

Vascular Dementia 2019: Iron homeostasis is changed in neurodegenerative diseases

Manolov Victor,

Medical University Sofia, Bulgaria

Alzheimer's disease (AD) is characterized by deposition of amyloid plaques of amyloid- β chelating peptide with transition metal ions (Cu^{2+} , Zn^{2+} и Fe^{3+}). The binding of Cu^{2+} and Fe^{3+} leads to toxic chemical reactions; a change in the oxidation of two metals, that leads to H_2O_2 production in the presence of transition metals and finally gives toxic free $\text{OH}\cdot$ radicals. Parkinson's disease (PD) is characterized by the deposition of inclusion bodies (Lewy bodies) of α -synuclein in substantia nigra, which is ubiquitously expressed in the brain and mutations in this protein are presented in familial forms of AD. 19 Alzheimer's disease patients and 23 Parkinson's disease patients were enrolled in this study. They were evaluated for serum iron, copper, selenium, zinc and hepcidin levels. Superoxide dismutase (SOD) and glutathione peroxidase (GPX) were measured as oxidative stress markers. Hepcidin and SOD were measured by ELISA methods. Serum Fe was evaluated by Ferrozine method; Cu and Zn were quantified by FAAS. The results from AD and PD patients were compared to age and gender matched healthy controls. Pearson's correlation and Student's paired t-test were used for statistical analysis of established results. We found statistically significant elevated serum iron, copper and zinc results in AD and PD patients (aver. $49.6 \pm 2.4 \mu\text{mol/l}$, $45.4 \pm 2.9 \mu\text{mol/l}$, and $43.7 \pm 1.1 \mu\text{mol/l}$) compared to healthy controls ($20.1 \pm 1.9 \mu\text{mol/l}$, $23.3 \pm 2.0 \mu\text{mol/l}$, and $14.8 \pm 2.1 \mu\text{mol/l}$); $P < 0.01$. Hepcidin concentrations were increased in AD and PD cases (aver. $71.1 \pm 4.1 \mu\text{g/l}$) compared to control group ($19.9 \pm 2.2 \mu\text{g/l}$); $P < 0.001$. SOD levels were decreased in Alzheimer's and Parkinson's diseases (aver. $10.1 \pm 1.1 \mu\text{g/ml}$) compared to normal values in healthy controls ($22.2 \pm 1.4 \mu\text{g/ml}$); $P < 0.001$. The expected contribution from our study is practical introduction of quantification of serum hepcidin as a potential marker for early diagnosis of impaired iron homeostasis, leading trace element in the pathogenesis of neurodegenerative diseases. brain and mutations in this protein are presented in

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