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NEW APPROACH TO PATHOGENESIS AND TREATMENT OF TROPHYCAL ULCERS

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Abstract. *Introduction.* Trophic ulcers are one of the most common medical ailments worldwide. Treatment of patients with trophic ulcers is difficult not only because they often recur, but also because they are difficult to treat. *Purpose.* Study the clinical features and present our own results of combined methods of treatment of trophic ulcers of venous etiology. *Materials and methods.* The study involved 100 patients with trophic ulcers of the lower extremities of venous, diabetic and atherosclerotic etiology, who had a history and were treated for varicose veins of the lower extremities and post-thrombotic disease. Examination of patients included questionnaires, clarification of complaints, anamnesis data, causes of development, examination and ultrasound diagnostics (duplex examination). The examination of the ulcer included determining the location, size, appearance, base of the wound, the level of exudation and assessment of the condition of the skin around the defect. *Results.* The etiological factors in the development of trophic ulcers were: varicose disease with chronic venous insufficiency – 25 patients, post-thrombotic disease – 55 patients. All patients underwent only conservative treatment of trophic venous ulcers with the appointment of a placenta compositum. After the treatment of a group of patients, out of 100 patients with venous trophic ulcers, 89% were cured within 4 months, 8% were cured within 2 years, and in 3 % ulcers did not heal for more than two years. The average duration of the course of ulcers was 12 months. The average size of trophic ulcers based on the results of the analysis was 8 cm². Complete healing of venous ulcers was most often observed in the age group 61–73 years in 30% patients. *Conclusion* The use of the compositum placenta contributed to the acceleration of wound healing, which made it possible to obtain more effective results.

Key words: trophic ulcers, varicose veins with chronic venous deficiency, posttrombotic disease, compositum placenta.

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НОВЫЙ ПОДХОД К ПАТОГЕНЕЗУ И ЛЕЧЕНИЮ ТРОФИЧЕСКИХ ЯЗВ

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Резюме. Введение. Трофические язвы являются одним из наиболее распространенных медицинских заболеваний во всем мире. Лечение пациентов с трофическими язвами затруднено не только потому, что они часто рецидивируют, но и потому, что их трудно лечить. Цель: изучить клинические особенности и представить собственные результаты комбинированных методов лечения трофических язв венозной этиологии. Материалы и методы. В исследовании приняли участие 100 пациентов с трофическими язвами нижних конечностей венозной, диабетической и атеросклеротической этиологии, которые имели в анамнезе и проходили лечение по поводу варикозного расширения вен нижних конечностей и посттромботической болезни. Обследование пациентов включало анкетирование, уточнение жалоб, данных анамнеза, причин развития, осмотр и ультразвуковую диагностику (дуплексное обследование). Обследование язвы включало определение местоположения, размера, внешнего вида, основания раны, уровня экссудации и оценку состояния кожи вокруг дефекта. *Результаты*. Этиологическими факторами развития трофических язв были: варикозная болезнь с хронической венозной недостаточностью – 25 пациентов, посттромботическая болезнь – 55 пациентов. Всем пациентам проводилось только консервативное лечение трофических венозных язв с назначением композитума плаценты. После лечения группы из 100 пациентов с венозными трофическими язвами, 89 % были излечены в течение 4 месяцев, 8 % были излечены в течение 2 лет, а у 3 % язвы не заживали более двух лет. Средняя продолжительность течения язв составила 12 месяцев. Средний размер трофических язв по результатам анализа составил 8 см². Полное заживление венозных язв чаще всего наблюдалось в возрастной группе 61-73 года - у 30 % пациентов. Заключение. Применение плаценты композитум способствовало ускорению заживления ран, что позволило получить более эффективные результаты.

Ключевые слова: трофические язвы, варикозная болезнь с хронической венозной недостаточностью, посттромботическая болезнь, композитум плаценты

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Trophic ulcers of venous etiology are defects in the skin and underlying tissues due to impaired venous circulation [1]. Trophic ulcers are one of the most common medical ailments worldwide, affecting about 1-2% of the working adult population in the world and about 5% of the elderly, and also leads to temporary or complete disability and disability [2]. Trophic venous ulcers have been known for a long time and are often encountered in clinical practice. They account for 80% of all leg ulcers [3].

