Traumatic brain injury

Traumatic brain injury (TBI) is an alteration in brain function, or other evidence of brain pathology, caused by an external force. The majority of TBI cases are of mild severity (mild TBI (mTBI)) with a Glasgow Coma Scale (GCS) score of 13 to 15. Clinical signs may not be observed in all cases of mild TBI. Symptoms are essential for diagnosis.

In addition to signs and symptoms at presentation, mild TBI may have other consequences and complications. Among them, the presence of intracranial lesions (like intracranial hemorrhage, fractures, or brain oedema) reflects structural damage following a traumatic event. Such lesions, dominated by hemorrhages, are identified in the acute phase in approximately 8% of cases. Complications, mostly requiring surgery, occur in about 1% of cases. One patient from 1,000 arriving at the hospital with a mild head injury will die (0.1%).

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Computed Tomography (CT) is a reference method to detect these intracranial complications, mostly acute intracranial hemorrhage. Management strategies are becoming more and more focused on selective CT use to effectively use health care resources. Selective management has received more attention following reports of increased cancer risk from CT scans, estimated at 1 in 5,000 to 10,000 for a single head CT scan in young adults. Selective CT use is based on risk assessment of detecting intracranial lesions in patients with suspected mTBI .

Blood-based brain biomarkers, including GFAP and UCH-L1, have been extensively studied for prediction of absence of intracranial lesions on head CT-scan in mild TBI.

GFAP is an astroglial biomarker of reactive gliosis and is found in the astroglial skeleton of both white and gray brain matter. GFAP is almost exclusively expressed in astrocytes, including in spinal cord astrocytes.UCH-L1 is a neuronal brain injury biomarker found in high abundance in neurons and has previously been used as a histological biomarker.

UCH-L1 is highly expressed in central nervous system (CNS) but also in neurons of the peripheral nervous system (PNS).

Following brain injury, due to astrocytes and/or neurons disruption, it is believed these proteins are released into the cerebrospinal fluid (CSF) and into the peripheral blood, via a blood-brain barrier (BBB) rupture. GFAP and UCH-L1 were evaluated in mTBI in adults in numerous studies, separately and in association, and their levels correlate with acute lesions on the head CT-scan. As UCH-L1 and GFAP appear to reflect different cell injury mechanisms, the combination of the two biomarkers may be more useful than either biomarker in isolation for predicting intracranial lesions on CT scanning. In mTBI both GFAP and UCH-L1 are detectable within 1 hour of injury. GFAP peaks at 20 hours after injury and slowly declines over 72 hours. GFAP levels are still detectable at 168 hours (7 days) after injury. UCH-L1 rises rapidly and peaks at 8 hours after injury and declines rapidly over 48 hours.

In patients with traumatic intracranial lesions on CT, GFAP and UCH-L1 levels are significantly elevated compared with those without lesions. Therefore, serum GFAP and UCH-L1 concentration below a pre-established cut-off in patients with mild head trauma may represent a reliable and objective indicator for the absence of acute intracranial lesions.