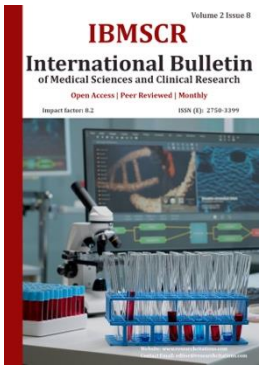

PATHOGENESIS OF PURULENT-NECROTIC COMPLICATIONS IN DIABETIC HEEL SYNDROME.

Hamdamov Baktiyar Zaripovich
doctor of Medical Sciences, prof.
Rakhimov Abdurasul Sharifovich
Bukhara State Medical Institute, Uzbekistan
https://doi.org/10.5281/zenodo.10947206

Abstract. This article covers the prevalence of diabetes mellitus, the epidemic scale of diabetic heel syndrome, a significant increase in the number of patients, an increase in the life expectancy of patients with diabetes due to the creation of effective means and methods of treatment, an increase in the number of late complications of the disease, problems of patient disability, optimal methods of treatment.

Keywords. Kandli diabetes, diabetic retinopathy, polyneuroangiopathy, nephropathy, hyperglycemia, atherosclerosis.

Late complications of diabetes mellitus (diabetic retinopathy, polyneuroangiopathy, nephropathy) are the greatest risk to the world community, causing severe social and economic consequences[3].

The increase in the number of heel diabetic lesions is due to an increase in diabetes mellitus and an increase in the life expectancy of patients against the background of monand treatment, an increase in the length of the course of the disease, a combination of risk factors such as arterial hypertension, hypercholesterolemia, obesity, tobacco smoking, microalbuminuria, delay in diagnosis at all stages of their.

Diabetic heel syndrome was considered the most common and life-threatening complication of diabetes, in which statistics on the frequency of injury to the legs and its conclusion are subsequently negative due to the development of the pus process in the heel of 30-80% of patients. One of the methods of optimizing the provision of medical care for patients is the development and use of a protocol for carrying patients with Dox purulent-necrotic complications. Early detection of risk factors for wound development in the heel, their assessment and Prevention, active treatment tactics and a multi-disciplinary approach make it possible to improve the condition of patients and prevent amputation in most cases[1,3,8].

The diversity of clinical signs of diabetic heel syndrome is reflected in most classifications, as evidenced by the fact that there is no single system of registration, as well as the need to develop a monand classification.

Diabetic heel syndrome the pathogenesis of purulent-necrotic complications is multifactorial in nature. Hyperglycemia disorders are the main link in blood supply and tissue innervation in the legs leading to systemic changes in macro - and microangiopathy, neuropathy, osteoarthropathy formation, changes in rheological properties of the blood, immunological disorders, the addition of infection [2,6,10].

The importance of metabolic disorders in the pathogenesis of complications of diabetic heel syndrome is due to the persistence of hyperglycemia, which leads to nonferment

glycosylation with changes in protein structure as well as tissue accumulation, lipid metabolism disorders and activation of lipid peroxide oxidation.

Diabetic angiopathy concentrates on changes occurring in vessels in QD, and is conditionally defined as macroangiopathy when arteries are damaged depending on location, and microangiopathy - when microcirculatory flow is damaged. Diabetes leads to the rapid development of atherosclerosis (Saltikov B.B., 2021). Changes in fibrosis rashes lead to vascular occlusions and a clinical pace of ischemia occurs. Modified lipoproteins involved in endothelial cell damage create conditions for the development of angiopathy. Diabetic angiopathy differs clinically from obliterating vascular atherosclerosis in the legs by slower development in adulthood, lack of multisegmentarity, symmetriality of the lesion. It is characteristic that there is no sequential chromotoma in diabetic heel syndrome due to the combination of Angio - and neuropathy. Compared to data from patients with obliterating atherosclerosis in their diabetic feet, specialized pathomorphological signs of atherosclerotic injury in individuals with diabetes were not identified[4,6].

Hyperglycemia triggers the development of changes in the vessel wall by polyol activation of glucose metabolism, glucose nonferment autooxidizing metabolism, glycosylation of various proteins, which leads to the development of low-density lipoproteid separation in the vessel wall as well as tissue hypoxia. Increased lipid peroxidation processes, changes in lipoproteid quality characteristics are the basis of atherosclerotic lesions in large vessels. The decrease in nitric oxide formation with endothelium as well as the development of hypoxia cause oxidative stress, which indicates a stagnation disorder between the prooxidants and the antioxidant protection system, and is directly related to the expression of macroangiopathic complications of diabetes mellitus. Strict control of glycemia makes it possible to reduce the number of complications of diabetes in the vein as well as the number of wound injuries in diabetic heel syndrome[3,8].