Trophic ulcers belong to the highest clinical class of venous diseases according to the CEAP-C6 classification. The cause of a leg ulcer can usually only be determined based on the history and physical examination. The main causes of ulcer development are varicose veins with chronic venous insufficiency and late effects of deep vein thrombosis (post-thrombotic or post-phlebitic syndrome) [4].

Despite the rapid development of modern scientific technologies and the accumulated centuries-old experience in solving many interdisciplinary problems, the issues of prevention and effective treatment of trophic ulcers remain relevant.

This is largely due to the current trend towards an increase in the number and increase in the prevalence of the so-called diseases of civilization, despite the numerous and promising scientific discoveries and achievements in the field of nanotechnology, robotics, genetic engineering, etc.

Trophic ulcers in the lower extremities are the result of various diseases that disrupt local hemodynamics of the arterial (chronic arterial insufficiency with obliterating diseases of the arteries), venous (post-thrombophlebitic syndrome, varicose veins), lymphatic systems, as well as developing against the background of diabetic micro-, macroangiopathy and neuropathy, including the microcirculatory level of the lesion.

Due to the development, the following types of trophic ulcers were distinguished:

- Non-healing venous trophic ulcers with varicose veins. In two out of three cases, trophic leg ulcers are caused by venous etiology.
- Arterial ulcers can form due to damage to the artery by atherosclerotic plaques.
- A trophic ulcer in diabetes appears due to a violation of metabolic processes in tissues.
- Ulcers, as a complication of systemic diseases collagenosis, inflammation of the walls of blood vessels.

- Congestive ulcers may appear due to congestive processes caused by cardiac pathology.
 - Pyogenic purulent ulcers.
 - Ulcers due to skin cancers.
 - Due to burns, including radioactive ones.
 - Ulcers after frostbite with tissue necrosis.
- There is another classification of this skin pathology according to the depth of tissue damage:
- $\bullet\,\mbox{The}$ surface layers of the skin are damaged, small sores appear.
- All layers of the skin and subcutaneous tissue are involved in pathological processes.
 - Severe deep tissue damage is diagnosed.

There is another classification of uncomplicated trophic ulcers (according to V. Ya. Vasyutkov. It determines the clinical stage of damage:

- 1. Damage on dry, thinned skin. Change in the color of the skin of the surrounding tissues, swelling.
- 2. The wound deepens. All layers of the skin are involved in the pathological process. It becomes blueviolet in color with a weeping hearth in the center.
- 3. The bottom of the ulcer thickens. There is swelling and soreness of the surrounding tissues.
- 4. Growth of the wound. All new tissues are involved in the process. The edges of the ulcer may bleed.
 - 5. The wound is cleaned. Healing is observed.
 - 6. The ulcer has healed. Scar formation.

The initial stage of the disease is manifested by alarming symptoms long before the appearance of direct wounds on the legs.

- First, blue or burgundy spots appear on the skin. This is due to areas of accumulation of red blood cells in the stage of decay.
- At the next stage, the skin on the leg becomes much paler than the rest of the skin, this is due to a violation of blood circulation.
- The skin over whitish spots becomes thin, itching and pain appear.
- Even a slight mechanical impact can cause a wound. The injury does not heal when treated with antiseptics, healing ointments.
- Common location of trophic ulcers feet, lower leg. At the site of the ulcer, necrotic processes develop. The depth and scale of damage increases.
- The wound becomes a favorable place for infection to penetrate into the tissues and bloodstream. Possible suppuration.

It is important to understand that a trophic ulcer is not an independent disease, but the result of a serious pathology that developed long before the appearance of non-healing wounds. Accordingly, successful treatment of a venous trophic ulcer is possible only with the normalization of the general condition, with parallel treatment of the underlying disease. And complex therapy should be carried out exclusively under the supervision of a doctor. The triggering factor for the occurrence of trophic ulcers are congenital and acquired diseases and injuries that cause the development of various pathological processes in the tissue (ischemia, hypoxia), microcirculatory (microthrombosis and sludge of blood cells, protein extravasation into the perivasal space with the accumulation of fibrin in the tissues) and cellular (activation of leukocytes with the release of lysosomal enzymes) levels.

