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The effect of spinal cord injury on beta-amyloid deposition and inflammation in TgCRND8 mouse model of Alzheimer's disease

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Traumatic BRAIN INJURY (TBI) is a risk factor for developing Alzheimer's Disease (AD) in humans. Mechanistically, TBI induces AD-like amyloid beta (Aβ) plaque pathology within hours of injury. However, the effect of TBI on the acute onset and progression of Aβ plaque pathology is not replicated in various experimental studies. It has postulated that Aβ plaque pathology induced by TBI may result from massive accumulation of AMYLOID PRECURSOR PROTEIN (APP) in damaged axons. This study utilized spinal cord transection to examine whether such extensive axon damage may acutely induce the onset and progression of Aβ plaque deposition at 3 days post-injury in the TgCRND8 transgenic model of AD at the age of 3- and 20-month-old. After injury, widespread axonal pathology indicated by intra-axonal co-accumulations of APP and its product, Aβ, was observed in perilesional region of the spinal cord in TgCRND8 mice at the age of either 3 or 20 months as compared to WT mice. However, no Aβ plaques were found in 3-month-old TgCRND8 mice. The 20 month-old TgCRND8 mice demonstrated spinal cord transection significantly reduced Aβ plaque load in the lesion site compared to sites distant to the injury and the corresponding area in sham mice ($p<0.01$). The lesion site of spinal cord area was occupied by CD68 positive macrophages/activated microglia in injured mice compared to sham animals. Therefore, our present data may not support the supposition that plaque formation may be correlated with amount of axonal pathology.

Biography

Qiuju Yuan has research focus on the following aspects: To understand mechanisms underlying neuronal death and regeneration after axonal injury; to explore therapeutic approached for neuronal injury and neurodegenerative diseases such spinal cord injury, peripheral nerve injury, stroke, multiple sclerosis and Alzheimer's disease.

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