FEATURES OF THE DEVELOPMENT OF CEREBRAL VASCULAR DYSFUNCTION IN CEREBROVASCULAR DISEASES

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Review of the literature and data from our own studies on changes in cerebral vessels during arterial hypertension and smoking in the clinic and experiment. Data from transcranial Doppler sonography, duplex scanning and magnetic resonance imaging of the cerebral vascular bed are presented. A decrease in the linear velocity of blood flow, an increase in the vascular resistance index and a greater severity of cerebral atherosclerosis in arterial hypertension and chronic tobacco smoking have been shown. A direct relationship has been established between the intensity of smoking and the incidence of atherosclerotic plaques, including hemodynamically significant stenoses. Severe vasomotor disorders of the brain were established, in the form of loss of the endothelial-dependent component of vasodilation and an increase in vasoconstrictor effects.

In addition to changes in resistive arteries, hypertension is considered as a predictor of disturbances in the elastic properties of large arterial trunks. Changes in the elastic characteristics of the aorta and a decrease in its damping properties lead to an increase in pulse pressure, the development of diastolic dysfunction of the left ventricle and determine the threat of cardiovascular accidents. One of the informative indicators associated with the state of the vascular wall in hypertension, namely its elastic properties, is the systole-diastolic ratio. This is the ratio of the maximum systolic to the end diastolic blood flow velocity. Assessing the systolic-diastolic ratio, we found that in hypertension this indicator is statistically significantly different from the norm. The causes of increased vascular stiffness are varied and include loss of elastin, accumulation of coarse collagen, progression of fibrosis, inflammation, muscle media necrosis, calcification and diffusion of macromolecules into the arterial wall. Thus, with hypertension, early and progressive thickening of elastic arteries develops, which should be considered as a predictor of cardiovascular events.

Thus, we attempted a systematic approach to the analysis of cerebral vascular dysfunction under the influence of such significant risk factors as hypertension and smoking. Morphofunctional changes in cerebral arteries both with smoking and with hypertension have a similar picture. Hypertension and nicotine intoxication provoke structural changes in the vascular system, which leads to the development and progression of atherosclerosis, and consequently, cardiovascular complications - myocardial infarction, cerebral stroke, etc. At the same time, Dr. E.L. Schiffrin and colleagues concluded that the structure of small arteries, which are affected before the development of proteinuria or left ventricular hypertrophy, is a prime target for the above risk factors.

It is obvious that a long-term increase in blood pressure and chronic tobacco smoking is accompanied by an increase in vascular stiffness, including cerebral ones, leading to disruption of the vasomotor function of the endothelium. Such changes, as proven in experimental models, can be caused by endothelial dysfunction due to insufficient activity of the vasodilator component and pathological constrictor reactions. In this case, not only the NO-synthase mechanism of vasodilation is disrupted due to direct damage to the endothelial lining, but also the guanylate cyclase mechanism of vasodilation, apparently due to disorganization of the smooth muscle framework of the arteries and a violation of their relaxation properties

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