Additionally, there are local and systemic shifts that form the syndrome of blood hyperviscosity. The final pathophysiological outcome of the development of trophic ulcers is severe wound sepsis and multiple organ failure syndrome.

Chronic venous insufficiency causes the occurrence of 70–90 % of trophic leg ulcers, which are observed in 1–2 % of the working population, and in the retirement age group their frequency reaches 34 % [5, 6].

Obliterating atherosclerosis is the cause of trophic ulcers of the lower extremities in 8 % of cases. Diabetic micro-, macroangiopathy and distal neuropathy cause the formation of trophic ulcers in 3 % of cases. Since the pathology of the venous system plays the most significant role in the formation of trophic ulcers of the lower extremities, the etiopathogenetic mechanisms of its development should be considered in more detail.

When studying the pathogenesis of varicose veins, trophic ulcers, it is necessary to take into account their versatility and complexity. According to modern concepts, under the influence of a number of risk factors (burdened heredity, female gender, age, etc.), conditions are formed for the development of venous hypertension, dilatation of the veins, followed by the occurrence of valvular disorders and backflow of blood. For CVI, as for any chronic pathology, the formation of the so-called vicious circle with the participation of all of the above factors and mechanisms, to which inflammatory changes are attached, is characteristic.

Valvular insufficiency of various parts of the venous bed of the lower extremities leads to the appearance of pathological retrograde blood flow, which is the main factor in damage to the microvasculature. In the presence of various risk factors and under the influence of gravity in the venous knee of the capillary, pressure increases, reducing the arteriovenular gradient necessary for normal perfusion of the microvasculature. As a result, first periodic, and then permanent tissue hypoxia occurs.

A change in body position and an uneven load on various parts of the venous bed of the lower extremities triggers another little-studied mechanism, called mechanotransduction, or shear forces. So, under the influence of pressure constantly changing in strength and direction, a gradual loosening of the connective tissue frame of the venule wall occurs. At the same time, disruption of the normal intercellular relationships of the venous capillary endothelium leads to the activation of genes encoding the synthesis of various adhesion molecules.

The flow of blood through the venous section of the microvasculature also undergoes certain changes. In patients with venous hypertension, the so-called "rolling of leukocytes" is observed – they roll over the endothelium with already activated adhesion receptors [7].

Leukocyte aggression towards the venous wall is considered to be the pivotal mechanism around which a vicious circle of disease pathogenesis is formed [8]. Subsequently, the processes of dysfunction and dysregulation of the endothelium, damage to the structure of the venous wall by metabolic products of activated leukocytes, hypertrophy of the venous wall as a result of phenotypic modulation of smooth muscle cells become the main links in the pathogenesis of varicose veins.

It should be noted that before the appearance of trophic ulcers as a late stage of varicose veins, many patients have various forms of systemic pathology of arterial vessels: coronary heart disease, arterial hypertension, diabetes mellitus, obliterating atherosclerosis of the vessels of the lower extremities, etc. The course of these diseases, in turn, can also be complicated by the formation of ulcers in the lower extremities, but already due to violations of arterial blood flow. Microcirculation disorders lead to pain syndrome and trophic disorders with the development of necrotic lesions of soft tissues.

Thus, a rather urgent question is on the agenda: how should the tactics of managing such patients be differentiated depending on the predominance of lesions of the venous or arterial vascular bed.

Chronic venous leg ulcers also represent a significant economic burden and the cost of treating them is a significant part of the health care budget. This disease affects almost all aspects of everyday life: sleep is often disturbed, mobility and ability to work are limited. Proper diagnosis is important to avoid inappropriate treatment that can slow wound healing, cause it to worsen, or harm the patient.

Treatment of patients with trophic ulcers is difficult not only because they often recur, but also because they are difficult to treat. To date, several treatments and protocols have been reported, all of them mainly focused on the outpatient treatment of venous ulcers [9].

There are various methods of treatment, such as elastic compression, local treatment, pharmacological drugs and surgical treatment.

