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The golgi apparatus in Alzheimer's disease

Stavros J Baloyannis
Aristotelian University, Greece

Alzheimer's disease (AD) is a progressive devastating disorder of the central nervous system, involving mental faculties, characterized by profound memory loss, learning inability, loss of skills, behavioral changes, resulting eventually to a vegetative state. The pathogenicity of AD, remains still unknown, in spite of the continuously ongoing research efforts. The implication of Aβ peptide, APP, the hyperphosphorilation of tau protein and the synaptic pathology, may contribute substantially in plotting the neuropathological pattern of the disease, which consists of accumulation of neuritic plaques in the cortex and the subcortical centers, neuronal loss, synaptic changes and cytoskeletal alterations, which are the typical morphological hallmarks of AD. However, these findings are unable to explain sufficiently the innermost mechanisms of the pathogenesis of the disease. Morphological alterations of the neuronal organelles, mostly concerned the mitochondria, have been described in AD, from the ultrastructural and histochemical point of view. In the present study it has been attempted to describe the morphological alterations of the Golgi apparatus (GA) in post mortem material of ten early cases of AD, studied in electron microscopy. It is known that GA plays an important role in glycosylation, sulfation, and in proteolytic processing and trafficking of proteins, which are synthesized in the endoplasmic reticulum of neurons and glial cells. The hyperphosphorylation of tau protein, a phenomenon tightly associated with the pathogenesis of AD, is also related with the pathophysiology of GA. In eight early cases of Alzheimer's disease we observed a marked fragmentation of GA in the perikaryon of polyhedral and pyramidal cells of the hippocampus, the large triangular neurons of the frontal cortex as well as the Purkinje cells of the cerebellum. The fragmentation of Golgi apparatus may be related to the action of γ-secretase on APP and it may be associated with the alterations of microtubules, the loss of dendritic branches and spines and the alterations of the dendritic arbors, which is a prominent phenomenon even in the early cases of Alzheimer's disease closely related with the decline of cognition.

Biography

Stavros J Baloyannis was born in Thessaloniki, Greece. He is a Honorary Member of the Academy of Hellenic Air Forces. President of the Society for the amelioration of the quality of life in chronic neurological diseases. President of the Orthodox Medical Association for support of health and medical education in Africa. He was visiting professor in Tufts University, Democretian University, Aristotelian University, School of Theology, Aristotelian University, School of Philosophy. Author of 28 textbooks on Neurology, Neuropathology, Neuropsychology and of 680 papers, published in Greek and International Journals on Neurology, Neuropathology, Neuroimmunology and Neuroethics. Head of the 1st Department of Neurology, Aristotelian University for 20 years (1992-2011). Emeritus Professor of Neurology, Aristotelian University, Thessaloniki, Greece. Director of Research Institute for Alzheimer's disease.

sibh844@otenet.gr