UDC 616.43
DOI 10.59598/ME-2305-6053-2025-115-2-8-16

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A MODERN APPROACH TO THE MANAGEMENT OF TYPE 2 DIABETES MELLITUS COMPLICATED BY DIABETIC FOOT SYNDROME

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Hyperglycemia, characteristic of diabetes mellitus, contributes to the production of oxidative stress agents that cause impaired immune function and the development of a chronic inflammatory process. These factors underlie the development of late complications of diabetes mellitus, such as coronary heart disease, unstable angina pectoris, myocardial infarction, peripheral microangiopathy and other articular diseases that worsen the prognosis for diabetic foot syndrome. Inflammatory processes and disorders of the immune system contribute to an imbalance of tissue homeostasis, are associated with the appearance of chronic ulcers and the need for amputation of the lower extremities. The consequences of chronic wounds create an unfavorable reality of disability and mental stress for patients. Both patients and medical staff will undoubtedly benefit from a deeper understanding of the pathogenesis and pathophysiology of various non-healing ulcers. New knowledge was acquired by comparing the similarities between chronic wounds of different natures with their differences from acute wounds.

Key words: type 2 diabetes mellitus; diabetic foot syndrome; vitamins; micronutrients; macronutrients; management

INTRODUCTION

Normal blood sugar levels are regulated by the body with the help of several organs, such as the liver, the autonomic nervous system, and the endocrine glands. Homeostasis of blood glucose levels is vital for humans. The liver plays a key role in responding to changes in blood glucose levels by activating hormonal reactions. A decrease in glucose levels leads to the release of glucagon, and an increase leads to the release of insulin by the pancreas. Any disruption in the production of these hormones can lead to diabetes and improper regulation of blood glucose levels. Type 2 diabetes mellitus is a metabolic syndrome characterized by metabolic disorders and hyperglycemia. It occurs due to insufficient insulin production by beta cells of the pancreas and insufficient sensitivity of the body to insulin. The initial causes of this disease are extremely complex and are most often associated with metabolic and immune processes. Risk factors contributing to the development of complications in diabetes mellitus include diet, overweight, obesity, smoking, alcohol consumption, physical activity level, hormonal balance, viral infections, atherosclerosis, cardiovascular diseases, insulin shock, diabetic ketoacidosis, and hyperosmolar hyperglycemia. Uncontrolled factors such as genetics and age are beyond people's control. However, people can reduce the impact of controlled risk factors such as exercise and proper nutrition by improving their healthy habits, which can reduce the likelihood of complications [3, 25].

According to the definitions of the World Health Organization and the International Diabetes Federation, diabetic foot syndrome is a serious complication of diabetes mellitus, which is manifested by ulcers and wounds on the tissues of the foot. Diabetic foot ulcers are usually associated with neuropathy, ischemia, and trauma. Diabetic peripheral neuropathy leads to insensitivity and weakening of the foot tissues, increasing the probability of ulceration due to repeated exertion and impaired blood supply. Hyperglycemia in diabetes causes metabolic disorders that interfere with wound healing. These include glycation, oxidative stress, skin dysfunction and inflammation, as well as increased stiffness of the extracellular matrix. This leads to a chronic inflammatory condition, circulatory dysfunction, and poor oxidative tissue perfusion due to lack of oxygen [30]. However, this process can be prevented and controlled through proper and accurate pharmacological therapy, moderate physical activity, as well as a balanced diet and dietary supplements to compensate for the deficiency of certain trace elements that play an important role in glucose and insulin metabolism.

The deficiency of nutrients in the body of patients suffering from type 2 diabetes mellitus leads to a decrease in fat deposits under the skin in areas with pressure, increasing the probability of pressure wounds, reduces the synthesis of collagen necessary for healing, reducing energy resources and affecting the mobility of

the patient, affects the body's response to infection, impairs immunity and contributes to the appearance of thin and flaky skin, which eventually leads to the formation of wounds. Slowing down the wound healing process in diabetic foot is a complex problem depending on various factors, and improper nutrition only worsens this situation [19, 26].

