Klinichna khirurhiia. 2021 May/June; 88(5-6):23-27.
DOI: 10.26779/2522-1396.2021.5-6.23

Анатомо–гемодинамічні типи рецидиву варикозної хвороби нижніх кінцівок

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Anatomic–hemodynamical types of recurrence of the lower extremities varicose disease

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Реферат

Мета. Покращення результатів лікування хворих із рецидивом варикозної хвороби нижніх кінцівок після хірургічних втручань шляхом вивчення особливостей порушень регіонарної венозної гемодинаміки та визначення анатомо–гемодинамічних типів захворювання.

Матеріали і методи. Обстежено 85 хворих із рецидивом варикозної хвороби нижніх кінцівок після хірургічних втручань. Ультразвукове дуплексне сканування вен виконували на апараті «EnVisor» фірми «Philips» (Нідерланди) з використанням мультичастотного лінійного датчика з частотою 12 – 5 МГц для підшкірних вен і конвексного датчика з частотою 5 – 2 МГц для глибоких вен.

Результати. Виділено 8 анатомо–гемодинамічних типів рецидиву варикозної хвороби нижніх кінцівок. Проведене дослідження дозволило систематизувати причини і прояви захворювання. Об'єктивні критерії прогнозування розвитку рецидиву варикозної хвороби нижніх кінцівок можуть бути використані для оптимізації обсягу первинного хірургічного втручання і профілактики його ускладнень.

Висновки. Основними причинами розвитку рецидиву варикозної хвороби нижніх кінцівок є тактичні та технічні помилки хірургів під час виконання первинних хірургічних втручань у хворих з варикозною хворобою нижніх кінцівок. Вивчення особливостей розвитку рецидиву варикозної хвороби нижніх кінцівок дозволило розробити практичну анатомо–гемодинамічну класифікацію, яка систематизує основні причини рецидиву варикозної хвороби і є основою для формування тактики повторних операцій.

Ключові слова: рецидив варикозної хвороби нижніх кінцівок; ультразвукова діагностика.

Abstract

Objective. To improve the results of treatment in patients with postoperative recurrence of varicose disease of the lower extremities with the help of studying of peculiarities of disorders in regional venous hemodynamics and determination of anatomic–hemodynamic types of the disease.

Materials and methods. There were examined 85 patients with postoperative recurrence of varicose disease of the lower extremities. Ultrasonic duplex scanning of veins was performed on apparatus «EnVisor», manufactured by «Philips» (Holland) using a multifrequency linear sensor with the 12 – 5 MHz frequency for subcutaneous veins and a convex sensor with the 5 – 2 MHz frequency – for deep veins.

Results. There were delineated 8 anatomic–hemodynamical types of recurrences of varicose disease of the lower extremities. The investigation conducted have permitted to systematize the causes and signs of the disease. Objective criteria for prognostication of the varicose disease of the lower extremities recurrence may be applied for optimization of volume of primary surgical intervention and prophylaxis of its complications.

Conclusion. The main causes of the recurrence development in varicose disease of the lower extremities are tactical and technical mistakes of surgeons during performance of primary surgical interventions in patients, suffering varicose disease of the lower extremities. Studying of peculiarities in development of recurrence of varicose disease of the lower extremities permits to elaborate the practical anatomic–hemodynamical classification, which systematizes the main causes of the varicose disease recurrence and constitutes the base for the reoperations tactics elaboration.

Keywords: recurrence of varicose disease of the lower extremities; ultrasonographic diagnosis.

Introduction

Varicose veins recurrence (VVR) of the lower limbs is a significant medical and social problem in the treatment of venous diseases, as it occurs in 25–65% of operated patients and tends to progress to the manifestations of chronic venous insufficiency (CVI) [1, 2, 3]. In most cases, the reasons for its development are due to inadequate preoperative diagnosis, tactical and technical errors in primary surgery, progression of varicose veins (VV) due to genetically determined and acquired disorders of metabolic processes in the walls of veins [4, 5]. Despite the generalization of extensive experience in the diagnosis and treatment of VVR, the causes and ways of disease development remain insufficiently studied.

