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Cerebral small vessels disease in Alzheimer's disease

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Background: The research focuses on the peculiarities of microcirculatory lesions (CSVD) in AD, the determination of the time during which these changes occur before clinical AD manifestation, the correlation between these changes and neurodegenerative processes in the brain, and the comparison of these changes to vascular changes during other neurodegenerative diseases.

Methods: 1110 examined patients with various types of neurodegenerative diseases: 98 (8.83%) patients featured various AD stages: preclinical TDR-0 - 10 (10.20%) patients, early clinical TDR-1 - 26 (26.53%), middle clinical TDR-2 - 40 (40.82%), late clinical TDR-3 - 17 (17.35%), 5 (5.10%) patients aged 8-11 were direct ascendants of AD patients – Test Group. 1012 (91.17%) patients had other cerebral neurodegenerative lesions: different atherosclerotic brain lesions - 946 (93,79%), Binswanger's disease (BD) - 23 (2.27%), Parkinson's syndrome - 34 (3.36%), Parkinson's disease - 9 (0.89%) – Control Group. The examination included MMSE, CDR, TDR assessment, cerebral CT, MRI, SG, REG, MUGA.

Results: All Test Group patients had dyscirculatory angiopathy of Alzheimer's type (DAAT), which is accompanied by specific CSVD changes in temporal and frontoparietal regions:

- reduction in the number of arterioles and capillaries in temporal and frontoparietal regions;
- development of multiple arteriovenous shunts in the same regions;
- early dumping of arterial blood via those shunts into the venous bed;
- abnormal widening of lateral venous branches receiving blood from arteriovenous shunts;
- stagnation of venous blood at the border of the temporal and parietal regions due to the increased amount of blood from arteriovenous shunts;
- increased looping of distal intracranial arterial branches.

Control Group patients with other cerebral neurodegenerative lesions did not have the same complex of the changes in the vascular and microcirculatory system.

Conclusions: The data received prove that CSVD during AD have a complex of specific features, which we named dyscirculatory angiopathy of Alzheimer's type (DAAT). Other neurodegenerative diseases have no complex of such CSVD changes, which means DAAT is characteristic only of AD. DAAT appears many decades before the primary clinical AD symptoms. Direct descendants of patients with AD acquire DAAT in their childhood; it is also characteristic for patients with AD pre-clinical stage. DAAT progression leads to disorders in the metabolism of abnormal proteins causing their accumulation in cerebral tissues and the vascular wall, which inhibits cerebral microcirculation even more leading to atrophic changes in the cerebral tissue and AD progression.

Biography

Ivan V Maksimovich, MD, PhD. ISTAART member, ESC member, EAPCI member, WSO member, ESO member, EPA 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. Over the past 20 years I have published over 200 scientific works on this subject.

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