PHYSIOLOGY OF WOUND HEALING

The process of normal wound healing, during which the skin barrier is restored and closed after damage, can be divided into three interrelated phases, including five key stages: inflammation with hemostasis processes, proliferation with granulation and repidermalization processes, as well as the remodeling phase, which can vary in duration, with nutrients playing an important role at each stage.

The first stage of the healing process is hemostasis and the formation of a temporary wound matrix. After the endothelium is damaged, the components of the extracellular matrix bind and activate circulating platelets, which then undergo adhesion and aggregation. Damaged tissue and aggregated platelets trigger external and internal clotting pathways, working together to stabilize the fibrin-platelet clot. This process creates the basis for migration and proliferation of other cells involved in wound healing and provides a reservoir for cytokines and growth factors.

Inflammation is a significant, non-specific, natural immune response aimed at destroying tissues and clearing cellular and extracellular waste, as well as remnants of pathogens. This process usually lasts about 6 days after injury, when the number and phenotype of infiltrated leukocytes return to baseline values. However, in the presence of an external stimulus that harms tissues, inflammation can become chronic and more intense. Like platelets, white blood cells produce inflammatory cytokines that create a chemotactic gradient to attract additional white blood cells, enhancing the inflammatory process. These include interleukin (IL)-1α, IL-1β, IL-6, IL-8, tumor necrosis factor (TNF)-α, platelet growth factor and transforming growth factor (TGF)-β. As a result, this process activates matrix-producing cells, which promotes rapid deposit of connective tissue for subsequent phases of inflammation and proliferation. This recovery process is mainly due to the mediated action of histamine by vasodilation, which prevails over vasoconstriction approximately 10 minutes after injury. Released from mast cells, histamine forms pores in blood vessels, promoting the release of protein and white blood cells into the wound. In the initial phase of the leukocyte response, neutrophils predominate during the first two to five days, but by about the third day macrophages become dominant. Neutrophils perform three main functions: they produce free radicals through the myeloperoxidase pathway to kill bacteria, clean the wound by isolating proteolytic enzymes that decompose some tissues, and phagocytize dead bacteria and matrix residues. After completing their tasks, neutrophils usually undergo apoptosis and are removed by macrophages. Monocytes migrate to the wound, where they turn into macrophages, and become key regulatory cells in the process of inflammation. Macrophages with high activity phagocytize non-functional host cells, neutrophils filled with bacteria, damaged matrix, foreign debris, and residual bacteria.

This leads to the next stage - the proliferation phase, which begins 3-5 days after injury and can last up to 3 weeks. At this stage, cellular activity predominates in response to elevated cytokine levels. The skin repair process begins when keratinocytes and epithelial stem cells begin to proliferate and migrate. With a decrease in inflammation, the skin is restored, and endothelial cells and fibroblasts begin to accumulate in the wound area for the synthesis of granulation tissue. Angiogenesis and fibroplasia occur simultaneously, providing the body with oxygen, nutrients, and a hydrated matrix to maintain a high level of cellular activity. Fibroblasts play a key role in the production of extracellular matrix substances (collagen, fibronectin, glycosaminoglycans, proteoglycans and hyaluronic acid) that interact with cells, regulating their migration, growth, and differentiation.

The process of reduction and remodeling is the final stage of wound healing. It usually begins two weeks after injury and can last up to two years. Under the influence of mechanical stress and cytokines, including TGF-β, fibroblasts increase the expression of α-smooth muscle actin, turning into myofibroblasts. These cells shrink the wound through interaction with the integrin receptor and extracellular matrix components such as fibronectin and collagen. In the process of remodeling, fibroblasts increase the synthesis of more durable type I collagen, while matrix metalloproteinase destroys disorganized old collagen. A carefully balanced ratio between collagen synthesis and destruction contributes to the formation of a normal scar, in which collagen fibers are rearranged into small parallel bundles along the stretch lines.

Previously, these stages were considered separate and sequential, but modern research suggests that such a traditional view is outdated. Wound healing occurs through a complex mechanism that starts immediately after skin damage, and the phases overlap and even occur in parallel both in time and in the space of the wound. The time required for wound healing depends on several factors, such as the type and size of the wound, the age of the person, his physical condition, the presence of comorbidities, the location of the wound and treatment methods.

