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## **CHANGES IN VASCULAR ENDOTHELIAL FUNCTION IN RATS WITH EXPERIMENTAL SCOPOLAMINE-INDUCED ALZHEIMER-TYPE DEMENTIA**

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Recently, increased attention of researchers has been attracted by severe cognitive disorders that occur in elderly people against the background of neurodegenerative changes in neurons and other brain structures in patients with Alzheimer's disease. Changes in the walls of small arteries, arterioles, and capillaries of the gray matter of the cerebral cortex, gradually lead to a decrease in blood supply, brain ischemia with subsequent disruption of clearance mechanisms, drainage of  $\beta$ -amyloid through the vascular wall, and increased breakdown of the amyloid precursor protein with subsequent accumulation of amyloid and damage neurons [1,2].

Regulation of cerebral blood flow is carried out with the help of smooth muscle cells of arteries innervated by cholinergic neurons [3]. Previous studies have shown that upon stimulation of the basal parts of the forebrain and striatum, acetylcholine is released and cerebral blood flow increases due to the activation of endothelial NO synthase (eNOS) by acetylcholine, which contributes to the release of NO (nitric oxide) and local vasodilation [4]. The loss of cholinergic neurons and the deficiency of acetylcholine can affect the development of vascular dysfunction, which remains a relevant issue in the study of factors in the progression of Alzheimer's disease.

The experiment was conducted with the participation of 24 male rats of the WAG population weighing 180-250 g (n=8 in each group): scopolamine-induced dementia Scop-14 (1), Scop-28 (2), (an aqueous solution of scopolamine butyl bromide was injected intraperitoneally (Scop) at a dose of 1 mg/kg of one rat once a day for 14 and 28 days; Rats of the control group (group C) received injections of saline instead of scopolamine butyl bromide according to the same scheme. Endothelin-1 (ET) concentrations -1), von Willebrand factor (vWF), vascular endothelial growth factor (VEGF-A), and endothelial nitric oxide synthase (eNOS) in blood serum were determined by the immunoenzymatic method using standard sets of reagents (Elabscience, Wuhan, Hubei, China, 2019) All numerical data were analyzed using GraphPadPrism 5.0 (GraphPad Software Inc., California, USA) and the Statistical Package for the Social Sciences (SSPS) [5].

Analysis of study data showed that compared to the control group in both study groups (Scop-14, Scop-28). It was observed: an increase in the concentration of ET-1 (a powerful endothelial vasoconstrictor) in the serum of cranial arteriovenous blood by



1.51 and 4.65, respectively, an insignificant increase in the level of eNOS (an endothelial enzyme that activates the production of NO) in the blood of rats gr. Scop-14, and vice versa, a significant decrease in the indicator by 1.2 times per gr. Scop-28; significant increase in the concentration of VEGF-A (vascular endothelial growth factor) and vWF (generally accepted criterion of endothelial dysfunction) (in 1.2 and 1.36 and in 1.6 and 6.54, respectively) in both study groups (Scop-14, Scop-28).

Therefore, a comparative analysis of indicators of vascular endothelial function in the blood of rats with scopolamine-induced dementia of the Alzheimer's type and rats of the control group revealed that the greatest changes in the content of ET-1, vWF, VEGF-A (increase in 4,65, 1,4, 6,5) were observed in rats after a 28-day administration of scopolamine butyl bromide and led to a significant increase in the content of ET-1 and vWF in blood serum, which is a confirmation of the violation of the integrity of the monolayer of endothelial cells, vasoconstriction and the risk of blood clot formation.

**Conclusions.** In rats, after long-term administration of the M-cholinoblocker - scopolamine butyl bromide, after 14 days of the "regeneration" period, even before the development of choline deficiency, signs of significant damage to the endothelium of the cerebral vessels were detected.

The degree of vascular endothelium damage depended on the duration of administration of scopolamine butyl bromide and was more expressed in the Scop-28 group, which was confirmed (compared to the control): an increase in the content of ET-1, vWF, in the blood plasma, which characterizes the level of damage and dysfunction of the endothelium, VEGF-A, which indicates the regenerative capabilities of the endothelium, as well as a decrease in eNOS, which reflects the compensatory capabilities of endotheliocytes.

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