients can have histomorphologic and proteinopathic characteristics that resemble Alzheimer's disease, it may be difficult to distinguish CTE from AD with an acceptable degree of professional confidence in such old patients. Previously, a small number of doctors and authors, particularly those who work or are affiliated with sports organisations, have questioned or disputed CTE's presence. CTE, on the other hand, has remained a well-established notion in medical science for generations. Shock to the brain becomes a well established medical principle that can result in irreversible brain damage that manifests as a variety of progressive symptoms. CTE has been recognised, documented, and named using a number of terminology over the years.

PTE and CTE are two different disease entities that both develop as a result of a TBI. While these diseases are separate, they can coexist in the same person as comorbidities. With brain trauma, PTE is described as a clinicopathologic illness caused by focal and perhaps diffuse, gross and/or microscopic loss of brain tissue. Blisters and lacerations of the brain, secondary traumatic hypoxic-ischemic cerebral injury, stress-related anoxic glutamatergic necrosis, lesions and loss of function,

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traumatic cranial haemorrhages, compression of the brain by trauma - related neuraxial and spinal cord compression haemorrhages, and stress-related cerebral herniations and necrosis are all examples of PTE modifications. Injuries caused by PTE destruction of brain tissue, cav and neurodegeneration tissue, anisomorphic astrogliosis, activation of microglia, and infiltrate by histiocytes (foamy or non-foamy) are all common effects (pigment-laden histiocytes), resulting in severe brain damage. Future study may look into these obscure subcellular & cellular mechanisms in the hopes of earlier treatment, protection, or possibly cure, as well as avenues for reducing protein quantities.