Chronic wounds are characterized by a disrupted healing process, which does not allow achieving stable anatomical and functional results in the expected time. Unlike the typical sequential healing process, they get stuck at the stage of inflammation and remain immune to adequate treatment. Further tissue damage supports a prolonged and increased inflammatory state of the wound, accompanied by abundant penetration of neutrophils, reactive oxygen species and destructive enzymes, which supports the stagnant process. Also, the presence of a wound affect's nutrition due to the need to mobilize metabolic resources to repair damaged tissues, as well as due to the loss of nutrients through wound fluid. Therefore, the main goal of proper nutrition in wound healing is to provide optimal calories and nutrients that will promote healing [2, 24, 30],

KEY NUTRITION CONCEPTS AND THE ROLE OF NUTRIENTS

Nutrition is considered one of the key and most controlled environmental factors that can reduce the risk of diseases throughout life. Proper and rational intake of nutrients and their metabolism provide the basis for the normal functioning of physiological processes in the human body. To analyze the nutritional needs of a diabetic foot patient, it is important to understand basic concepts and terminology. All nutrients consumed by the body are divided into macronutrients, micronutrients, and water. According to the ASPEN guidelines and standards, macronutrients are defined as «the nutrients needed in the body in the largest amounts (proteins, carbohydrates, fats)». Amino acids serve as building materials for proteins in the body. Carbohydrates can be used to form carbon scaffolds in the synthesis of amino acids; however, they can only be used for those amino acids that the body can synthesize on its own. Essential amino acids must enter the body as part of proteins or in the form of ketoacid for the synthesis of all essential amino acids. Fatty acids and cholesterol are nutrients that are broken down by beta oxidation to produce cellular energy. They play an important role in cellular function, for example, providing isolation of nerve axon membranes and are necessary for the creation of a lipid bilayer – an important component of organelles and cell membranes. Micronutrients are nutrients that the body needs in small amounts, such as vitamins and certain minerals (trace elements). Vitamins are organic nutrients that are needed in limited quantities and are not available for synthesis by the human body. The main types of vitamins are fat-soluble and water-soluble. Water-soluble vitamins require more frequent use compared to fat-soluble ones. Some minerals, such as calcium, magnesium, and phosphorus, called macronutrients, are present in the body in large quantities. One of the main functions of trace elements and some macronutrients is to provide the necessary cofactors for enzymatic reactions. Basic, or essential, nutrients are those that cannot be synthesized in the body. Conditionally essential nutrients are those that are necessary in the diet under certain metabolic conditions when the body cannot meet its needs through internal synthesis [18].

Vitamin A is an essential micronutrient present in various forms such as retinols, retinals and retinoic acid. The body assimilates vitamin A, which comes from food from animal products such as dairy products, fish, and meat, in the form of retinol or retinyl esters from ready-made retinoids or in the form of carotenoids, which are provitamin A, which are yellow, orange, and red compounds contained in plants, which are converted into retinol in enterocytes. After that, they are transported to the liver for storage using chylomicrons, and then released into the bloodstream and into biologically active tissues, binding to the protein responsible for binding retinol. Retinoids regulate important cellular processes such as cell proliferation, differentiation, and apoptosis, and therefore play a key role in many physiological processes such as immune maintenance, barrier integrity, male and female reproduction, and embryonic development. Retinoids regulate the growth and development of various types of skin cells. The lack of retinoids leads to abnormal formation of the stratum corneum of the epidermis. In case of tissue damage, vitamin A promotes the restoration of the epidermis, accelerates the healing process, and restores the structure of the skin, and has the ability to eliminate the negative effects of anti-inflammatory steroids on the healing process. In addition to its function in the inflammatory healing phase, retinoic acid promotes the production of extracellular matrix components such as type I collagen and fibronectin, stimulates the division of keratinocytes and fibroblasts, and reduces the activity of matrix-destroying metalloproteinases [15, 21].

