Correlation between vascular disorders and atrophic changes in the brain in Alzheimer’s disease

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Background: There have recently appeared many reports dedicated to cerebral hemodynamics disorders during AD progression. However, certain aspects of cerebral blood flow and microcirculation during AD are not fully understood. This research focuses on the identification of cerebral angioarchitectonics features and the study of micro-circulatory disorders arising during AD progression and on the determination of their importance in AD etiology and pathogenesis.

Methods: 164 patients participated in the research: Test Group-81 patients with different AD stages; Control Group-83 patients with etiologically different neurodegenerative brain lesions accompanied by manifestations of dementia and cognitive impairment of varying severity but without AD. All patients underwent: Assessment of cognitive function (MMSE), severity of dementia (CDR) and AD stages (TDR), laboratory examination, computed tomography (CT), magnetic resonance imaging (MRI), brain scintigraphy (SG), rheoencephalography (REG) and cerebral multi-gated angiography (MUGA).

Results: All Test Group patients with both pre-clinical and clinical AD stages had disorders of cerebral microcirculation manifested in dyscirculatoryangiopathy of Alzheimer’s type (DAAT), namely: Capillary bed reduction in the hippocampus and frontal-parietal regions; multiple arteriovenous shunts in the basin of arterial branches supplying the hippocampus and fronto-parietal brain regions; early venous dumping of arterial blood through these shunts with simultaneous filling of arteries and veins; abnormally enhanced lateral venous trunks receiving blood from the arteriovenous shunts; anomalous venous congestion at the border of frontal and parietal lobe caused by the increased blood inflow from the arterial-venous shunts and increased looping of distal intracranial arterial branches. Control group patients had no combinations of such changes in angioarchitectonics and microcirculation.

Conclusions: The primary basis for these changes is cerebral microcirculation disorders manifested in capillary bed reduction and decreased blood flow to cerebral tissues. The process is accompanied by natural arteriovenous shunts leading to arterial blood dumping to the venous bed followed by temporal and fronto-parietal lateral veins enlargement and subsequent blood congestion. Finally, long-term chronic hypoperfusion of these areas follows causingspecific cerebral tissue hypoxia and ischemia. These changes may then affect amyloid beta metabolism and facilitate its accumulation in brain tissue stimulating AD progression.

Biography
Ivan V Maksimovich, MD, ISTAART member, Head Physician of Clinic of Cardiovascular Diseases named after Most Holy John Tobolsky (Moscow, Russia) since 1993. One of the major problems the clinic deals with is the diagnosis and treatment of various brain lesions including Alzheimer’s disease. For a long time I have fully concerned myself with the diagnosis and treatment of Alzheimer’s disease. Over the past 15 years I have published over 60 scientific works on this subject.

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