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Research Article

CHRONIC VENOUS INSUFFICIENCY AND TROPHIC ULCERS OF THE LOWER EXTREMITIES

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ABSTRACT

Chronic venous hypertension (CVH) triggers a whole cascade of pathological reactions, the ultimate result of which is gross changes in the trophism of soft tissues of the lower extremities. The main hemodynamic factor leading to hypertension in the superficial venous system and, subsequently, to trophic changes in the lower limb, is blood reflux from deep veins, and it is associated with valve insufficiency at the mouth of the great and small saphenous veins (vertical reflux), as well as incompetency of perforators (horizontal reflux). Moreover, 90% of the latter are localized in the calf region, of which 87% belong to the veins of the Cockett I-III zone.

KEYWORDS

Chronic venous insufficiency, trophic ulcers.

INTRODUCTION

Chronic venous insufficiency (CVI) can be considered a general medical problem, since doctors of various specialties encounter it every day. CVI is characterized by a progressive and often complicated course, with significant social and economic consequences. This is due to the prevalence of the pathology, the high cost

of diagnosis and treatment, as well as the long periods of disability of patients. Numerous epidemiological studies show that the incidence of CVI in some population groups is no less than 60-75% of observations [6, 7]. Based on international research

data, V.S. Savelyev and A.N. Kiriyyenko rightly call CVI a “disease of civilization” [2,4].

Another important aspect of the problem is that against the background of existing CVI, the risk of acute thrombosis of the deep venous system of the inferior vena cava (IVC) sharply increases, which is known to be extremely dangerous with the likelihood of developing such a formidable complication as pulmonary embolism, characterized by high mortality.

Recently, the number of patients suffering from CVI has begun to increase sharply, largely due to young people [11,14], who, as is known, are extremely demanding regarding the cosmetic results of surgical correction of this disease. The next aspect is associated with patients of the older age group due to the difficulties encountered in the treatment of venous trophic ulcers, which develop mainly in people with a long history of CVI [6, 7, 8]. Moreover, in 50-60% of gerontological patients, due to severe concomitant pathology and the extensiveness of the ulcerative surface, performing the traditional Linton operation in such cases, due to its traumatic nature and high frequency of purulent complications, turns out to be very problematic. At the same time, refusal of surgical intervention and limitation of only conservative measures for the management of venous ulcers is associated with an extremely long recurrent course of the disease, leading to persistent disability of patients, and ultimately extremely low cure rates [3].

Based on the fact that surgery is still considered the most effective and “radical” type of treatment for many manifestations of CVI, more than 80% of patients with VD are operated on in general surgical hospitals. At the same time, it is well known that the results of such operations are significantly inferior to those of specialized departments and centers [10]. Traditional surgical treatment is dominated by operations of a

standard volume, which are often insufficient in some situations and excessive in others. At the same time they are distinguished by hightraumatic, remaining unsatisfactory in terms of aesthetic requirements, and are accompanied by a long period of postoperative rehabilitation. Unfortunately, the frequency of relapses with existing types of surgical treatment is quite high, and the risks of their occurrence are estimated as 50% for every 5 years after surgery, and, depending on the follow-up period, range from 20 to 80% [9,11].

The insufficient effectiveness of surgical treatment is often due to late presentation of patients, when surgical treatment is not able to eliminate all the symptoms of CVI. This is due to shortcomings in the work of the outpatient department and delayed diagnosis, and above all, the absence and underestimation of the results of ultrasound angioscanning, which is currently the main method for identifying phlebohemodynamic disorders.

The main hemodynamic factor leading to hypertension in

superficial venous system and, subsequently, to trophic changes in the lower limb, is the discharge of blood from the deep veins, and it is associated with insufficiency of the valves at the mouth of the great and small saphenous veins (vertical reflux), as well as the failure of perforators (horizontal reflux). Moreover, 90% of the latter are localized in the lower leg area, of which 87% belong to the veins of the Cockett zone I-III.

Subsequently, pathological reflux is aggravated by dysfunction of the muscle pump of the lower extremities, especially in patients with limited physical activity. Long-term hemodynamic disturbances invariably lead to disturbances at the microcirculatory

level. Constant venous hypertension causes the development of skin trophic changes with hyperpigmentation, fibrosis of subcutaneous fat and, finally, a trophic ulcer.

Insufficiency of the valves of the deep venous system, as a rule, is a consequence of deep vein thrombosis. Incompetence of the saphenous vein valves can be primary, as a result of low tone of the vascular wall or weakness of their valves, and secondary, as a consequence of previous phlebitis, stretching of the venous wall during hormonal therapy or due to a transmittable increase in pressure in the areas of connection with the deep venous system, more often - at the sapheno-femoral and sapheno-popliteal anastomosis [12]. In the latter case, varicose veins of the saphenous veins form in the proximal part and subsequently spread to the distal parts of the limb.

In horizontal reflux, the action of the muscular pump forces blood under pressure back into the saphenous veins through incompetent perforator valves. Due to the resulting venous hypertension, there is an expansion of the venous wall and, as a consequence, secondary incompetence of the saphenous vein valves [18].