The purpose of the study is to study the clinical features and present our own results of combined methods of treatment of trophic ulcers of venous etiology.

Materials and methods

The study involved 100 patients with trophic ulcers of the lower extremities of venous, diabetic and atherosclerotic etiology, who had a history and were treated for varicose veins of the lower extremities and post-thrombotic disease for the period from 2017 to 2022. The criterion for inclusion in this study was the presence of trophic ulcers of venous etiology for more than three months.

Exclusion criteria: damage to peripheral arteries, patients after surgical treatment of varicose veins, endovasal laser obliteration, sclerotherapy, trophic ulcers of non-venous etiology. If the above pathologies were detected in patients, they were excluded from the study.

Examination of patients included questionnaires, clarification of complaints, anamnesis data, causes of development, examination and ultrasound diagnostics (duplex examination) in all 100 patients.

Examination of the ulcer should include location, size, appearance, base of the wound, level of exudation, and evaluation of the skin around the defect.

Surrounding areas should be examined for pain, edema, erythema, temperature, induration, discoloration, maceration, dryness, scarring from previous wounds, gangrenous lesions of the fingers, capillary refill, and varicose veins. It is important to remember that venous and arterial diseases can coexist in the same patient. Venous ulcers are significantly different from arterial ulcers.

Duplex scanning is a non-invasive method of choice in venous pathology, which provides valuable information regarding venous blood flow, thrombotic obstruction, patency and reflux, and the effect of muscle contraction. It was performed in all 100 patients.

Results

The ratio of men and women was as follows: men - 60 (60 %), women - 40 (40 %).

The average age of the examined patients was 75 years (fig. 1).

The main complaints of the patients were: the presence of an ulcer, itching, swelling of varying intensity, an unpleasant odor without discharge (fig. 2).

According to the history of patients, the etiological factors in the development of trophic ulcers were: varicose disease with chronic venous insufficiency – 25 patients, post-thrombotic disease – 55 patients.

To assess the effectiveness of the treatment, the wound area was measured before treatment during treatment and at the end of treatment (fig. 3). Such measurements must be accurate and reliable, and it is desirable to conduct them by non-contact methods to avoid cross-contamination.

Based on the data obtained, the following localization of trophic venous ulcers on the lower extremities in relation to the anatomical regions was revealed.

45~% of patients had bilateral leg ulcers, 33~% had ulcers only on the left limb, and 22 % had ulcers only on the right.

All patients underwent only conservative treatment of trophic venous ulcers with the appointment of a placenta compositum. Below we provide detailed information about the composition and use of this drug.

Release form: solution for injection – 5 ampoules of 2.2 ml.

Registration certificate No. UA/2465/01/01 dated 12/24/2004.

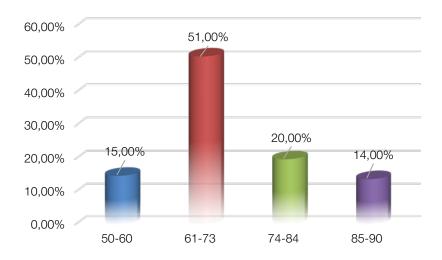


Figure 1. Distribution of patients (n = 100) by age groups **Рисунок 1.** Распределение пациентов (n = 100) по возрастным группам