Damage to large vessels is associated with the accumulation of laminin, type IV collagen, fibronectin and hyaluronic acid in their wall. The middle shell of the arteries is calcinosis, a characteristic alteration of the menkeberg arteriosclerosis, which reduces elasticity but does not lead to loss of pulsation and narrowing of the esophagus.

More typical for Type 2 diabetes is diabetic macroangiopathy (Trusov V. et al., 2017) can be considered a particular cause of the formation of changes in mild diabetic heel syndrome that lead to the formation of wounds and necrosis, which increases the risk of injury-free amputations by up to 22 times and is the main cause of patients being disabled and dying from vascular complications[5].

The pathogenetic basis of polyorgan pathologies in patients with diabetes is the systemic nature of angiopathic disorders. In 90% of patients, changes in vascular diameter, increased pressure in the capillaries, microaneurysms, intravascular aggregation of erythrocytes have been found. Similarity in the morphological pace of microcirculatory flow vessel damage in Type 1 and type 2 diabetes mellitus, the dependence of changes on the duration of the course of the disease suggests that the mechanisms of development of diabetic angiopathy are in harmony with damage to the structure of the vessel wall, increased vascular permeability and plasmatic saturation. Long-existing Vasa vasorum microangiopathy causes hypoxia as well as major vascular trophic disruption, creating conditions for macroangiopathy formation[7,9].

In diabetes mellitus, endothelial dysfunction develops at different stages of the disease, since endothelium is the main target of the influence of various endogenous factors: hyperglycemia, oxidative stress, polyol pathway of glucose metabolism, C proteinkinase, end products of glycylation. In addition, the specific receptors of endotheliocytes are influenced by biologically active substances that are developed by platelets, leukocytes, basic cells or activated in blood plasma, which leads to vasodilation or vasoconstriction. The main role of endothelium is to support vascular function by means of the synthesis of vasoactive substations of endothelin and nitric oxide (no), which control the tone and growth, thrombogenicity and atherogenicity of the vessel wall, proliferation of smooth muscle elements[3,5,8].

With an increase in age, the ability of the endothelium to develop vasodilators decreases, its reaction to the action of humoral vasoconstrictors increases (Hozyainova N.Yu. et al., 2015), which is aggravated by metabolic disorders in diabetes mellitus. Nevertheless, the leading pathogenetic factors that damage the structure-functional organization of endotheliocytes at the initial stage of the formation of angiopathy, the problem of endothelial dysfunction in diabetes mellitus is not sufficiently studied and requires further research.

In hyperglycemia (an endothelial relaxation factor that calls for endothelium-dependent vasodilation), the main mediator that calls for large-to medium-caliber arterial dilation is nitric oxide. It has a deaggregation and antiaterogenic effect, with adgesive molecules calling for a blockade of smooth muscle proliferation and expression. The synthesis of NO, prostaglandins and endothelial hyperpolarizing factor (EDHF) is carried out in the endothelium. EDHF is formed mainly in the vessels of the restrictive type, in much smaller quantities - in large arteries.

In arteries, mostly NO is a vasodilator, in arterioles less than 100 μm in diameter - EHDF, in arterioles more than 100 μm, NO and EDHF are equally important. Insulin triggers the synthesis of no with endothelium, so the ability of the vessel wall to vasodilation is reduced in the case it lacks[4,6].

The decrease in the formation of no vascular tone leads to a lack of monand regulation of the ankle creates conditions for the development of a wound defect. In addition, a decrease in the formation of no with macrophages is observed with a decrease in intracellular killing of bacteria, which leads to excitatory persistence in the tissues of the ankle. Violation of the formation of NO in the nerve fiber calls for dysfunction as well as subsequent Vasa nervorum occlusion. In neuropathic wounds, over - synthesis of NO associated with high expression of non-synthase indusibel form leads to disruption of trophic wound healing process[2,6].