Vitamin D is believed to have a variety of effects on the body, including an important role in the work of insulin and the differentiation of keratinocytes and fibroblasts by regulating growth factors and cytokines. Vitamin D comes from foods rich in this vitamin and is formed in the skin when exposed to sunlight. Ultraviolet rays convert 7-dehydrocholesterol in the skin into previtamin D3. Previtamin D3 can then undergo the process of isomerization into vitamin D3 under the influence of membrane structures. Vitamin D3, also known as cholecalciferol, is released from the keratinocyte cell membrane, and absorbed by the skin capillaries using a protein that binds to vitamin D [17]. Vitamin D is converted to 25-hydroxyvitamin D by the enzyme hydroxylase, which is a key non-specific regulator of the immune system. This enzyme stimulates the production of antimicrobial peptides in monocytes or macrophages. The receptor of this enzyme is found on the surface of almost all types of immune cells, including activated CD4 and CD8 T cells, B cells, neutrophils, and antigen-presenting cells such as macrophages. 25-hydroxyvitamin D reduces the level of inflammatory cytokines and promotes anti-inflammatory reactions. In patients with diabetic foot syndrome, characterized by weakened immunity, impaired production of 25-hydroxyvitamin D is considered more common and severe, especially in the presence of non-healing ulcers [22].

Vitamin E. There is a long-recognized link between oxidative stress and type 2 diabetes mellitus that hyperglycemia, hyperinsulinemia, and insulin resistance can contribute to the formation of free radicals, which contributes to the development of oxidative stress. Oxidative stress, in turn, can lead to glycation of hemoglobin, deterioration of insulin signaling and decreased secretion of insulin by beta cells in type 2 diabetes mellitus. Therefore, it is logical to assume that antioxidants such as vitamin E can have a positive effect on blood sugar control in diabetes. Vitamin E (tocopherols and tocotrienols) is a powerful fat-soluble antioxidant that the body mainly receives from fruits, vegetable oils, nuts, and green leafy vegetables. It can prevent glycosylation of hemoglobin by blocking the formation of end products of advanced glycosylation, mitigating long-term dysfunction of pancreatic β-cells caused by oxidative stress in type 2 diabetes mellitus [29].

Ascorbic acid acts as an antioxidant and influences various body tissues, including participation in the processes of reproduction, immunity, growth, and infection control. It is an important element in the production of collagen, which is necessary for the health of the skin and the formation of cartilage tissue. Vitamin C can slow down several

stages of the wound healing process, including inflammation, proliferation, and remodeling, which in turn contributes to overall healing. In the inflammatory phase, vitamin C is necessary for the breakdown of neutrophils, and in the proliferation phase it plays an important role in the synthesis, maturation, release, and decomposition of collagen, which makes up from 30 to 40% of the total amount of protein in the body. The range of collagen-related diseases is very extensive and includes various pathologies such as involuntary bleeding, cardiovascular diseases, arterial aneurysms, premature skin aging and impaired wound healing. Insufficient vitamin C levels can slow down the healing process of wounds, which increases the risk of infection and negative effects on the body. One of the limitations for humans is that they are not able to synthesize ascorbic acid on their own since this requires the presence of the enzyme gulonolactone oxidase. Therefore, to complete the inflammatory phase, it is recommended to consume vitamin C, which can be obtained from fruits, vegetables, or special additives [11, 13].

B vitamins are a set of water-soluble organic compounds that have a variety of structures but play an important role in maintaining vital body functions, such as proper cell function, growth, and tissue development. The family of B vitamins includes vitamins B1 (thiamine), B2 (riboflavin), B3 (niacin), B5 (pantothenic acid), B6 (pyridoxine), B9 (folic acid) and B12 (cobalamin). Except for vitamin B3, which can be produced from tryptophan, all other B vitamins necessary for the human body must come from food or other external sources. Consequently, a deficiency of B vitamins can occur within a few weeks with insufficient intake of a certain type of vitamin B. It is important to note that B vitamins (B1, B6 and B12) play a key role in the nervous system, both in terms of its structure and maintenance of its normal functioning [8].

