H76 Postmortem Evaluation of Mild Traumatic Brain Injury (Concussion): Importance and Relevance

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After attending this presentation, attendees will: (1) understand the pathophysiology of mild Traumatic Brain Injury (mTBI); (2) understand the medicolegal implications of mTBI, (3) understand appropriate case selection for postmortem mTBI evaluation; and, (4) understand techniques used to evaluate for mTBI.

This presentation will impact the forensic science community by explaining the importance and relevance of mTBI evaluation in the medicolegal setting, expanding upon the predominantly animal-based research, and increasing the ability of forensic pathologists to appropriately evaluate for mTBI.

MTBI, also referred to as concussion, is caused by mechanical trauma. The clinical features of mTBI include possible transient loss of consciousness and/or amnesia surrounding the event, a Glasgow Coma Scale between 13-15, and only rare imaging abnormalities.[1] The pathophysiology of concussion involves a predominantly excitatory neurotransmitter cascade with variable long-term sequelae.[2,3] Although animal studies have suggested structural changes following mTBI, trauma in humans resulting only in concussion rarely leads to death. Two recent cases elucidate the existence and extent of structural Central Nervous System (CNS) changes in humans following concussion and suggest the importance of postmortem evaluation for diffuse Traumatic Axonal Injury (dTAI) in select cases.

In the first case, a helmeted 72-year-old female bicyclist was struck by a motor vehicle traveling at approximately 30mph. Clinical evaluation revealed anterograde amnesia, a likely transient loss of consciousness, and Glasgow Coma Scale of 14 (of 15) which was 15 (of 15) upon arrival to the emergency department, consistent with mTBI. She sustained clavicle, rib, transverse process, and pelvis fractures and required respiratory support for a "flail chest." Head Computed Tomography (CT) was negative for acute pathology. She refused surgical intervention for her traumatic injuries and died two days later. At autopsy, petechial hemorrhages were extensive within white matter. Histological examination revealed organizing fat emboli. Immunohistochemical evaluation with Amyloid Precursor Protein (APP) unexpectedly revealed distinct axonal swellings in regions characteristic of dTAI.

In the second case, a 77-year-old female pedestrian was struck by a motor vehicle traveling between 20mph and 30mph. She was amnestic toward the event and had a brief loss of consciousness. She suffered head trauma and a scalp laceration, but maintained a Glasgow Coma Scale of 15 (of 15) and was diagnosed with a concussion. She sustained rib, clavicle, and pelvic fractures. Head CT revealed a temporal bone fracture, focal subarachnoid and small subdural hematoma without midline shift. She was anti-coagulated at the time of her injury, so thus was followed with serial head CT scans which unfortunately showed subdural hematoma expansion and midline shift. In spite of medical management, she became globally encephalopathic and died five days after her injury. At autopsy, neuropathological examination confirmed the clinical findings and demonstrated dTAI in addition to changes associated with increased intracranial pressure.

Academic and public interest in concussions is growing. While the sequelae of repetitive mTBI are becoming better understood, research demonstrating structural changes following a single episode of mTBI is predominantly animal-based.[3-8] These two cases show similar findings in humans, supporting the co-existence of both metabolic and structural post-concussive pathology. This is relevant to the medicolegal community for numerous reasons. The finding of structural damage upon neuropathological evaluation may be key to an investigation by elucidating incongruent details. For example, it allows postmortem evaluation for brain injury that may be unexpected given limited gross and/or intracranial pathology but prohibited the decedent from escape or self-defense. Alternatively, it may be relevant when considering if the injury could have impacted decision making. Finally, this microscopic finding enables postmortem correlation with imaging techniques designed to evaluate for mTBI during life.[9]

Appropriate sampling for histology and/or stock tissue is straightforward but must be thorough.[10] Although gross findings are unlikely in mTBI, postmortem evaluation for dTAI following known or suspected concussion samples the same white matter regions as in severe TBI. The posterior corpus callosum just anterior to the splenium is particularly high-yield.[9] Cerebral hemispheric white matter, posterior limb of the internal capsule, and the dorsal brain stem including pons and superior cerebellar peduncle(s) should also be sampled. Both Hematoxylin-Eosin (H&E) and the APP immunostains should be requested on these sections. In addition to highlighting TAI, APP demonstrates Vascular Axonal Injury (VAI) and other trauma sequelae. Experience is needed to differentiate these processes histologically; neuropathology textbooks and colleagues are helpful resources.

Postmortem evaluation for dTAI following concussion can yield information that is highly relevant to the cause and manner of death. Although the demonstration of structural changes of concussion is relatively new, a lower threshold for dTAI sampling is likely warranted. Forensic pathologists and neuropathologists should consider evaluating for dTAI when antemortem mTBI is reported or suspected as part of a complete medicolegal examination.

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Forensic Neuropathology, Traumatic Brain Injury, Concussion

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