Statistical data concerning the frequency of VVR development vary considerably depending on the terms of observation, research methods, thorough diagnosis, and clinical classification. The published results are also difficult to compare with each other due to the lack of protocols of ultrasound ex-

aminations on the anatomical and hemodynamic features of VV, methods of performing primary operations [6, 7]. Typically, the basis for diagnosis is the presence of clinically detected recurrent veins, but many patients do not attach importance to these varicose veins and not all patients have minor symptoms that force them to seek re–intervention [8].

To characterize VVR, it is proposed to use the following classification [9, 10, 11]:

– A (anatomy) – necessary clarifications;

– T – localization of recurrent varicose veins: groin (Tg), thigh (Tt), popliteal fossa (Tr), shin (T1), other (To);

– S – source of recurrence: S0 – no source of reflux, S1 – pelvic veins and abdominal veins, S2 – saphenofemoral joint (SFJ), S3 – perforated femoral veins, S4 – saphenopopliteal joint (SPJ), S5 – perforated veins (PV) in the popliteal fossa, S6 – PV , S7 – PV of the distal part of the leg;

– R – degree of reflux: R + – clinically significant reflux, R – clinical value of reflux is unlikely, R? – the clinical significance of reflux is unknown;

– N – the nature of the source of recurrence, ie the connection with the preceding operation (in the area of operation); NSs – recurrence outside the area of primary surgery (1 – persistent pathology, 2 – new pathology, 3 – time of varicose veins unknown), NDs – recurrence in the area of the first operation (1 technical error, 2 – tactical error, 3 – the formation of new vessels, 4 – the reason is unknown, 5 – several reasons);

– C – filling from a persistent insolvent trunk of a great or small saphenous vein; C0 – absent;

– F – possible concomitant factors: Fg – general (family history, overweight, taking hormonal drugs), Fs – specific (primary deep vein insufficiency of the lower limbs).

This classification is not widely used due to the cumbersomeness and lack of ability to characterize all these factors. Despite this, scientists continue to look for answers to many questions related to the causes of VVR, the phenomenon of VVR is extremely variable and often due to the presence of many sources of blood reflux , which complicates the diagnosis and planning of reoperation [12].

Objective**.** To improve the results of treatment of patients with recurrence of varicose veins of the lower limbs after surgery by studying the features of disorders of regional venous hemodynamics and determining the anatomical and hemodynamic types of disease.

Materials and methods

We examined 85 patients with VVR after open surgery who were treated during 2016 – 2020. The mean age of patients was (39.3 ± 9.4) years (from 24 to 56). The ratio of men to women was 1: 2.32 (16:69). The duration of the disease varied from 8 to 29 years (average – 9.2 ± 3.5 years). According to the international CEAP classification of chronic venous insufficiency of the lower extremities [13], class C2 was registered in 4 (4.71%), C3 – in 14 (16.47%), C4 – in 47 (55.29%), C5 – in 11 (12.94%), C6 – in 9 (10.59%) patients.

Clinical examination of patients with VVR was performed using conventional methods of history taking, examination, palpation of varicose veins and soft tissues of the lower limb. To assess the degree and dynamics of edema used anthropometric method of measuring the circumference of the limb at four standard levels (middle of the foot, the level of the ankle joint, the level of the middle third of the shin, the level of the upper third of the shin).

The anatomy and hemodynamics of the subcutaneous venous system were studied by the color ultrasound method. Ultrasound characteristics of the anatomy of the subcutaneous veins and the ways of spreading pathological venous reflux were studied in all examined patients.

The study was performed in a vertical position of the patient. Transverse vein scanning made it possible to obtain information about the diameters of the veins, longitudinal – about the spread of reflux and the anatomical relationship of subcutaneous and deep vessels. Complete sagging of the veins during compression by the sensor was the main criterion for their patency. A manual muscle compression maneuver was used to detect blood reflux.

Examination of GSV began with the groin area. SPJ, possible zones of reflux formation and ways of its distribution were evaluated, anatomical relations of GSV or its stump with tributaries, presence of not removed GSV trunk or its segments were determined. When scanning the SSV, the presence and level of SPJ location were determined. An important step in the study was to identify alternative sources of reflux formation, including saphenous veins.