Thiamine, or vitamin B1, is found in whole grains, dairy products, and red meat. This vitamin plays the role of a precursor of a biochemical coenzyme in metabolic processes and is an important component of the biological membranes of the peripheral and central nervous systems. The physiologically active form of thiamine is thiamine pyrophosphate (or sodium thiamine diphosphate), which acts as a key enzyme in carbohydrate metabolism, especially in the brain, where it plays an important role in glucose metabolism and energy production. A lack of thiamine can lead to a decrease in the rate of nerve conduction and disturbances in the electrical activity of the nervous system due to changes in the process of myelinogenesis, which ultimately leads to a decrease in the diameter of myelin fibers. Prolonged thiamine deficiency can cause distal sensorimotor polyneuropathy, especially affecting the lower extremities. Polyneuropathy due to vitamin B1 deficiency is characterized by a slow, bilateral, distal manifestation of a painful tingling pathology, also known as a burning sensation in the feet. In the absence of adequate treatment, the condition can progress to a change in gait (stepping) and the development of muscle weakness in the lower extremities [8].

Vitamin B5 is widely distributed in food and is provided by ordinary intestinal bacteria. Foods rich in pantothenic acid include animal organs (liver and kidneys), fish, shellfish, dairy products, eggs, avocados, legumes, mushrooms, and sweet potatoes. Vitamin B5 has a beneficial effect on keratinocytes and fibroblasts, promotes fibroblast migration, reproduction, and differentiation of keratinocytes, regulates the maturity of macrophages, and plays a key role in cell development. Its topical application can promote the natural healing process of wounds. Vitamin B5 regulates the immune system, stimulates epithelial cells to express inflammatory cytokines such as tumor necrosis factor and interleukin, promotes the maturation of macrophages and enhances the differentiation of T helper cells. A lack of vitamin B5 can lead to a decrease in cortisol synthesis, increased pain in arthritis and myalgia, as well as increased levels of fatigue, headache, depression, insomnia and widespread "pro-inflammatory" effects affecting the immune system. The lack of this vitamin also reduces the level of acetylcholine, a neurotransmitter of the parasympathetic nervous system, which is manifested by paresthesia and «burning in the legs syndrome» [9].

In nature, there are many foods containing ***vitamin B6,*** such as fish, beef, starchy vegetables, and fruits, and it is also produced by the intestinal microflora. One of the metabolically active forms of vitamin B6 is pyridoxal-5'-phosphate, which is formed from pyridoxine, pyridoxamine and pyridoxal, three pyrimidine derivatives. Vitamin B6 performs two different biological functions: it acts as a cofactor in many metabolic and physiological processes, as well as in developmental processes, and works as an antioxidant. Pyridoxal-5'-phosphate is a cofactor for several enzymes involved in more than 140 important enzymatic reactions, including the metabolism of glucose, amino acids and fatty acids, the synthesis of histamine, hemoglobin, and neurotransmitters, as well as in the regulation of gene expression. Vitamin B6 deficiency in adults can lead to neuropathy depending on body length, starting with the legs, and spreading to the lower extremities. Early signs of this neuropathy include a feeling of burning pain or numbness, as well as paresthesia. Neurological examination shows a decrease in sensitivity in the extremities and a weakening of deep tendon reflexes, often accompanied by ataxia and mild weakness in the extremities [8].

The term ***«Vitamin B12»*** is used to refer to cobalamines, a group of cobalt-containing corrinoids. The metabolically active forms of vitamin B12 are adenosylcobalamin, methylcobalamin and hydroxocobalamin; while cyanocobalamin contained in dietary supplements does not perform direct biological functions and requires conversion to adenosylcobalamin or methylcobalamin. Dietary sources of hydroxocobalamin present in food include animal products such as fish, beef, poultry, and dairy products. Vitamin B12 is synthesized by some bacteria, but not by plants or animals, so it is important to consume it with food to maintain proper levels and prevent deficiency. Vitamin B12 can selectively block the conduction of certain sensory nerves and reduce ectopic nerve arousal. Deficiency of B vitamins in diabetic patients can increase oxidative stress and lead to deterioration. Oxidative stress occurs when the amount of prooxidants exceeds the ability of antioxidants to compensate for their effects. Reactive oxygen species

can damage functional compounds and tissues by altering carbohydrates, proteins, lipids, and DNA. A lack of vitamin B12 can lead to increased levels of homocysteine, which is thought to contribute to the accumulation of reactive oxygen species. Vitamin B12 deficiency is associated with an increase in prooxidants and a decrease in antioxidants [7].

