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Neurochemical changes in chronic traumatic encephalopathy

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Chronic traumatic encephalopathy (CTE) is a progressive neurodegenerative disease associated with repetitive concussive and sub-concussive head injuries. This disease has become a popular topic due to its close association with sports involving repetitive and high impact collisions such as football and hockey. Post-mortem pathological analysis has defined CTE as a tauopathy with neurofibrillary tangles (NFTs) accumulating in both neurons and astrocytes. It is believed that these pathological abnormalities result in progressive deficits in cognition, behavioral, mood and motor function. CTE has been neuropathologically classified into four stages according to the location and spread of NFTs. As early as the stage II of the disease, NFTs are noted to involve deep-seated groups of neuro-chemically diverse brain structures (cholinergic, dopaminergic and serotoninergic). These deep and discrete neuronal groups often show few NFTs and there is no obvious evidence of neuronal death. However, the extent of the loss of their projections to their main targets is not known. Any loss of these biochemically varied projections to the cortex would seriously affect the function of cortical neurons in general and lead to neurological and psychiatric deficits. In this study we have examined the fiber densities of cholinergic (ChAT+), serotonergic (5HT+) and dopaminergic (Th+) fibers in the cortex of our cohort of young hockey players with neuropathologically proven CTE. Our results indicate significant deficits in all three systems in different cortical areas (with and without tau pathology). These findings indicate that multiple head traumas induce important brain biochemical imbalances that extend beyond the areas of tau deposits and can be suspected to cause more directly the behavioral and mood disorders associated with CTE.

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Is anti-RA 33 has specificity and sensitivity in diagnosis of early rheumatoid arthritis?

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Rheumatoid arthritis is a chronic inflammatory disease with uncertain etiology. It is characterized by symmetric polyarthritis in peripheral joints. Its diagnosis is based on clinical findings and serologic tests. Diagnosis of RA is based on its typical signs and symptoms, with laboratory and radiographic confirmation. However, its diagnosis is rarely conclusive in early course of the disease. So, its early diagnosis could be difficult. The present study was designed to evaluate the role of anti-RA33; an auto-antibody against RA33 in early diagnosis of the disease. Forty three patients with RA who had been visited in a rheumatology clinic were randomly selected. Their disease has been diagnosed by a rheumatologist. They served as the case group. 55 persons were also chosen from healthy individuals who had attended in other clinic. They served as control. Their age and sex were matched with the case group. The level of anti -RA33 was compared between the groups. It was measured by ELISA. The cut-off point curve of its level was drawn, in which the sensitivity, specificity, and negative and positive predictive values of the test have been calculated, as well as its age correlation, and its association with RF. Anti-RA33 and RF titers were measured in their blood sample using standard methods. According to findings, RF and anti-RA33 titers had significant correlation in the case group (p=0.015). Anti-RA33 test had 98% sensitivity, 20% specificity, 50% positive predictive value, and 90% negative predictive value. Anti-RA33 could have diagnostic and prognostic importance in diagnosis and evaluation of patients with RA, and its differentiation from other small joint disorders, particularly when the other serologic tests are negative. The current study showed that anti-RA33 test can be used as diagnostic clue for recognition of RA.

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