H42 Axonal Injury Is Detected by ß-Amyloid Precursor Protein (ßAPP) Immunohistochemistry in Near Instantaneous/Rapid Death From Head Injury Following Road Traffic Collision (RTC)

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Learning Overview: After attending this presentation, attendees will better understand that traumatic axonal injury can be detected in short survival if the brain looks unremarkable, the pathophysiology of axonal injury, and the limitation of diagnostic immunohistochemistry.

Impact on the Forensic Science Community: The axonal injury can be detected in the brain in those who died rapidly or near instantaneously following a fatal RTC. It is proposed that the early accumulation of ßAPP is probably due to the high magnitude of loaded forces in head injury resulting from RTC in this cohort, which could possibly cause higher proportions of primary axotomies and/or that the stretching axonal injuries are more severe causing quicker alteration in the axonal cytoskeleton. This presentation will impact the forensic science community by examining how this study's result is expected to have implications on the timing of head injuries in medicolegal practice, but caution is recommended to not overinterpret the results, which require close correlation with autopsy findings, circumstances of the incidence, and additional investigation.

Introduction: Timing of traumatic axonal injury is one of the challenging tasks in forensic neuropathology. It depends on assessing the morphological evaluation of axonal injury via demonstration of axonal injury by Hematoxylin-Eosin (H&E) stain, silver stain, and, more accurately, by immunostaining for βAPP. The accumulation of βAPP caused by axonal injury is an active energy-depended process thought to require blood circulation and is therefore closely related to the patient's post-injury survival time. The earliest reported time that axonal injury can be detected by immunohistochemistry following traumatic brain injury by autopsy brain examination is currently 35 minutes.

Goal: To investigate if βAPP staining for axonal injury can be detected in the autopsy brain of patients with near instantaneous/rapid death following RTC.

Material and Methods: This study involved retrospective examination of 49 deaths following RTC and one fatal single-occupancy aviation crash where there was reliable information concerning the time between the incident and the death. Thirty-seven patients (Group 1) died virtually instantaneously or very rapidly at the scene of the incident. A further three patients died after 30 minutes to 11 hours (Group 2) and eight patients died between 2 and 31 days after the RTC (Group 3). The brains from patients who died of instantaneous death due to sudden unexpected death in epilepsy (Group 4) were used as a non-traumatic control group. The brains were comprehensively examined, and a full set of tissue blocks from the cerebral cortex, white matter, corpus callosum, basal ganglia (including the internal capsule), cerebellum, and brainstem were sampled in all cases and immunohistochemically stained with βAPP.

Results: The βAPP immunoreactivity is demonstrated in minute amounts of variable pattern and low frequency in 35/37 brains of Group 1, in more intensity and frequency in 3/3 brains of Group 2, and 7/8 brains from Group 3, compared with no βAPP immuno reactivities in all four brains from the control group (Group 4).

Conclusion: Axonal injury can be detected in the brain of those who died rapidly or near instantaneously following fatal RTC. The very early accumulation of βAPP is possibly due to higher proportions of primary axotomies and/or that the stretch axonal injuries are more severe and cause quicker alteration in the axonal cytoskeleton.

Traumatic Axonal Injury, ßAPP, Short Survival

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