Magnesium plays a key role as a cofactor in the transport of glucose into cells and carbohydrate metabolism and is also involved in many enzymatic processes. It is necessary for the activation of insulin, the synthesis of collagen and proteins. Insufficient magnesium intake increases the risk of developing diabetes. Magnesium deficiency reduces the protective functions of cells from oxidative stress, which reduces resistance to oxidative stress caused by diabetes and contributes to the progression of complications associated with this disease [6, 13].

The concentration of ***calcium*** in the wound area varies according to the biochemical activity of the healing process. At the stage of hemostasis, calcium promotes blood clotting, contributing to the formation of a platelet plug. It is believed that in the inflammatory phase, a high content of extracellular calcium penetrates neutrophils and causes an increase in intracellular calcium, which ultimately modulates the function of neutrophils. One of the signs of the proliferation phase is the overgrowth of the wound with a new epithelium. Extracellular calcium plays an important role in regulating epidermal homeostasis, and its receptor (CaSR) initiates calcium signals that promote adhesion, differentiation, and survival of keratinocytes by increasing intracellular calcium and transmitting signals associated with E-cadherin. The rapid diffusion of calcium ions at the wound site causes a damage signal that does not depend on transcription and stimulates the healing process of the epithelium [27].

Zinc plays a key role in various processes of cellular metabolism and promotes wound healing. It can enhance platelet activity and aggregation and performs an important function in the adhesion of monocytes to the endothelium. Zinc can regulate the differentiation of monocytes into various types of macrophages: proinflammatory (M1 macrophages responsible for initial inflammation and elimination of microbes/residues) or immunoregulatory/wound healing processes (M2 macrophages involved in suppressing the immune response and subsequent tissue repair/remodeling). Macrophages use zinc transporters to transport it to or from bacterial phagosomes. Depending on the presence of microbes, macrophages can deprive bacteria of zinc, effectively starving them, or, conversely, harm bacteria with toxic levels of zinc and other heavy metals. Zinc metalloproteinases are involved in DNA and RNA synthesis, protein, and collagen production, and support immune function, cell division, and wound healing processes. In addition, zinc is an important cofactor for lysyl oxidase enzymes, which play a key role in the formation of collagen cross-links [13, 16].

Iron plays an important role in the process of collagen synthesis, promotes wound healing and is necessary for the hydroxylation of proline and lysine. Patients with diabetic foot syndrome often have problems with anemia and iron deficiency. Lactoferrin, a glycoprotein that binds to iron and is secreted by iron-containing epithelial cells, promotes the healing process, having a beneficial effect on the initial phase of inflammation. It can reduce excessive immune responses, contributing to the development of granulation tissue and the process of epithelialization. Lactoferrin also promotes the movement of fibroblasts and keratinocytes by stimulating collagen synthesis.

Copper is necessary as a cofactor for protein synthesis and plays an important role in the formation of collagen during wound healing, therefore, copper deficiency can slow down the wound healing process [13].

Selenium plays an important role as an immunonutrient, supporting human metabolic activity through its chemical bonds. Organic forms of selenium that are naturally present in the human body include selenocysteine and selenoproteins. These forms have a special mechanism of synthesis and translational coding. Selenoproteins act as antioxidants, help regulate thyroid function, increase male fertility, and have an anti-inflammatory effect. One of the main selenoproteins present in the human body is glutathione peroxidase, which helps control the excessive formation of free radicals in inflammatory foci. In addition to glutathione peroxidase, other selenoproteins include selenoprotein-S, which regulates inflammatory cytokines, and selenoprotein-P, which plays a role in maintaining homeostasis. During wound healing, selenoproteins such as GPX-1, GPX-4, selenoprotein S and selenoprotein P interact to perform various functions, including antioxidant activity, suppression of inflammatory cytokines and neutralization of peroxynitrate during the inflammatory process [10].

