The role of advanced glycation of proteins in the etiopathogenesis of multiple sclerosis

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Background: Advanced glycation end products (AGE) are involved in the pathogenesis of many diseases, including neurodegenerative diseases such as multiple sclerosis (MS). It is suggested that many xenobiotics may initiate abnormal immune response in individuals with certain genetic predispositions. Many of these compounds can also adversely affect the state of redox balance of the body, exacerbating radicalization and reducing antioxidant defense mechanisms. This can be a bridge linking the etiopathogenesis of MS to the processes of advanced glycation of proteins in the human body. The aim of the study was to evaluate the intensity of protein glycemia in MS and their possible involvement in disease activity. Material and methods: The study involved an authoritative questionnaire which helped to survey a group of MS patients from the Upper Silesian region (n=52; mean age-37.9±9.4 years); control blood samples came from healthy volunteers (n=40; age-41.1±10.4 years). Concentrations of selected parameters of advanced glycation of proteins: AGE, carboxymethyllysine (CML), carboxyethylolysin (CEL), and their soluble receptor (RAGE) in sera of patients and controls were determined by immunoenzymatic method (ELISA) using commercially available kits. Results: MS is accompanied by a statistically significant increase of protein glycemia. The duration of the disease and the degree of motor impairment do not appear to affect the progression of the glycation processes. However, the disease process associated with MS alters the correlation between individual protein glycation products, particularly the correlation between CML and CEL concentrations. Conclusion: Advanced glycation of proteins should be taken into account in the etiopathogenesis of MS.