Perforated veins were located on the thigh and lower leg. When a failed PV was detected, its diameter was measured at the level of the muscular fascia and the duration of retrograde blood flow was recorded. The perforated vein in which retrograde blood flow lasting more than 0.5 s was registered was considered insolvent.

Ultrasound duplex scanning of veins was performed on an EnVisor device by Philips (Netherlands) using a multifrequency linear sensor with a frequency of 12–5 MHz for subcutaneous veins and a convex sensor with a frequency of 5–2 MHz for deep veins.

Results

Ultrasound examination of the subcutaneous venous system in patients with VVR, at first glance, visualised a chaotic picture that can not be systematized. However, a detailed segmental study of all areas of recurrent veins, determining the causes and development of reflux, identified the main anatomical and hemodynamic types of varicose veins recurrence (*Table*), which provided an opportunity to develop treatment tactics and determine the technical features of its implementation.

Discussion

According to the developed classification, the most common (31 patients – 36.47%) cause of VVR (type I) was inadequate treatment of the saphenofemoral junction with leaving an excessive stump of the great saphenous vein. The length of the stump played an important role in the severity of the

Anatomical and hemodynamic types of varicose veins recurrence of the lower limbs

VVR type	Source of blood reflux	Number of patients	
		n	%
I	Excess stump of the great saphenous vein	31	36,47
II	Residual trunk of GSV	4	4,71
III	Excess stump of the great saphenous vein and the residual trunk of the GSV	3	3,53
IV	Residual segments of the GSV trunk on the thigh and shin	15	17,65
V	"Serpentine varicose veins" in the groin area	9	10,59
VI	Small saphenous vein	10	11,76
VII	Perforated veins of the shin	6	7,06
VIII	Nonsaphenous varicose veins	7	8,23

manifestations of VVR. Short stump (up to 2 cm) was found in 22 (70.97%) patients, long (2 cm and more) – in 9 (29.03%).

With a linear stump size of up to 2 cm in 4 of 22 patients (18.18%, 4.71% of the total number of patients with VVR was preserved only the upper tributary of the GSV – superficial epigastric vein, with no signs of recurrence;

in 5 (22.73%, 6.02% of the total) revealed a network of varicose inflows.

In 12 cases (54.54%, 14.12% of the total) the main insolvent inflow was the trunk of the anterior accessory saphenous vein (AASV). The expansion of many branches of a vein on an anterolateral and lateral surface of a hip and a shin drew attention. Such a significant varicose transformation of the subcutaneous venous system was hemodynamically justified by a pronounced discharge of blood through the GSV in AASV.

In 1 patient, 3.23%, 1.18% of the total number of patients with VVR observed reflux from the stump of GSV into the posterior accessory saphenous vein (PASV) and Giacomini vein, with varicose veins on the posterior surface of the thigh.

When the length of the stump was more than 2 cm, the retrograde blood flow was distributed not only on the PASV, but also on many other branches that flowed into the stump. With such a loose redistribution of reflux, the clinical manifestations of VVR were less pronounced.

In the second anatomic–hemodynamic type of VVR in 1 of 4 patients, the trunk of the GSV vein was not ligated in the area of the junction and was not removed, despite the presence in the history of a reference to the performed phlebectomy. Reflux of blood on the trunk not removed for any reason, but ligated GSV in 3 (75%, 3.53% of the total number of patients with VVR) patients caused the preservation and even progression of the initial symptoms of the disease.

Our study refutes the theory supported by some surgeons of eliminating the symptoms of chronic venous insufficiency on the basis of isolated separation of GSV from the common femoral vein (CFV). Evaluating the hemodynamics of primary varicose veins, this fact can be explained by the fact that the SFJ, in fact, is the largest perforating vein that connects the GSV with the deep venous system. By itself, ligation of this perforator does not solve the problem of eliminating blood reflux along the trunk, because the GSV continues to be filled from many inflows, and its failed valve apparatus does not provide translational blood flow, resulting in retrograde blood flow and venous hypertension.

In type III VVR, the progression of ultrasound and clinical manifestations of the disease was more significant, because not only was reflux of blood due to the GSV stump into the junction tributaries, but also its spread to the GSV trunk through many collateral branches.

