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2105

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SHIFT 5 - TRAUMATIC BRAIN INJURY

Use of ERP markers in patients with whiplash and concussion with cognitive complains

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Background: Deficits in attention, executive function and memory are frequently found in patients with history of Traumatic Brain Injury(TBI). Electrophysiological markers such as Event-related Potentials (ERP) may be helpful in revealing cognitive deficits not detected in routine neuroimaging studies.

Objective: We used an automated Event Related Potentials (ERP) technique to assess cognitive complains in patients with Traumatic Brain Injury (TBI), and investigate whether neurophysiological testing is a more sensitive measure of abnormalities than standard neuroimaging techniques in this population.

Patients and Methods/Material and Methods: Patients with a history of subacute and chronic TBI, mild to moderate-severe injuries and cognitive complains underwent an auditory oddball ERP paradigm in conjunction with neuro imaging studies.

Results: ERP testing frequently showed decreased amplitude and/or prolonged latency for some of the ERP markers in our group of patients, suggesting deficit in executive function, attentional impairment and/or slowing of processing speed.

Conclusion: Analysis of ERP data provides valuable information in patients with cognitive complains after TBI, even in mild cases of whiplash injury, particularly when standard neuroimaging studies are non revealing. Further data in a larger population and longitudinal study are suggested.

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SHIFT 5 - TRAUMATIC BRAIN INJURY

Human umbilical cord mesenchymal stem cells improve outcomes of traumatic brain injury by normalizing inflammatory genes in rats contused cortex

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Background: It has been well documented that neuroinflammation plays a pivotal role in the pathogenesis of traumatic brain injury (TBI). Several cytokines and chemokines in the injured cortex are involved in the development of neuroinflammation after a TBI. It is unknown whether umbilical cord mesenchymal stem cells (HUMSC) improve outcomes of TBI via modulating neuroinflammation.

Objective: We aim to ascertain several inflammatory genes in the injured cortex.

Patients and Methods/Material and Methods: We performed quantitative real-time PCR (qPCR) to quantify the genes encoding 84 cytokines and chemokines in the injured cortex of rats caused by lateral fluid percussion. Cerebral contusion and neurological dysfunction were verified by TUNEL stainings and mNSS, respectively.

Results: First, we observed that systemic administration of HUC-MSC significantly attenuates the brain contusion and neurological motor deficits in TBI rats. Second, qPCR array analyses revealed that TBI upregulates a mixture of both pro-inflammatory/ neurodegenerative genes and anti-inflammatory/ neuroregenerative genes in the injured cortex. Therapy in HUC-MSC, in addition to attenuating brain contusion as well as neurological deficits, significantly inhibited five pro-inflammatory/ neurodegenerative genes but augmented three anti-inflammatory/ neuroregenerative genes in the injured cortex. The former genes contain *pf4, il7, ccl19, spp1, and ppbp*, whereas the latter genes contain *gpi, bmp2, and bmp4*.

Conclusion: It can be derived from the present results that MSCs therapy may attenuate neurological injury by inhibiting several cortical genes driving pro-inflammatory neurodegeneration processes and augmenting several cortical genes driving anti-inflammatory, neuroregenerative events.

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SHIFT 5 - TRAUMATIC BRAIN INJURY

Calcium release-activated calcium channel inhibition is effective for neuroprotection against inflammation by brain injury

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Background: Calcium release-activated calcium (CRAC) channel contributes to immune response related with microglia activation. The roles of CRAC channel in brain have not been examined.

Objective: The purpose of this study is to clarify it and assess the outcome for CRAC channel inhibition.