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Editorial

Chronic Venous Disease: From Theory and Leukocyte-Endothelial Interaction to Possibilities of Pharmacotherapy

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The pathogenesis of chronic venous diseases (CVD) is rather multidimensional, there is still no unified interpretation of many pathological changes. This applies to both macrohemodynamic and microcirculatory changes. Even in primary forms of CVD, pathological changes are multidimensional and often multidirectional. The literature presents a number of pathogenetically significant factors that play a role in the development of this pathology. At the same time, their significance and priority are assessed ambiguously.

In spite of certain disagreements in the assessment of trigger mechanisms of lower limb varicose veins disease development, the majority of researchers currently are supporters of the polyetiological theory of the disease. The pathogenesis of CVD is complex, multifaceted, the role of trigger mechanisms of venous outflow disorders and the sequence of pathophysiological changes leading to the formation of chronic venous insufficiency (CVI) is assessed by the researchers in a controversial way.

The results of these studies allowed us to approach the understanding of the main pathogenetic mechanisms involved in the occurrence of CVD and the progression of CVD.

The genetic basis for variceal transformation is currently recognized as a significant but not fully understood factor. Obviously, certain gene mutations play an important role and act as an intractable risk factor for CVD.

Scientific advances have led to an understanding of the importance of inflammatory processes in the pathogenesis of CVD, in the valves and walls of veins of all sizes, and in the skin, resulting in varicose vein transformation and trophic disorders.

Current understanding of pathophysiological mechanisms allows to identify potential therapeutic targets, which can be effective not only in reducing or eliminating the symptoms and signs of CVD, but also in slowing its progression.

Drug therapy to suppress inflammation is the most promising in preventing disease complications. Currently available drugs, particularly venoactive drugs (VAP), are capable of suppressing various components of the inflammatory cascade, particularly the leukocyte-endothelial interaction.

Most VAPs increase venous tone through a mechanism related to the noradrenaline pathway. In animal models of ischemia/reperfusion, VAPs reduce the release of inflammatory mediators such as free oxygen radicals, prostaglandins and thromboxane, thereby blocking inflammatory interactions both within and outside the venous wall itself.

A number of authors have demonstrated the ability of VAP to effectively reduce the levels of endothelin-1 (ET-1) and tumor necrosis factor-α (TNF-α) in patients with varicose vein disease, which shows the positive effect of the drug on the regulation of oxidative stress, correction of the antioxidant system imbalance observed in CVD, as well as endothelial cell protection. One possible explanation for the protective mechanism of VAP may be a dose-dependent decrease in the content of hydrogen peroxide released from activated leukocytes.

VAP increase capillary resistance and decrease transcapillary filtration. These effects are observed with the administration of MOFF, rutosides, escin, proanthocyanidins and calcium dobesylate. The protective effect of VAP on capillaries may be due to inhibition of leukocyte adhesion to capillaries.

The role of pharmacotherapy as a possible means of influencing, or maybe preventing (?) the natural course of CVD continues to be actively discussed. Scientific advances have led to an understanding of the importance in the pathogenesis of venous remodeling of chronic inflammatory processes in the valves and walls of veins of all sizes, as well as in the skin, the result of which are varicose vein transformation, disease and trophic disorders (C2-C6).

At the present stage of science, we still have insufficient data on the role of VAP in the prevention of the natural course of CVD, respectively, the effectiveness of this group of drugs in the prevention of future complications is still under some doubt.

In the current clinical guidelines for the treatment of lower limb CVD, the degree of recommendation and level of evidence for phlebotropic drugs and the effect on venous symptoms are consistent. VAPs have also been shown to be effective to varying degrees in reducing lower limb edema.

The findings on the possibility of perioperative use of VAP are very important for us. In patients with chronic venous insufficiency (CVI) phlebotropic drugs can be used in combination with open surgery, endovenous procedures such as stenting, thermal or chemical ablation of the saphenous vein, compression therapy or a combination thereof.

Currently, CVI can be considered as a relatively independent pathological condition, the main cause of which is venous stasis-induced cascade of pathological changes at molecular, cellular and tissue levels. Taking into account the hemodynamic theory of venous valve functioning, the theory of "venous specific inflammation", the basis for which is leukocyte aggression, becomes more understandable.

The search for a universal remedy to combat this phenomenon is a certain utopia, but the role of VAP is quite significant here. The use of drugs of this group in real phlebology practice, along with compression, is the main option of conservative treatment strategy of CVD and CVI.