Quite unexpectedly, the second most common cause of VVR was the varicose transformation of segments of the residual trunk of GSV in the middle and / or lower third of the thigh (type IV VVR). The indication in the medical history of the performed phlebectomy suggested that the cause of recurrence of the disease was retrograde filling of the second, not removed during surgery, GSV trunk or expansion with the development of valvular insufficiency of the previously hypoplastic segment of GSV. The lack of direct links with the GSV implied the need to clarify the sources of filling the trunk in each case. Thus, in 5 patients (33.33%, 5.88% of the total number of patients with VVR) filling of the residual trunk of GSV was carried out from the area of varicose veins of the groin area, which could be formed due to neovasculogenesis. In 3 (20%, 2.35% of the total number of patients with VVR), the residual GSV trunk was filled with a medial femoral perforated vein located in the middle or at the border of the upper and middle thirds. The reason for the segmental expansion of GSV in the lower third of the thigh in 4 (26.67%, 4.71% of the total number of patients with VVR) was the discharge of blood from the superficial femoral vein through the perforated Dodd vein. In 1 case (6.66%, 1.18% of the total number of patients with VVR) filling and development of valvular insufficiency of the GSV segment was due to the failure of the medial knee perforator. In 2 patients (13.33%, 2.35% of the total number of patients with VVR) the residual trunk of GSV was varicosely transformed due to the spread of hypertension from dilated Giacomini venous inflows as a result of not eliminated or newly formed antigravity reflux.

A key point in the rapid progression of the disease in patients with type IV VVR was the involvement in the process of segments of the trunk of GSV with the subsequent development of a wide network of varicose veins. Identifying the source of reflux required careful examination of all available inflows, including in the basin of the small saphenous vein,

and incompeted perforated veins. It should be noted that in all cases, the truth of the GSV trunk was confirmed on the basis of its location in the interfascial space, which was visualized by ultrasound.

The nature of "serpentine varicose veins" (type V VVR) has not yet been definitively clarified. At the ultrasound location in the groin area was registered a network of tortuous veins, the diameter of which did not exceed 3 mm. Their origin could be due to the expansion of pre–existing small vessels, and neovasculogenesis. As a rule, the valvular device in the specified veins was absent. In some patients, atypically dilated, tortuous veins of the inguinal lymph nodes were involved in the process. Subcutaneous veins of the anterior–medial surface of the thigh were retrogradely filled from the "serpentine veins", which determined the further spread of blood reflux.

A large number of options for inflow of SSV into the deep venous system and the topography of its femoral branch implies ambiguity of the ways of blood reflux in this basin, the underestimation of which leads to inadequate interventions and recurrence of the disease. Because the structure of the SSV trunk is quite constant, the most common cause of VVR (type VI), detected in 5 cases (50%, 5.88% of the total number of patients with VVR), was a long stump vein. The expansion of the inflows of SSV, which flowed into the stump, led to the development of clinical manifestations of VVR on the posterior surface of the tibia. One patient 8 months after surgery developed antigravity reflux of blood from the stump of SSV into the vein Giacomini, due to the failure of sapheno–popliteal junction and the throwing of blood into the femoral branch of the small saphenous vein. These changes were the cause of dilation of the subcutaneous veins of the posterior thigh.

Blood reflux on the varicose trunk of the SSV was the cause of VVR in 3 (30%, 3.53% of the total number of patients with VVR). It should be noted that in these patients VVR in the system of SSV was detected within 2 to 4 months after removal of GSV and could not be a consequence of disease progression, which clearly indicated insufficient preoperative examination and, accordingly, the tactical error of the operating surgeon.

In one case, the cause of disease progression was not eliminated blood reflux on the stump of GSV and PASV, which spread into the vein Giacomini and trunk SSV.

Despite the slight variability of the ways of spreading pathological venous reflux after operations on SSV, the study once again showed that in most cases, VVR develops due to errors made during preoperative diagnosis and during surgery.

In 6 patients (10.59%) the only reason for the development of VVR was the failure of PV, while other sources of blood reflux were not registered (type VII RV). It should be noted that the combination of failure of perforated veins with other identified causes of VVR in patients of group II occurred in 78 (91.76%) cases.