Obstruction of the deep veins can significantly limit blood flow from the limb, causing a sharp increase in venous pressure during contraction of the lower leg muscles and secondary dysfunction of the muscle pump. A mechanical obstruction to the outflow of blood through the deep veins can be caused by purely “venous” reasons, such as PTF with inadequate recanalization or stenosis of the lumen of the vessel, or external factors such as May-Thurner syndrome (compression of the left common iliac vein passing between the right common iliac artery and lumbosacral region) [12]. Some researchers are inclined to believe that mechanical obstructions to the outflow

of blood from the lower extremities have a more important role in the pathogenesis of CVI than previously thought [8].

Impaired function of the muscle pump of the limb causes a decrease in the efficiency of emptying its distal parts. Muscular dysfunction of the venous outflow rarely develops primarily as a consequence of neuromuscular diseases or muscle atrophy. Typically, the pumping function of the muscles becomes unproductive due to severe perforating insufficiency or deep vein obstruction. At the same time, the transfer hydrodynamic pressure exerted from the deep venous system to the superficial one remains high both in conditions of active movements and during any movements that occur after long-term rest. Today, muscle pump dysfunction is considered one of the main mechanisms for the development of secondary varicose veins of the saphenous veins and its complications, incl. and trophic ulcers.

It is known that the severity of hemodynamic and trophic disorders is determined not by the type of anastomosis, but by the level of its location. Certainly, the shunt pressure will be greater the more distal the incompetent perforating vein is located. In this case, the high pressure caused by retrograde blood flow from the proximal parts of the superficial venous system is joined by even higher pressure from the deep system, transmitted through incompetent perforators.

Acting as a permanent distal obstacle to venous circulation, gravity is present in all aspects of the pathogenesis of CVI and dominates all generally known interpretations of the development of venous pathology. A chronic increase in venous pressure, most pronounced in the distal parts of the leg, leads to leveling of the arteriolar-venular gradient, slowdown, and in advanced cases, stasis of the microvasculature

with subsequent hypoxia and tissue edema, and, finally, the development of trophic disorders [2, 4].

Clinically significant disturbances in the hemodynamics of large veins are inevitably transmitted to the level of microcirculation of the lower limb and ultimately lead to the development of venous microangiopathy [2]. Morphologically, microangiopathy is manifested by elongation, expansion and tortuosity of the capillary networks, thickening of the basement membrane with an increase in the content of collagen and elastic fibers, damage to the endothelium with expansion of the interendothelial spaces, increased pericapillary edema with the formation of a fibrous halo. Pathologically altered capillaries with increased permeability and high venous pressure lead to the accumulation of excess fluid, macromolecules and extravasation of red blood cells into the interstitial substance of tissues. In addition to the pathomorphosis of the microvascular bed and connective tissue, changes in the lymphatic network and nervous system of the limb are soon added. Fragmentation and destruction of microlymphatic vessels further aggravate the drainage system of the limb, and disruption of innervation is fraught with a complete loss of microcirculatory tone.

Several mechanisms have been formulated in the literature that determine the occurrence and development of venous microangiopathy, which, in particular, include the theory of fibrin cuff formation, growth factor stimulation and accumulation of white blood cells. According to the fibrin cuff theory, excess fibrin-rich tissue fluid accumulates in the pericapillary space. This extremely weak fibrinolysis cuff promotes thickening of the diffusion barrier, inhibits repair processes and supports the inflammatory response. As fibrin thickness increases and other macromolecules accumulate, healing processes become impossible. Closely related to this theory is another mechanism,

according to which fibrin, which progressively accumulates in the area of edema and ischemia, stimulates growth factor and attracts macromolecules to this area, which makes it impossible to fully initiate healing mechanisms. A decrease in the number of functioning capillaries and impaired regulation of vascular tone leads to a decrease in the reactivity and functional reserve of the microvasculature. [24,25,27]

Further, the accumulation of white blood cells in capillaries and post-capillary venules, their adhesion and activation continues, accompanied by the release of inflammatory mediators and proteolytic enzymes with damage to the endothelium, which, in turn, increases vascular permeability and blood stagnation.

Tissue hypoxia activates endothelial cells, which also release inflammatory mediators and mitogenetic molecules. Inflammatory mediators, completing the pathological chain, induce adhesion and aggregation of leukocytes and indirectly induce proliferation of smooth muscle cells. The result of inflammation is an increase in capillary permeability, the development of edema, damage to the endothelium by oxygen free radicals, aggregation and adhesion of neutrophils and platelets, and the release of coagulation factors with the formation of platelet clots. In addition, the rheological properties of blood change towards pronounced hypercoagulation and a tendency to thrombus formation [16,17].

Proteolytic enzymes and the free radicals released can also damage numerous other biological structures, such as collagen. Proliferating smooth muscle cells change their phenotype and lose the ability to physiologically contract, unlike a healthy venous wall. Tissue hypoxia and leukocyte aggression cause tissue damage, the occurrence of edema, lipodermatosclerosis and trophic ulcers of the limb in its distal part.

