230. RELATIONSHIP BETWEEN CLINICAL STAGES AND DISTRIBUTION OF NEUROFIBRILLARY TANGLES IN ALZHEIMER'S DISEASE

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Introduction. Clinical signs can suggest the diagnosis of Alzheimer's disease and can help in choosing the tactics of later diagnosis and treatment, usually it can be rendered with a degree of probability, because the definitive diagnosis is established by post-mortem cerebral biopsy.
Aim of the study. In this paper, we aim to analyze the literature and to make a synthesis of the clinical signs and distribution of neurofibrillary tangles which can provide data about the severity of the Alzheimer's disease. The main purpose is to identify the clinical signs in each microscopic stages of Alzheimer disease.
Materials and methods. Literature sources were accessed via Sciencedirect by a search on the terms "Stageing of Alzheimer" and "Neurofibrillary tangles".
Results. The literature study has identified 3 clinical stages and 6 microscopic stages, which were combined for practical reasons, these stages are: (transentorhinal 1 and 2), (limbic 3 and 4) (isocortical 5 and 6). Transentorhinal stage represents the preclinical phase of disease, Limbic stage the incipient phase, and Isocortical stage, the presence of dementia.
Conclusions. Each clinical stage of Alzheimer's disease has its microscopic equivalent, therefore, in establishing the presumptive diagnosis of Alzheimer's disease using the NINCDS-ADRDA criteria, the clinician may assume the degree of distribution of neurofibrillary tangles and affected areas, which will dictate the diagnostic, treatment and prognostic approach.
Key words: Alzheimer's disease, neurofibrillary tangles, microscopic stages, clinical stages

231. ROLE OF MATRIX METALLOPROTEINASES IN ANGIOGENESIS AND PROGRESSION OF ATHEROSCLEROTIC PLAQUE

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Introduction. Atherosclerosis is a chronic disease characterized by multifocal structural alterations of the vascular wall of medium and large arteries, leading to the accumulation of cholesterol and continuous inflammation. Inflammatory angiogenesis in atherosclerotic lesions plays a major role in plaque progression and instability.
Aim of the study. The review examines the role of the MMPs in plaque angiogenesis, destabilization, and its relation to inflammation.
Materials and methods. Informational support for the development of this review is based on current international journals, including more than 50 references in English and Russian languages.
Results. It is firmly established that extracellular proteolysis mediated by MMPs is an absolute requirement for angiogenesis. MMPs released by inflammatory cells, are implicated in the sprouting phase, including basement membrane degradation and cell migration/ECM invasion. The neovascularization prevents cellular death due to better supply of O2 and nutrients. But simultaneously allows lipid core expansion, leukocyte afflux, plaque growth and destabilization due to the compromised structural integrity of imature vessels (discontinuous basement membrane, low number of tight junctions between the ECs, lack in pericyte coverage)highly susceptible to intraplaque hemorrhage. In atherosclerotic plaques, MMPs not only induce the