Most often (5 patients – 83.33%, 5.88% of the total number of patients with VVR) recurrent PV was located in the paratibial zone of the medial surface of the leg. It should be noted that recurrence of the disease due to reflux of blood on PV most often progressed in patients with initially severe varicose veins (class IV–VI according to the CEAP classification).

In our opinion, the question of the causes of recurrence of blood reflux in the perforating veins closely echoes the question of the primary expansion of PV and their role in the pathogenesis of VV. In a general sense, the process of development of horizontal blood reflux has the following direction: under the influence of changes in superficial venous hemodynamics PV increase in diameter, resulting in impaired function of their valvular apparatus. According to clinical studies and ultrasound, it has been proven that the presence of failed PV causes a more severe course of the disease, so the elimination of horizontal reflux is an integral part of surgery.

The generally accepted criteria for assessing the failure of PV are not sufficiently substantiated, as they include only two unreliable criteria – the diameter of the vein (more than 2 mm), which often depends on the individual anatomical features of the patient, and blood reflux on it. In our opinion, the study of blood reflux in the short segment of the PV is methodologically inaccurate, because it can be mistakenly registered in the reverse flow of blood through the deep or subcutaneous system. In this regard, the indication for ligation of PV was its expansion (more than 2 mm) and direct connection with varicose veins.

An attempt to answer the question of whether the perforated veins dilated due to disease progression or were not ligated during the initial intervention did not yield definitive results. At the same time, the detection of a incompeted perforated vein in the projection of the postoperative scar could indicate both a careless attempt to ligate the PV and the variability of their anatomy. Ultrasound has shown that a perforated vein can have two legs located subfascially. At suprafascial ligation of PV one of legs could remain a source of a reflux that was the reason of development of VVR.

Analyzing the data of the study, we can draw the main conclusion: the failure of perforated veins is an integral part of the development of VV and VVR. Horizontal blood reflux, especially in the paratibial region of the lower extremity, contributes to the progression of CVI with the development of tissue trophic disorders. In this regard, it should be emphasized that the mandatory procedure of preoperative preparation of patients with PV and VVR is careful detection of failed PV with ultrasound and their intraoperative ligation. Due to the fact that in a large number of patients VVR was due to failure of perforating veins, before surgery, all patients should be informed about the possibility of recurrence not only through the surgeon's fault, but also due to disease progression and warned of the need for dispensary monitoring. at least for 5 years.

The presence of so–called "nonsaphenous varicose veins" is an exception to the standard options for the development of VV of the lower extremities. Underestimation of atypical blood reflux caused the development of VVR in 7 (8.23%) patients (type VIII VVR). It should be noted that in 5 cases (71.43%, 5.88% of the total number of patients) VVR developed in female patients during pregnancy due to reflux of blood from the veins of the pelvis. In 4 of them (80%, 4.7% of the total number of patients with VVR) noted dilation of external pu-

bic veins and blood reflux through the subcutaneous veins of the posterior medial and posterior femoral surface, in 1 – involvement in the process of Giacomini veins. One patient was found to have reflux of blood through the sciatic perforator due to hypertension in the pool of the internal iliac vein.

In 2 cases (28.57%, 2.35% of the total number of patients with VVR) there was dilation of the subcutaneous veins of the posterior surface of the leg, while SSV was removed. This fact suggested that the operation to remove the SSV was performed incorrectly, because initially there was a failure of the PV of the popliteal fossa.

Conclusions

1. The main reasons for the development of VVR are tactical and technical errors of surgeons during the performance of primary surgical interventions in patients with VV of the lower limbs.

2. The study of the peculiarities of the development of VVR of the lower limbs allowed to develop a practical anatomical and hemodynamic classification, which systematizes the main causes of VVR and is the basis for the formation of tactics of repeated operations.

Funding. There were no external sources of funding and support. No fees or other compensation were paid.

Authors' contribution. All authors made the same contribution to this article.

Competing interests. The authors have no conflict of interest.

Consent for publication. All authors approved the paper to be published.

Ethical approval. This article does not contain any studies with human participants performed by any of the authors.

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Надійшла 02.04.2021