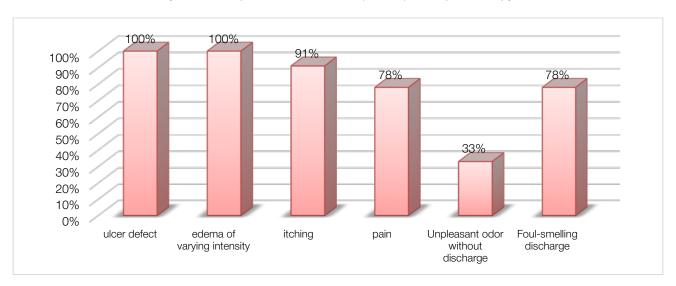


Figure 2. Main symptoms
Рисунок 2. Основные симптомы

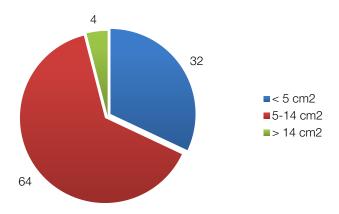


Figure 3. Area of ulcers **Рисунок 3.** Площадь язв

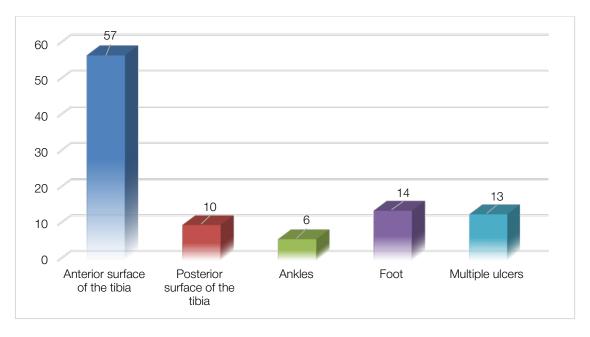


Figure 4. Localization of trophic ulcers in relation to the anatomical regions **Рисунок 4.** Локализация трофических язв по отношению к анатомическим областям

Composition: 2.2 ml injection solution contains: Placenta suis D6, Embryo suis D8, Vena suis D8, Arteria suis D10, Funiculus umbilicalis suis D10, Hypophysis suis D10, Secale cornutum D4, Acidum sarcolacticum D4, Nicotiana tabacum D10, Strophanthus gratus D6, Aesculus hippocastanum D4, Melilotus officinalis D6, Cuprum sulfuricum D6, Natrium pyruvicum D8, Barium carbonicum D13, Plumbum jodatum D18, Vipera berus D10, Solanum nigrum D6 – 22 µl each. Excipients: isotonic (0.9 %) sodium chloride solution q.s.

Indications:

- Violations of peripheral blood circulation and microcirculation in case of:
- atherosclerosis, diabetes mellitus, obliterating endarteritis, trophic ulcers, bedsores, residual effects of stroke, corneal clouding, degenerative and vascular diseases of the inner ear and fundus, elephantiasis, varicose veins, hypogalactia
 - Fetoplacental insufficiency
 - Alopecia

Contraindications: individual sensitivity to the components of the drug.

Side effects: not identified.

Single dose: adults: usually 1 ampoule.

The drug is administered subcutaneously, intravenously, intramuscularly, including segmentally, at acupuncture points, by mesotherapy, if necessary – intra-

venously 1–2 times a week; in acute cases – 1 ampoule daily, for 2–3 days, or every other day No. 3–5.

If parenteral administration is not possible, the drug can be taken orally (in the form of "drinking ampoules"): shake a single dose into the mouth or dissolve in 50 ml of water and drink throughout the day.

Usually the course of treatment lasts 3–6 weeks. Interaction with other drugs: no features.

Special instructions:

With hyperthyroidism, thyrotoxicosis, due to the content of the Plumbum jodatum D18 component containing iodine in the preparation, an increase in the functional activity of the thyroid gland is possible. Therefore, such patients are recommended to carry out constant medical supervision and in case of increased symptoms of thyrotoxicosis, it is necessary to reduce the dose of the drug and increase the intervals between its doses or temporarily stop taking the drug.

Pharmacological properties: the composition of the drug Placenta compositum includes 18 natural potentiated components:

suis organ:

Placenta suis D6

(from placenta) Peripheral circulatory disorders, bedsores, painful skin cracks, eczema, ulcers. Cramps of the calf muscles. Scleroderma.

Embryo suis D8

(from embryo) Arteriosclerosis, muscular dystrophy.

Means for therapy of cellular phases (according to the table of homotoxicosis).

Vena suis D8

(from the vein wall) Circulatory disorders, venous congestion, leg ulcers, varicose veins. Intermittent lameness. Periarteritis. Cramps of the calf muscles.

Arteria suis D10

(from artery wall) Circulatory disorders. Gangrene of the extremities, including diabetes mellitus. Cramps of the calf muscles. Periarteritis. Intermittent lameness.

Funiculus umbilicalis suis D10

(from umbilical cord tissue) Any chronic diseases and connective tissue damage. Arteriosclerosis. Vascular diseases. Blood supply disorders. Vegetative dystonia.

