

UDC: 616.14:616.61-089:616.147.3-005.2

**PATHOMORPHOLOGICAL PICTURE OF CHANGES IN THE
SUPERFICIAL VEINS OF THE POPLITEAL REGION.**

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Abstract. Varicose veins are the most common vascular disease in humans. Veins have valves that help the blood return gradually to the heart without leaking blood. When these valves become weak, blood and fluid collect and pool by pressing against the walls of the veins, causing varicose veins. In the cardiovascular system, mechanical forces are important determinants of vascular homeostasis and pathological processes. Blood vessels are constantly exposed to a variety of hemodynamic forces, including shear stress and environmental strains caused by the blood flow. In varicose veins within the leg, venous blood pressure rises in the vein of the lower extremities due to prolonged standing, creating a peripheral tension in the vessel wall thereby causing mechanical stimulation of endothelial cells and vascular smooth muscle.

Key words: varicose veins, plasmatic swelling, elastolysis, venostasis.

Relevance of the topic. Lower extremity venous insufficiency, also known as reflux or incompetence, is a condition where the normal one-way return of venous blood back to the heart has been disrupted and blood flow is bidirectional. Thin, pliable valves normally present in all peripheral veins normally prevent retrograde flow of blood; failure or damage to the valves is thought to be responsible for venous insufficiency.

According to the literature, varicose veins account for 5% of the overall morbidity structure, and when peripheral vessels are affected, it reaches 30-40% (Van DerVelden SK, et al., 2015). In Russia, 30 million people suffer from various forms of varicose veins of the lower extremities, while 40% of women and 20% of men suffer from varicose veins (Kiriyenko A.I. et al., 2015). Although the problem of treating varicose veins has a centuries-old history, it remains relevant today (Shevchenko Yu.L. et al., 2018). The continued relevance of the problem depends on the prevalence of this pathology, rejuvenation, an increase in the frequency of relapses and a decrease in functional capabilities. The multi-million dollar costs of treating this pathology give varicose veins a socio-economic dimension (Gauw SA et al., 2016). The general rule is that the surgical method leads to a complete cure at any stage of varicose veins. However, due to the prevalence of varicose veins, existing specialized departments do not have the capacity to treat patients with varicose veins in a timely manner. This is also the cause of relapses of varicose veins. Relapses of varicose veins after surgery

are observed in 50%, in some cases up to 70% after 5 years. Relapse of varicose veins is clinically more pronounced than the primary form. Reoperation of relapse of varicose veins is difficult, i.e. innervation is often impaired, lymphostasis develops, cosmetic defects appear, therefore the rehabilitation period is extended and costs increase.

Materials and methods . We morphologically studied sections of the vein wall and the area of the muscular-venous pump of 75 patients who received inpatient treatment and surgery for varicose veins in the Fergana Multidisciplinary Medical Center, which is considered the clinical base of the FMIOH.

Results of the study. It was established that venous congestion occurs due to sharp dystrophic and atrophic changes in the myocytes of the parietal muscle layer with morphological changes in the vessels of the superficial veins of the popliteal region. It is caused by continuous expansion of the vessels of the main vein, hypodynamia, compression of compression factors in the heart area, right ventricular failure occurring with congenital heart disease, etc. As a result, a continuous full-fledged process occurs in the veins. As a result, it was found that the surface formed from the intima of the vein is covered with flat endotheliocytes, and the reticular and collagen fibers in the structures that act as a free one-way valve are destroyed and defragmented, and rare and coarse fibrous structures on the surface have formed at different levels. This means that the valve is enlarged in morphofunctional terms and the dimensions are changed and do not perform its function. Morphological changes in the form of swelling, destruction and elastolysis are detected in all fibrous structures, plasmatic swelling of the intima layer of the vein, formation of interstitial tumors in space. This was evidenced by the detection of an infiltrate consisting of poorly formed lymphocytes, macrophages, 1-3 neutrophils, histiocytes in a 200x magnification focus in areas affected by elastolysis. On the surface of the intima layer, foci of red thrombi of a mixed type are detected around foci of erythrosis on the surface of the damaged valve system.

In conclusion, it should be noted that due to the fine structure of venous vessels, the greatest damage is manifested mainly in the form of elastolysis, destruction and defragmentation in fibrous structures. At the same time, the formation of mixed-type thrombi on damaged endothelial surfaces continues.

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