DISCUSSION

Most individual, both primary and secondary studies provide insight and evidence of differences in macro- and microelements in patients with diabetic foot. Thus, a study by the authors Pena et al., 2020 showed that the prevalence of micronutrient deficiencies, especially vitamins D, C, A and zinc, is especially high in patients with diabetes mellitus with foot ulcers [20]. A review by the authors of Putz et al., 2022 indicated that insufficient vitamin D content may play a key role in the development of peripheral neuropathy, diabetic foot syndrome, as well as cardiovascular autonomic neuropathy in patients with type 2 diabetes mellitus [23]. A systematic review and meta-analysis by Kurian et al., 2023 identified a significant relationship between diabetic foot ulcers and levels of vitamin D, vitamin C, as well as magnesium, copper, and selenium. Based on the data obtained, the authors also make recommendations to determine the level of micronutrient status in patients with diabetic foot syndrome and to investigate their relationship [14]. In their study, Dai et al., 2019 conducted a systematic review and meta-analysis to assess the relationship between vitamin D deficiency and foot ulcers in patients with diabetes mellitus. The analysis included data from seven studies with a total of 1,115 participating patients. It was found that the level of vitamin D in foot ulcers in patients with diabetes mellitus was significantly reduced (mean difference – 13.47 nmol/l, 95% CI – 16.84-10.10; P = 0.34, I2 = 12%). It was also found that severe vitamin D

deficiency significantly increases the risk of developing foot ulcers in patients with diabetes mellitus (Odds ratio 3.22, 95% CI 2.42-4.28; P = 0.64, I2 = 0%) [4]. In another promising study, the authors evaluated the relationship between vitamin D and diabetic foot in Chinese patients. The total number of hospitalized patients was 1,721, who were divided into two groups depending on the presence of a diabetic foot. According to the results, the level of 25-OH-vitamin D in patients with Wagner scores from 0 to 5 showed a downward trend (p=0.114) and was also associated with diabetic foot independently (p=0.001, OR=0.986). Vitamin D deficiency and the frequency of its deficiency were higher in the group of patients with diabetic foot (77.51%) compared with the group without it (59.2%). The level of 25-OH-vitamin D was also lower in the group with diabetic foot (35.80 nmol/l) compared with the group without it (45.48 nmol/l). The researchers concluded that despite seasonal changes in vitamin D levels, patients with diabetic foot syndrome always face an increased risk of vitamin D deficiency [28]. An updated systematic review and meta-analysis of randomized controlled trials conducted by researchers Asbaghi et al., in 2023, revealed that vitamin E intake significantly reduces levels of glycosylated hemoglobin, fasting insulin and insulin resistance index in patients with type 2 diabetes mellitus. In addition, there was a significant decrease in fasting blood glucose levels when taking vitamin E in studies with varying duration of intervention. The researchers also found that the optimal doses of vitamin E to regulate the level of glycosylated hemoglobin and insulin are in the range of 400 to 700 mg per day [1]. In a prospective double-blind placebo-controlled study among 90 patients with type 2 diabetes mellitus, it was found that treatment with oral methylcobalamin at a dose of 1 mg for twelve months led to an increase in plasma vitamin B12 levels. In addition, there was an improvement in all neurophysiological parameters, somatosensory function, pain level, as well as an improvement in the quality of life of patients. These results highlight the importance of maintaining optimal nutritional status in people with chronic diseases [5]. A study by Turkish authors examined the relationship between serum magnesium levels and diabetic foot ulcers in a sample of 147 people. The participants were divided into three groups: a main group of 49 patients with diabetes mellitus and foot ulcers, a control group of patients with diabetes mellitus without foot ulcers, and a control group of 49 healthy individuals, the groups were similar in age and gender (p= 0.116 and 0.897, respectively). According to the results, magnesium levels in patients with diabetes mellitus and foot ulcers were lower than in patients with diabetes mellitus without ulcers and in healthy individuals (p<0.001). It was found that there is a strong relationship between the magnesium content in the blood and the incidence of ulcers on the diabetic foot (OR 5.9, CI 95% 2.7–12.6, p < 0.05) [12].