Autoaggressive diseases. collagenoses. Otosclerosis. Elephantiasis.

Hypophysis suis D10

(from pituitary tissue) Violations of the functions of the pituitary and endocrine glands. Violations of the functions of enzymes. Vertigo. Violation of the functions of the connective tissue biocatalysts:

Acidum sarcolacticum D4 (dextrorotatory isomer of lactic acid) Disorders of cellular nutrition. Diabetes. Muscle pain.

Natrium pyruvicum D8

(sodium salt of pyruvic acid) Violations of the central and peripheral circulation. Gangrene, ulcers of the leg. Peripheral circulatory disorders in diabetes mellitus (diabetic gangrene).

Frostbite. vascular collapse. Angina. Migraine. vegetable:

Secale cornutum D4

(Ergot is a fungus that parasitizes on ears of rye) Disturbances in the blood supply to tissues in diseases of peripheral arteries. Feeling of numbness of the limbs, crawling. Flabby, pale, cold skin, pinpoint hemorrhages on the skin. Gangrene. Varicose ulcers. Vegetative regulation disorders. Violations of mental and intellectual functions (difficulty thinking and speech, forgetfulness, distortion of perception, agitation). Flushing to the head, dizziness, tinnitus, hearing loss.

Nicotiana tabacum D10

(Tobacco – dry leaves) Collapse with cold sweat, pallor and coldness of extremities. Angina. Migraine.

Strophanthus gratus D6

(Strophanthus attractive – mature seeds) Cardiac decompensation.

Tachycardia.

Aesculus hippocastanum D4

(Horse chestnut – fresh mature seeds, peeled) Venous stasis, hemorrhoids, varicose veins, paresthesia.

Headaches in the frontal and occipital parts with dizziness, flashing "flies" before the eyes.

Melilotus officinalis D6

(Clover melilot – fresh leaves and flowers) Headaches with reddening of the face, feeling of pressure over the eye sockets, cold extremities. Congestion of blood to various organs. Renal hypertension. Stagnation in the female genital organs. Nervous and physical exhaustion.

Solanum nigrum D6

(Black nightshade – all fresh flowering plant) Irritations of the brain and meninges.

Increased nervous excitability. Convulsions, disorientation.

mineral:

Cuprum sulfuricum D6

(copper sulfate) Muscle cramps that begin in the fingers and toes and spread throughout the body. Arteriosclerosis.

Barium carbonicum D13

(barium carbonate) High blood pressure. Arteriosclerosis. Physical weakness, loss of strength, feeling of fatigue and numbness in the limbs. Itching of the leg. Noise, ringing in the ears.

Plumbum jodatum D18

(lead iodide) General calcification of vessels, high blood pressure.

Progressive muscular atrophy. Weak paresis of any origin.

animals:

Vipera berus D10

(Poison of the common viper) Weakness of cardiovascular activity. Fainting.

Feeling of heaviness in the legs. Leg ulcers.

The combination of extracts from various organs and biological agents has a stimulating effect in case of violations of the peripheral blood supply, incl. blood supply to the brain and heart.

In general, the drug Placenta compositum has a complex effect:

- improves peripheral circulation and microcirculation (venotonic, vasodilating, antispastic)
- trophic (in all tissues and the pituitary gland, provides hormonal regulation of the body)

Additionally, the drug provides:

- analgesic,
- detoxification effect.

In addition to the main indications, Placenta compositum is prescribed as part of complex therapy for: gangrenous ulcers, neurocirculatory dystonia, nodular erythema, embolism, residual effects after encephalitis, constantly cold extremities, dysmenorrhea, endometritis, nephrosis, migraine, neuralgia, mastodynia, myalgia with rheumatism, syndrome Cushing disease and other diseases that occur with impaired peripheral circulation.

Endovasal laser obliteration, sclerotherapy, and other methods for eliminating venous reflux were not used.

Discussion

Methods of treatment of trophic ulcers of venous etiology consisted of complex treatment. It included: the correct position of the leg, local treatment – the treatment of an ulcer and the imposition of a compression bandage or knitwear using wound coverings.

