G16 Severe Degenerative Tauopathy Following Closed Head Injury: Alzheimer's Disease vs. Chronic Traumatic Encephalopathy

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After attending this presentation, attendees will be able to identify the significance of traumatic brain injury on the development of spreading tauopathy and its potential as a pathogenic mediator of neurodegenerative disease.

This presentation will impact the forensic science community by highlighting the potential impact of a closed head injury in causing or accelerating the progression of chronic neurodegeneration, highlighting the role of tau protein as a toxic mediator of disease, and raises the issue of qualitative differences between chronic traumatic encephalopathy, aging, and Alzheimer's disease.

The role of head trauma in neurodegenerative disease has been recognized at least as far back as the 1920s. However, the role of head trauma in producing chronic disease has become a significant public health concern in recent years in light of the association between contact sports such as football, soccer, and boxing, as well as blast injuries in armed conflicts, with progressive degenerative tauopathy now termed chronic traumatic encephalopathy. History of closed head injury is also a known risk factor for Alzheimer's disease and confers an approximate twofold relative risk for development of disease. This presentation discusses the case of a 55-year-old woman who suffered a fall at a department store resulting in multiple cerebral contusions. Following the fall and the closed head injury, she developed frequent seizures and increasing confusion. She expired three years after the closed head injury. At autopsy, she had remote contusions with encephalomalacia involving the orbitofrontal regions bilaterally, the bilateral temporal poles, and the right posterior medial temporal lobe. Hydrocephalus *ex vacuo* and a cavum septum pellucidum were also present. Microscopic examination revealed extensive neurofibrillary degeneration using phospho-tau (AT8) immunohistochemistry, qualifying for advanced/end-stage Alzheimer's disease (Braak stage VI). Extensive amyloid plaques throughout the neocortex were also noted on histology. The pattern of phospho-tau deposition overlapped between that seen in chronic traumatic encephalopathy (superificial layer, perivascular, glial tau), and the pattern seen in Alzheimer's disease. This case highlights a potential role of head trauma in accelerating neurodegeneration and raises the issue of spreading tauopathy as a pathogenic mediator of neurodegenerative disease.

Traumatic Encephalopathy, Tauopathy, Alzheimer's Disease

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