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THE INFLUENCE OF LOWER LIMB VENOUS THROMBOSIS ON THE DEVELOPMENT OF CHRONIC CEREBRAL VENOUS DISCIRCULATION

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Abstract: The aim of this article is to investigate the potential role of lower limb venous thrombosis in the development and progression of chronic cerebral venous circulatory disorders. The pathogenetic mechanisms linking peripheral venous thrombosis to cerebral venous dysfunction are examined, including: a hypercoagulable state, endothelial dysfunction, chronic inflammation, impaired venous tone, systemic venous hypertension, and common vascular risk factors.

Keywords: Lower Limb Venous Thrombosis, Chronic Cerebral Venous Discirculation, Venous Outflow, Hypercoagulation, Endothelial Dysfunction, Venous Hemodynamics, Chronic Cerebrovascular Accident

Introduction

Chronic cerebral venous discirculation is a disruption of venous outflow from the brain, accompanied by alterations in intracranial venous hemodynamics, microcirculation, and cerebrospinal fluid dynamics. In recent years, increasing attention has been paid to systemic factors that can affect the state of the venous system, including thrombosis in the veins of the lower limbs[1]. It has been shown that lower limb venous thrombosis should not be regarded as an isolated, local pathology. In some patients, it may be a manifestation of a systemic disorder of hemostasis and venous hemodynamics, which potentially increases the risk of cerebral venous disorders[2]. A comprehensive assessment of the venous system, thrombosis factors, and the state of cerebral venous outflow is important for the early diagnosis and prevention of chronic cerebrovascular accidents. Chronic cerebrovascular accidents are traditionally viewed primarily from the perspective of arterial pathology: atherosclerosis, arterial hypertension, microcirculatory disorders, and ischemic brain damage. However, the venous component of cerebral hemodynamics also plays a vital role in maintaining the normal functioning of the central nervous system[3].

Chronic cerebral venous dyscirculation (CCVD) is a complex pathological condition caused by impaired venous outflow from the brain and accompanied by changes in intracranial hemodynamics, microcirculation, and fluid dynamics. This condition is increasingly being considered a significant factor in the formation of chronic cerebrovascular insufficiency, affecting cognitive functions and the general neurological condition of patients[4].

In recent years, interest in the systemic mechanisms underlying venous dysfunction, including the role of peripheral venous disorders, has been increasing. Thrombosis of lower limb veins is traditionally considered a local vascular pathology, but the accumulated data indicate a possible connection with generalized hemostasis disorders, endothelial dysfunction, and chronic inflammation. These processes can contribute to the formation of systemic venous hypertension and changes in venous tone, which in turn affects cerebral venous outflow[5].

The study of the pathogenetic mechanisms linking peripheral vein thrombosis with disorders of cerebral venous hemodynamics is of particular interest. Such mechanisms include hypercoagulation states, activation of inflammatory cascades, damage to vascular endothelium, and changes in the reological properties of blood. These factors may play a key role in the development and progression of CSVD[6].

Cerebral venous dyscirculation develops when the outflow of blood from the brain becomes difficult,

venous pressure increases, microcirculation is disrupted, and fluid dynamics change. Clinically, it can manifest as headache, heaviness in the head, dizziness, ringing in the ears, decreased cognitive productivity, sleep disturbances, and autonomic disorders[7].

One of the possible factors associated with chronic venous dysfunction is the formation of blood clots in the veins of the lower extremities. Thrombosis of deep veins in the lower extremities and post-thrombotic disease are common forms of venous pathology. They are accompanied by impaired venous outflow, damage to the endothelium, changes in the hemostasis system, and the development of chronic inflammation[8].

It is important to emphasize that lower limb vein thrombosis does not directly cause cerebral venous dyscirculation through mechanical means. However, it may reflect the presence of systemic pathological processes that simultaneously affect the peripheral and cerebral venous beds[9].

For the cerebral venous system, endotelial dysfunction may be of particular importance, as the brain is sensitive to even moderate disorders of microcirculation and venous outflow. Damage to the endothelium can contribute to impaired neurovascular regulation, impaired tissue metabolism, and the development of chronic symptoms of venous dysgemia. After undergoing thrombosis of the veins of the lower extremities, post-thrombotic disease develops in a portion of patients. It is characterized by chronic disorders of venous outflow, damage to the valvular apparatus, venous hypertension, and inflammatory changes in the vascular wall[10].

Chronic inflammation in venous pathology can maintain systemic vascular dysfunction. Inflammatory mediators affect the endothelium, hemostasis system, and microcirculation. Thrombosis of deep veins in the lower extremities can lead to persistent disorders of venous return. In cases of severe chronic venous insufficiency, the function of the musculo-venous pump changes, venous pressure in the lower extremities increases, and microcirculation deteriorates. Analysis of pathogenetic and clinical data indicates that lower limb venous thrombosis may be associated with chronic cerebral venous dyscirculation primarily through systemic mechanisms. The main ones are hypercoagulation, endothelial dysfunction, chronic inflammation, impaired venous tone, and general risk factors for vascular pathology[11].