Each patient underwent obligatory treatment of the ulcer in the area of the defect by removing fibrin and necrotic tissue in order to ensure the formation of granulation tissue, adequate epithelialization and reduce the likelihood of infection. The jet was washed with sterile saline warmed up to 37 °C. At the next stage, wet dressings based on Ca2+ alginate obtained from Suprasorb A brown seaweed (Lohmann, Austria) were used, guided by the principle of wound management of trophic ulcers in a humid environment. Alginate dressings are designed to form a soft gel upon contact with wound exudate.

On contact with exudate, this dressing forms a gel that promotes rehydration of ulcers with moderate to severe drainage, promoting autolytic clearance. The calcium ions present in the dressings help control bleeding by promoting blood clotting. Currently, there are no data indicating any differences in the timing of wound healing between alginate and hydrocolloid dressings [10–12].

The next step in treatment was compression therapy, which is still considered the primary treatment for venous ulcers, and its ability to heal venous ulcers is unequivocally supported by many studies, such as the Cochrane Review, which provides evidence that venous ulcers heal faster with compression than without its use [13]. Compression, due to the frictional force between the knee socks together they

provided even more pressure than the knee socks of the 3rd degree of compression.

After the treatment of a group of patients, out of 100 patients with venous trophic ulcers, 89 (89 %) were cured within 4 months, 8 (8 %) were cured within 2 years, and in 3 (3 %) ulcers did not heal for more than two years.

The average duration of the course of ulcers was 12 months. The average size of trophic ulcers based on the results of the analysis was 8 cm².

Complete healing of venous ulcers was most often observed in the age group 61-73 years in 30 (30 %) patients.

Trophic ulcers are the most common of all lesions on the legs, with a high morbidity and have a negative impact on quality of life. Their treatment remains an urgent medical and social problem. In most cases, chronic venous ulcers of the lower extremities are formed due to post-thrombophlebitic syndrome. And in the vast majority of patients, they are localized on both lower extremities.

Comprehensive treatment is very important for the successful outcome of treatment, as a result of its use in the vast majority of patients, trophic ulcers completely heal within four months. A prerequisite for the management of trophic ulcers is the use of a compositum placenta, wound debridement, and exudate control using wet dressings based on Ca 2+ alginate.

The main and basic principles of treatment are to improve blood circulation and improve venous return, for this purpose, compression therapy is used, which reduces venous hypertension, while increasing the healing rate of venous ulcers in most patients.

Such treatment is effective and is the basis of therapy for this pathology.

Speaking about the treatment of patients with trophic ulcers, it should be remembered that the main efforts of doctors should be aimed at the speedy healing of the ulcer, preventing the progression of the process and the development of complications, and, ultimately, the maximum possible improvement in the quality of life of patients.

Patients with trophic ulcers of the lower leg, combined with hemodynamically significant damage to the arteries, undergo complex conservative treatment (correction of microcirculatory disorders, relief of infection, careful use of elastic compression), based

on the results of which the issue of surgical intervention is decided.

Currently, in preoperative preparation and postoperative rehabilitation, as well as as an independent type of treatment, more and more importance is attached to methods of conservative therapy that have a positive effect on microcirculatory processes in tissues, since its effectiveness is largely degree depends on the patient's quality of life and further prognosis of his disease [14–16].

Despite the long existence of the problem of trophic lesions of the lower extremities, there are no specific treatment regimens for the combined genesis of ulcers. There are many publications in the literature that highlight the features of managing patients with chronic venous insufficiency or chronic arterial insuffi-

ciency of the lower extremities, and only a few - with trophic ulcers of mixed etiology. Given the multifactorial nature of this disease, conservative therapy should include drugs that act simultaneously on several links of pathogenesis. The main goals of the use of pharmacotherapy should be to reduce cytokine aggression, relieve severe pain, and normalize hemo-rheology.

Conclusion Thus, although trophic ulcers are not a common cause of death, they are the cause of significant morbidity and disability, and timely diagnosis and treatment can prevent the development of severe complications. The use of the compositum placenta contributed to the acceleration of wound healing, which made it possible to obtain more effective results.

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