H48 Expression of Heat Shock Protein 70 (HSP70) After Human Brain Injury in Different Post-Traumatic Intervals

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After attending this presentation, attendees will better understand immunoistochemestry as it applies to blunt trauma.

This presentation will impact the forensic science community by increasing knowledge about brain injury and its correlation with the expression of HSP70, which is an important issue in forensic cases.

Traumatic Brain Injury (TBI) is a very frequent cause of death after traffic accidents or assaults. The response to TBI is complex and induces various biological pathways in all brain regions that contribute to bad outcomes.[1] TBI frequently leads to brain edema and hemorrhage due to disruption of the Blood Brain Barrier (BBB). This promotes the neurotoxic cascade such as the energy-dependent ion pumps failure, acidosis, membrane depolarization, the influx of calcium and sodium, the release of glutamate, and the activation of apoptosis and inflammation.[1] The inflammatory response, after TBI, involve microglial activation, leukocyte recruitment, and upregulation of cytokine secretion.[2]

The Heat Shock Proteins (HSPS) comprise a highly conserved family of Adenosine Triphosphate (ATP) -dependent, cytosolic chaperones that function primarily in facilitating protein folding, degradation, complex assembly, and translocation, consequently preventing harmful protein aggregation.[3] Heat shock proteins are induced by many stressful stimuli, including a variety of central nervous system insults, such as cerebral ischemia, neurotoxin exposure, and when normal cellular processes are interrupted by stress.[4] The 70kDa inducible HSP (HSP70) is increased in brain vessels following experimental TBI and its induction can protect against a variety of insults including brain ischemia, trauma, and hemorrhage.[5-8] Finally, a human study indicates that the highest expression of HSP70 is found at 0h after brain contusion; the intensity of HSP70 staining decreases to the minimum at 24h after TBI, then increases gradually.[9]

The goal of this research is to assess the different expression of HSP70 immunohistochemically and molecularly in individuals with different posttraumatic intervals. This data will allow this study to determine if there is a linear relationship between the expression of this protein and the time of survival.

This study includes the reference group (control) and the TBI group. In the reference group (n=30), there were no brain contusions and death had occurred within 0-30min as a result of other reasons (myocardial infarction, pulmonary embolism, ruptured aneurysm). The TBI group (n=30) comprised individuals with a frontal cortical contusion zone and cranial traumata as the cause of death. The survival time in the TBI group varied between 0-30min, 30min-2h, 4h-12h, 24h-48h. The causes of injury in the TBI group were falls, household accidents, and vehicle accidents.

In each group, samples of brain tissue (middle-brain and brain parenchyma) were fixed with 10% formaldehyde for more than one week and embedded in paraffin; four to five micron-thick sections were stained with hematoxylin and eosin and by immunohistochemistry for HSP70 antigen.

A preliminary analysis, after hematoxylin and eosin staining, performed on the control group and on the TBI group (0min-30min), showed no difference in the distribution of cerebral edema. Edema thus is not dependent on the factor that induces stress (anoxia, trauma, etc.). The preliminary investigation of immunohistochemistry has shown a correlation between the application time of the trauma and the expression of HSP70.

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