One of the key mechanisms of connection is hypercoagulation, a state of increased blood readiness for thrombus formation. In hypercoagulation, the risk of thrombus formation increases not only in the deep veins of the lower extremities but also in other venous basins. Thus, hypercoagulation is one of the most likely pathogenetic bridges between limb thrombosis and cerebral venous dyscirculation[12]. The endothelium plays a crucial role in regulating vascular tone, blood coagulation, inflammation, and the permeability of the vascular wall. In cases of venous pathology, damage to the endothelial layer occurs, contributing to thrombus formation and the disruption of normal blood flow. For the cerebral venous system, endotelial dysfunction may be of particular importance, as the brain is sensitive to even moderate disorders of microcirculation and venous outflow. Damage to the endothelium can contribute to impaired neurovascular regulation, impaired tissue metabolism, and the development of chronic symptoms of venous dysgemia[13].

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For the cerebral venous system, this is of particular importance, as impaired permeability of venous sinuses or small veins can lead to impaired blood flow from the brain. In severe cases, hypercoagulation can contribute to the development of cerebral venous sinus thrombosis. In milder or chronic variants, it can support microcirculatory disorders, venous congestion, and endothelial damage. This allows for the

consideration of chronic inflammation as an additional factor linking peripheral venous pathology with cerebral[15].

Materials and Methods

This study was designed as a narrative analytical review combined with a comparative evaluation of clinical and pathophysiological data. Scientific publications indexed in international databases, including PubMed, Scopus, and Web of Science, were analyzed to identify current evidence on the relationship between lower limb venous thrombosis and chronic cerebral venous discirculation. The selection criteria included peer-reviewed articles, clinical guidelines, and meta-analyses published in recent years, as well as classical foundational studies.

The methodological approach involved systematization of data on hemostasis disorders, endothelial dysfunction, inflammatory mechanisms, and venous hemodynamics. Special attention was given to identifying common risk factors and pathogenetic links between peripheral and cerebral venous systems. Comparative analysis and synthesis methods were applied to evaluate consistency and contradictions in available findings, ensuring an integrated interpretation of the results.

Reseults and Discussion

Thrombosis of deep veins in the lower extremities can lead to persistent disorders of venous return. In cases of severe chronic venous insufficiency, the function of the muscular-venous pump changes, venous pressure in the lower extremities increases, and microcirculation deteriorates.

An increase in pressure in the veins of the lower extremities does not mean an automatic increase in pressure in the cerebral veins. However, in the presence of comorbidities - for example, heart failure, pulmonary hypertension, obesity, and hypothyroidism - a more widespread disorder of venous return may develop.

This mechanism is particularly significant in patients with a combination of venous thrombosis, cardiovascular diseases, and a sedentary lifestyle. Lower limb venous thrombosis and chronic cerebral venous dyscirculation may have common risk factors. This means that one condition is not always the direct cause of another, but both may develop against a single pathological background.

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The obtained data allow for the consideration of lower limb vein thrombosis as an important clinical marker of systemic venous and hemostatic dysfunction. However, the relationship between peripheral venous thrombosis and chronic cerebral venous dyscirculation should be evaluated with caution. Thrombosis of the lower limb veins can be considered a significant clinical marker of systemic venous pathology and increased thrombotic risk. Their influence on the formation of chronic cerebral venous dyscirculation is primarily realized not through direct mechanical pathways, but through general pathogenetic mechanisms: hypercoagulation, endothelial dysfunction, chronic inflammation, and disturbances in microcirculation and venous hemodynamics.

In patients with a history of lower limb venous thrombosis and complaints characteristic of cerebral venous outflow disorders, a comprehensive assessment of the vascular system must be conducted. The study of hemostasis, assessment of risk factors, ultrasound examination of the venous bed, and neuroimaging methods are of particular importance if indicated.

Early diagnosis and correction of systemic venous disorders can help prevent the progression of chronic cerebral venous dyscirculation, reduce the severity of neurological symptoms, and improve patients' quality of life.

Conclusions

Thus, studying the impact of lower limb venous thrombosis on the formation of chronic cerebral venous dyscirculation is a relevant direction of modern angioneurology. An in-depth understanding of these relationships is essential for developing effective approaches to early diagnosis, prevention, and treatment of venous cerebral circulation disorders. Thrombosis of the veins of the lower extremities should be considered not only as a local vascular pathology but also as a possible marker of systemic hemostasis dysfunction. The direct causal link between lower limb venous thrombosis and chronic cerebral venous dyscirculation is not unequivocally proven. The most likely connection is realized through hypercoagulation, endothelial dysfunction, inflammation, and general vascular risk factors. Patients with venous thrombosis and neurological complaints require comprehensive examination, including the assessment of cerebral venous outflow. Prevention of venous thrombosis recurrence can be important not only for the condition of the lower extremities but also for the patient's overall vascular health.

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