The increase in the level of cytokines is associated with leukocyte adgesia, it grows in the natiage of increased expression of adgesive molecules in endotheliocytes, leukocytes, platelets and leads to microcirculation disorders as early as the initial stages of diabetes mellitus. In endothelial dysfunction against the background of diabetes mellitus, the level of aggregation as well as the Villebrand factor, which induces platelet adgesia, increases significantly, which leads to a violation of blood rheology[6,7].

References:

1. Аметов А.С. Сахарный диабет 2 Типа: проблемы и решения. — М.: ГЭОТАР-Медиа, 2012.

2. Майоров А.Ю., Суркова Е.В. Сахарный диабет1 Типа. — М.: Перспектива, 2007.
3. Бреговский В.Г., Зайцев А.А. Поражение нижних конечностей при сахарном диабете. — М., 2004.
4. Дедов И.И., Удовиченко О.В., Галстян Г.Р. Диабетическая стопа // Практическая медицина. — М., 2005.
5. Славин Л.Е., Годжаев Б.Н., Анчикова Л.И. Синдром диабетической стопы // Практическая медицина. — 2008. -—№ 27. — С. 54-57.
6. Wagner FW. A classification and treatment program for diabetic, neuropatic and dysvascular foot problems. // In The American Academy of Ortopaedic Surgeons instructional course lectures. — St. Louis. — Mosby Year Book. — 1979. — P. 143-165.
7. Оболенский В.Н., Семенова Т.В., Леваль П.Ш., Плотникова А.А. Синдром диабетической стопы в клинической практике // Русский медицинский журнал. — 2010. — № 2. — С. 45.
8. Armstrong DG, Lavery LA, Harkless LB: Validation of a diabetic wound classification system. Diabetes Care 21: 855-859,1998.
9. Храмилин В.Н., Демидова И.Ю. Синдром диабетической стопы: Методическое пособие для самоподготовки слушателей циклов усовершенствования врачей. — М., 2004. — 54 с.
10. Российская ассоциация эндокринологов, Московская ассоциация хирургов. Синдром диабетической стопы: клинические рекомендации. — М., 2015. — 46 с.
11. Sharipova Gulnihol Idiyevna. THE EFFECTIVENESS OF THE USE OF MAGNETIC-INFRARED-LASER THERAPY IN TRAUMATIC INJURIES OF ORAL TISSUES IN PRESCHOOL CHILDREN//Academic leadership. ISSN 1533-7812 Vol:21Issue 1
12. Sharipova G. I. The use of flavonoid based medications in the treatment of inflammatory diseases in oral mucus //Asian journal of Pharmaceutical and biological research. India. – 2022. – Т. 11. – №. 1. – С. 2231-2218. (Impact factor: 4.465)
13. Sharipova G. I.Changes in the content of trace elements in the saliva of patients in the treatment of patients with traumatic stomatitis with flavonoid-based drugs // Journal of research in health science. Iran. – 2022. – Т. 6. – № 1-2. – С. 23-26. (Scopus)
14. Sharipova G. I. Paediatric Lazer Dentistry //International Journal of Culture and Modernity. Spain. – 2022. – Т. 12. – С. 33-37.
15. Sharipova G. I. The effectiveness of the use of magnetic-infrared-laser therapy in traumatic injuries of oral tissues in preschool children //Journal of Academic Leadership. India. – 2022. – Т. 21. – №. 1.
16. Sharipova G. I. Discussion of results of personal studies in the use of mil therapy in the treatment of trauma to the oral mucosa //European journal of molecular medicine. Germany. – 2022. – Т. 2. – №. 2. – С. 17-21.
17. Sharipova G. I. Peculiarities of the morphological structure of the oral mucosa in young children // International journal of conference series on education and social sciences. (Online) May. Turkey. – 2022. – С. 36-37.
18. qizi Zarifova, D. O. (2023). RANGLARNING FAZOVIY BIRIKMALARI VA XUSUSIYATLARI. Educational Research in Universal Sciences, 2(5), 159-162.
19. Sharipova G.I. Assessment of comprehensive dental treatment and prevention of dental diseases in children with traumatic stomatitis // National research in Uzbekistan: periodical conferences: Part 18. Tashkent. -2021. - S. 14-15.

20. Sharipova G. I. Dynamics of cytological changes in the state of periodontal tissue under the influence of dental treatment prophylactic complex in young children with traumatic stomatitis // Multidiscipline Proceedings of digital fashion conference April. Korea. – 2022. – C. 103-105.