Thus, chronic venous hypertension triggers a whole cascade of pathological reactions, the end result of which is gross changes in the trophism of the soft tissues of the lower extremities [5, 14, 24, 30].

Diagnosis of CVI today is not very difficult and is based primarily on clinical and anamnestic data. The main purpose of examining the patient is to assess the anatomical and functional state of the inferior vena cava (IVC) system. When diagnosing primary varicose veins, it is necessary to differentiate it from varicose dilation of the saphenous veins that occurs during postthrombophlebitic syndrome, as well as from congenital varicose dilation of the saphenous veins due to the discharge of arterial blood into the venous system through multiple network-like arteriovenous anastomoses in Klippel-Trenaunay and Parkes diseases. Weber.

It is equally important to differentiate a trophic ulcer in varicose veins from an ischemic ulcer of the leg in Martorell syndrome, which is based on arterial hypertension. Ischemic ulcers with Martorell syndrome occur more often in older women. The disease is characterized by constant sharp pain in the distal parts of the leg, where spots appear, followed by the formation of ulcers in the absence of skin pigmentation, disorders of regional venous and arterial circulation. [15]

With an undoubtedly clear diagnosis of diseases of the venous system, which does not require surgical correction, we can limit ourselves only to a clinical examination. Auxiliary diagnostic methods used in clinical practice are plethysmography, computed spiral tomography, magnetic resonance imaging, phlebotonometry, oscillography, and intravascular ultrasonography.

X-ray contrast venography allows you to visualize deep and superficial veins, obtain information about the condition of the valve apparatus, its competence or failure and other morphological changes in the venous system. However, the widespread use of this method is associated with a number of disadvantages: invasiveness, radiation exposure to the patient and staff, the risk of complications, the impossibility of frequent repetition, and high cost. Therefore, today it is used in complex diagnostic situations, as well as in preparing patients for reconstructive or endoscopic interventions [13,14,19].

Due to the improvement of ultrasound methods in recent years, the demand for traditional functional tests and radiopaque venography has noticeably decreased [7,13,15].

Using Doppler ultrasound, sound information is obtained that allows one to verify the presence or absence of blood flow through the main veins, and in combination with functional tests, it helps to detect reflux of blood from the deep venous system to the superficial one. This examination method is safe for the patient, has a lower cost and is widely available. However, this technique has low sensitivity and specificity compared to color duplex ultrasound scanning. [14]

Today, the main method for assessing the state of the IVC system is duplex scanning with color Doppler mapping, which can answer almost all questions of interest in assessing the anatomical and functional state of the IVC system: 1) the presence of incompetence of the sapheno-femoral and saphenopopliteal anastomosis; 2) the prevalence of reflux in the saphenous veins and their diameter; 3) localization, diameter and functional state of perforating veins; 4) the state of the deep venous system, including the consistency of the valve apparatus, signs of previous

venous thrombosis, localization of blood clots, as well as the presence of their flotation [5, 17, 27, 28, 31].

With the beginning of the widespread use of CDS, the authors' priorities for many details of treatment radically changed. So, according to Ricci S. et al. (27) when performing CDS in patients of clinical class C4-C6, in 26.7% of cases, no hemodynamically significant perforating veins were detected. On the contrary, in patients with lymphostasis and edema, many small incompetent PVs can be identified [13]. But at the same time, they do not require surgical correction, since they play a compensatory role in conditions of sclerotic tissue.

Undoubtedly, the question of the hemodynamic significance of perforating veins of various groups has not been resolved. All the more importance should be attached to constant CDS monitoring of this group of patients and assessment of changes in hemodynamics after treatment. Summarizing the above, it is necessary to emphasize the introduction of “the art of seeing the invisible” (J. Swift), assessing and providing complete information about the state of the venous system leads to the fact that many pathological processes under the targeted central nervous system have become accessible to minimally invasive treatment methods.

Recently, significant progress has been made in the development of diagnosis and assessment of the severity of venous dysfunction. In this regard, in 1994, experts from the consensus group of the American Venous Forum developed the CEAP classification. The CEAP classification covers clinical, etiological, anatomical and pathophysiological signs, which is why researchers from different countries were able to achieve standardization and objectification of results in their work and compare the effectiveness of various treatment methods. [17]

The clinical section of the classification describes the clinical status of the patient. The reason for classifying a patient into one class or another is the presence of the most pronounced objective symptom of CVI. In defining the clinical section of CEAP, it is more correct to use the term “class”; the terms “stage” and “form” are not suitable in this case. There is no consistent relationship between the classes of CVI; the disease can manifest itself immediately, for example, with edema and even trophic disorders. [14] CO - no visible or palpable signs of CVI C1 - telangiectasia or reticular varicose veins C2 - varicose saphenous veins (diameter more than 3 mm) C3 - edema C4 - trophic changes in the skin and subcutaneous tissues a - hyperpigmentation and/or venous eczema b - lipodermatosclerosis and/or white skin atrophy C5 - healed ulcer C6 - open venous ulcer.

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