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Neurodegenerative diseases: A new view through iron homeostasis

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Alzheimer's Disease (AD), Parkinson's Disease (PD) and Amyotrophic Lateral Sclerosis (ALS) are part of neurodegenerative diseases. Their development is slow, progressive and most common is seen in elderly patients. Hepcidin leads to iron deposition in neuronal structures as a result from oxidative stress. We tried to evaluate serum hepcidin levels in neurodegenerative diseases and search for connection to disturbed iron homeostasis. 23 patients with AD, 17 cases with PD and 13 with ALS were included, 24 males (45.3%). They were clinically and neurologically reviewed, EMG, IMT and ABI were measured. They were evaluated for routine biochemical parameters and additional serum hepcidin were quantified. AAS, nephelometric, ELISA and statistical methods were used during analyzes and obtained results interpretation. All results were compared to age and gender matched healthy controls. We found statistically significant elevated serum hepcidin in patients with neurodegenerative diseases (AD: 47.9±3.1 μg/L, PD: 49.8±5.1 μg/L, ALS: 53.8±4.9 μg/L) compared to healthy controls (19.9±4.1 μg/L); $P<0.001$. Serum hepcidin correlates negatively to glutathione peroxidase and superoxide dismutase changes in evaluated neurodegenerative diseases patients ($0.9<r<0.7$, $P<0.05$). Our findings support role of serum hepcidin quantification as a marker for iron deposition in neurodegenerative diseases and might bring new view to early therapeutic implementation in Alzheimer's disease, Parkinson's disease and amyotrophic lateralSclerosis.

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