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Chronic Cerebrovascular Disorders in Venous Pathology: Clinical and Pathogenetic Relationships, Therapeutic Approaches

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Abstract: Chronic cerebrovascular disorders combined with venous pathology represent a significant medical and social problem, characterized by high prevalence, progressive course, and substantial impact on patients' quality of life. Cerebral venous circulation disorders, including impaired drainage through the internal jugular vein system, dural sinuses, and deep cerebral veins, are often underestimated in clinical practice, leading to inadequate therapy and progression of cerebral disorders.

Keywords: Chronic cerebrovascular disorders, venous pathology, cerebral circulation disorders, venous drainage, intracranial hypertension, cerebrovascular diseases, phlebopathies, venous insufficiency.

Introduction. Venous phlebopathies of the lower extremities (VPLE) represent a global medical and social problem of modern times. Their prevalence reaches 25-30% in women and 10-15% in men. Such high frequency of occurrence of venous phlebopathies of the lower extremities allows calling this disease a "disease of civilization." Varicose disease (varicose veins of the lower extremities) and consequent venous thromboembolic complications, thrombophlebitis - a disease from ancient times, described by Hippocrates, Avicenna, Galen; the disease is confirmed by excavations of Egyptian burials from 1500 BC. According to various authors, prevalence by gender distribution is within women/men 3:1. Moreover, if previously the disease was attributed to problems of older age groups (over 50 years), currently 10-15% of schoolchildren aged 12-15 years show first signs of venous reflux.

Frequent reports of recent decades are data on pathology detection among the young generation (adolescent period). The causes of varicose disease development remain insufficiently studied; many sources of scientific publications attribute their role to hereditary predisposition, congenital anomalies (formed during pathological pregnancy, TORCH infections).

The basis of clinical manifestations of chronic cerebral ischemia is the disproportion between brain needs and vascular system capabilities. Traditionally, most publications in the field of physiology and pathology of cerebral circulation discuss mainly questions of arterial bed pathology. Thanks to the introduction into clinical practice of several non-invasive high-tech radiological techniques allowing comprehensive assessment of cerebral hemodynamics, modern angioneurologists increasingly indicate the important role in the pathogenesis of dyscirculatory encephalopathy of systemic hemodynamic disorders caused by venous blood flow insufficiency in the cranial cavity.

Significant variability in the structure of the cerebral venous system and the absence of a generally accepted complex of diagnostic techniques have led to the fact that currently there is no unified opinion about the nature of cerebral venous hemodynamic changes at various stages of dyscirculatory encephalopathy. Moreover, literature sources about the connection of the general venous system, and in particular, the influence of lower extremity phlebopathies on the process of chronic cerebral ischemia (CCI) development are scarce.

The pathomechanism of the disease is not unambiguous; it involves venous wall fibers and muscle cells, collagen level, characteristics of the body's coagulation system, mechanical load of venous

valves, static-dynamic imbalance of blood perfusion in tissues, etc., but the main triggering mechanism of the disease is venous valve dysfunction and blood reflux. The most dangerous remains thrombus formation, vessel lumen occlusion, and detachment from the vessel wall, with entry into vital body parts: lungs, brain. This fact complicates the assessment of the role of venous dysgemias in the formation of cerebrospinal fluid dynamics and structural brain changes in chronic cerebrovascular diseases.

Important roles in the development of various peripheral circulation complications, including thrombotic ones, are played by hemostasis system disorders, including the hemostatic component of endothelial dysfunction (Makarov M.S., 2015). Most authors recognize endothelial dysfunction as the main predictor of cardiovascular events, and endothelium is characterized as a distinct type of tissue participating in the development of a wide spectrum of pathological conditions and adverse outcomes.

Endothelial dysfunction is a complex multifaceted process, the main manifestation of which is impaired synthesis of a large number of biologically active substances involved in vasoconstriction and vasodilation, inflammation, coagulation, and angiogenesis processes.

At the same time, the role of coagulation markers of endothelial dysfunction, as well as their relationship with coagulation factors, remains insufficiently studied in patients with chronic dysgemias.

During invasive (surgical) intervention, inevitable endothelial trauma occurs, leading to its dysfunction, for example, during phlebectomy. In rare cases, chronic venous insufficiency begins to develop after trauma (bruising, rupture, deep burns, or hypothermia). Another example is phlebopathy.

Changes in endotheliocyte structure or function are crucial in vascular damage, and thus assessment of kinetic profile, secretion characteristics, and activity of various biological substances specific to endothelium can help in evaluating biomarkers of both acute and chronic vascular damage. Main markers of endothelial dysfunction directly involved in hemostasis include coagulation factor FVIII, vWF, PAI-1, sEPCR, and NO.

It is indisputable that in the pathogenesis of both acute and chronic forms of cerebrovascular disorders, important roles are played by insufficient blood supply to brain tissues and impaired venous outflow, which sometimes come to the foreground in the clinical picture of the disease. Long-existing venous disorders in most patients can be the cause of characteristic clinical symptom development, accompanied by decreased patient performance and deterioration of their quality of life.

Chronic lower extremity ischemia is a disease where vessels are affected for various reasons, disrupting blood flow in the lower extremities and pelvis. This symptom complex has shown a growth trend in recent decades (smoking, sedentary lifestyle, dietary disorders), progresses, and is a provocateur of acute cerebral and cardiac circulation disorders. Questions about hemodynamic parameters throughout the entire circulatory system at various stages of cerebrovascular disorders remain open; early subclinical signs of combined cerebrovascular disorders and phlebopathies have not been established. According to Virchow's version, which many scientists adhere to, dangerous are specifically thrombi of lower extremity veins, detaching from the femoral segment and distal vessel sections.

Such patients need deep blood composition analysis, assessment of cardiovascular system condition, pulmonary system, neurosonography, and angiopulmonography of extremity vessels. Patients with chronic cerebral ischemia against the background of phlebopathies should be under observation not only by neurologists but also vascular surgeons, phlebologists, and should undergo timely comprehensive rehabilitation.

Currently, available literature lacks sufficient information about the level of central nervous system damage against the background of venous thrombosis formation and varicose vein expansion. The problem is insufficiently covered in the gender aspect. Available literature is devoted mainly to pathomorphological studies as a post-factor of acute strokes, the etiology of which is related to

pulmonary artery thromboembolism. Aspects of chronic cerebral circulation insufficiency occurrence, risk factors of which include lower extremity venous insufficiency disorders, complication percentage, gender and age characteristics, specifics of clinical-neurological disorders, optimization of diagnosis, treatment, rehabilitation, and patient prevention are insufficiently studied, which dictates the necessity of comprehensive study of pathogenetic parallels between CCI and chronic phlebopathies of lower extremities (CPLE).

Conclusions. Effective treatment of chronic cerebrovascular disorders in venous pathology requires a personalized approach considering pathogenetic mechanisms, including pharmacotherapy, non-pharmacological correction methods, and rehabilitation measures. A comprehensive therapeutic strategy should be aimed at improving venous outflow, normalizing blood rheological properties, neuroprotection, and correction of concomitant disorders.

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