CONCLUSIONS

According to the results of the presented data, it is worth noting that the state of vitamins, macro- and microelements plays an important role, because monitoring the micronutrient status and proper care make it possible to identify deficiencies, take the necessary measures to restore them, optimize the wound healing process, strengthen the immune system and improve the patient's body. Using this strategy, in turn, will lead to significant improvements, more effective rehabilitation and prevention of complications. Since improper nutrition negatively affects the complex process of wound healing, it is important to screen patients with wounds and identify risk factors for nutritional deficiency in advance. And the introduction of clinical guidelines based on the results of foreign and domestic research will provide medical personnel with systematic information about the state of trace elements and macronutrients in the patient's body and the levels of eating disorders, which will allow developing a new approach to caring for patients with diabetic foot syndrome in clinical practice.

Authors' contribution:

Sh. Batarbekova, D. Zhunussova, Zh. Bekbergenova – data collection and analysis, writing, editing.

G. Derbissalina, N. Maksimova – conceptualization, editing.

Conflict of interest:

The authors claim that there is no conflict of interest.

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Received 02.05.2024
Sent for revision 24.06.2024
Accepted 28.02.2025
Published online 30.06.2025

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СОВРЕМЕННЫЙ ПОДХОД К ЛЕЧЕНИЮ САХАРНОГО ДИАБЕТА 2 ТИПА, ОСЛОЖНЕННОГО СИНДРОМОМ ДИАБЕТИЧЕСКОЙ СТОПЫ

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Гипергликемия, характерная для сахарного диабета, способствует производству выработки агентов окислительного стресса, которые вызывают нарушение иммунной функции и развитие хронического воспалительного процесса. Эти факторы лежат в основе развития поздних осложнений сахарного диабета, таких как ишемическая болезнь сердца, нестабильная стенокардия, инфаркт миокарда, периферическая микроангиопатия и другие сочлененные заболевания, которые ухудшают прогноз при синдроме диабетической стопы. Воспалительные процессы и нарушения деятельности иммунной системы способствуют дисбалансу тканевого гомеостаза, ассоциируются с появлением хронических язв и потребностью в ампутации нижних конечностей. Последствия хронических ран создают неблагоприятную реальность инвалидности и душевного стресса для пациентов. Как пациенты, так и медицинский персонал, несомненно, получат выгоду от более глубокого понимания патогенеза и патофизиологии различных незаживающих язв. Новые знания приобретены путем сравнения сходства между хроническими ранами различной природы с их различиями от острых ран.

Ключевые слова: сахарный диабет 2 типа; синдром диабетической стопы; витамины; микронутриенты; макронутриенты; ведение

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ДИАБЕТТІК ТАБАН СИНДРОМЫМЕН АСҚЫНҒАН 2 ТИПТІ ҚАНТ ДИАБЕТІН БАСҚАРУДАҒЫ ЗАМАНАУИ ТӘСІЛ

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Қант диабетіне тән гипергликемия иммундық функцияның бұзылуына және созылмалы қабыну процесінің дамуына әкелетін тотығу стрессінің агенттерін өндіруге ықпал етеді. Бұл факторлар жүректің ишемиялық ауруы, тұрақсыз стенокардия, миокард инфарктісі, перифериялық микроангиопатия және диабеттік табан синдромының болжамын нашарлататын басқа да артикуляциялық аурулар сияқты қант диабетінің кеш асқынуларының дамуына негіз болады. Қабыну процестері және иммундық жүйенің бұзылуы тіндік гомеостаздың теңгерімсіздігіне ықпал етеді, созылмалы жаралардың пайда болуымен және төменгі аяқтарды ампутациялау қажеттілігімен байланысты. Созылмалы жаралардың салдары пациенттер үшін мүгедектік пен психикалық стресстің қолайсыз шындығын тудырады. Науқастар да, медициналық қызметкерлер де әр түрлі емделмейтін жаралардың патогенезі мен патофизиологиясын тереңірек түсінуден пайда көретіні сөзсіз. Жаңа білім әртүрлі сипаттағы созылмалы жаралар арасындағы ұқсастықтарды олардың өткір жаралардан айырмашылықтарымен салыстыру арқылы алынады.

Кілт сөздер: 2 типті қант диабеті; диабеттік табан синдромы; витаминдер; микронутриенттер; макронутриенттер; басқару