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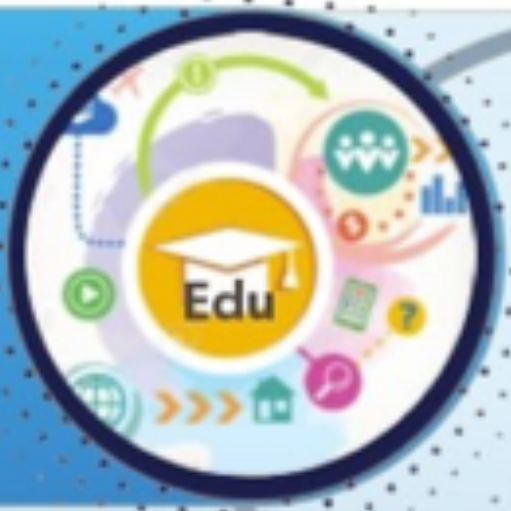

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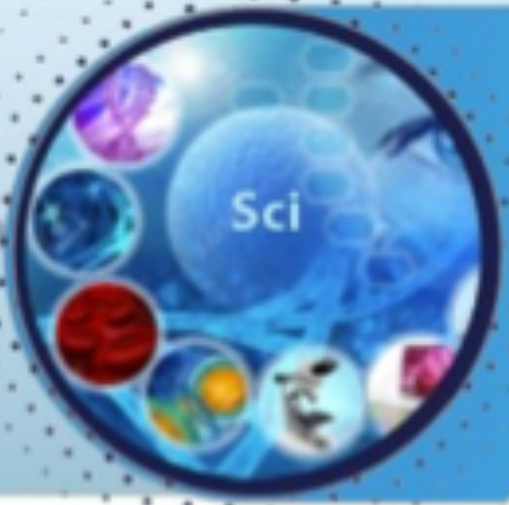

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Review Article

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Prevalence and Risk Factors for Diabetic Foot Syndrome

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ABSTRACT

The prevalence of diabetic foot syndrome among diabetic patients averages 4-10%. Even though the prevalence of diabetes mellitus in different countries ranges from 1.5 to 6%, patients with diabetic foot syndrome account for 40-60% of all non-traumatic lower limb amputations. Diabetic foot as an independent complication of diabetes mellitus, along with diabetic lesions of the eyes, kidneys, cardiovascular and nervous systems, was identified in the Report of the WHO Research Group in Geneva, in 1987.

This review article presents literature data for the last 10 years on the prevalence of diabetic foot syndrome as the main cause of disability and mortality among patients with diabetes mellitus.

Keywords: Diabetes mellitus, diabetic foot syndrome, prevalence, etiology

According to the results of epidemiological studies in various countries, lower limb amputations in patients with diabetic foot syndrome account for 50-70% of the total number of amputations not caused by trauma [1].

In European countries, the number of annual amputations for vascular diseases and diabetes mellitus varies from 20 to 32 per 100,000 population [2].

Elderly and senile patients have a high rate of postoperative complications and mortality (40-60%), as well as the frequency of reamputation (34-57%) [3].

In different countries, mortality during the first year after amputation ranges from 11 to 41%, and the total frequency of ipsilateral and contralateral reamputations in the next 5 years ranges from 26 to 51% [4].

Interventions at the hip level prevail (78.7%). The mortality rate for hip amputation is 16.3%. The main causes of death were acute cardiovascular failure (66.4%), progressive hepatic and renal failure (24.3%), myocardial infarction (5.7%) and stroke (3.6%).

Epidemiological studies have shown that amputation of the other limb after the first high amputation is performed in the next five years in 28-51% of patients with diabetes mellitus [5].

In arterial diseases and diabetes mellitus, amputations are performed mainly at the hip level [6]. Under the conditions of the existing system of medicine, there is a situation in which it is economically profitable for surgical hospitals to perform amputation at a deliberately higher

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level to achieve speedy healing of the postoperative wound and discharge of the patient. After a high amputation, about 50% of patients lose the ability to move [7].

Limb amputation is an operation aimed at saving the patient's life, which subsequently makes significant adjustments to the lifestyle of the patient and his family. Amputation is accompanied by a severe physical and psychological impact on the patient [8]. That is why reducing the frequency of amputations is one of the most important tasks of WHO, as proclaimed by the St. Vincent Declaration of 1989. However, the goal of halving the number of high amputations in 5 years has not been achieved, and the available data on the frequency of amputations in different countries are not encouraging.

Data from national diabetes registries show a 45% increase in the total number of amputations in England between 1990 and 1994. A similar trend can be observed in the United States, where the amputation rate increased from 36,000 (1980) to 54,000 (1990) and amounted to 86,000 cases in 1996 [9].

According to some estimates, the number of amputations in the world in patients with diabetes mellitus on a global scale is 55 amputations per hour [10].

In addition to the unfavourable prognosis for life, amputation is expensive for health care, so if the cost of primary healing of an ulcer ranges from 7-17 thousand US dollars, then the costs associated with the amputation of the lower limb reach 30-60 thousand dollars [11].

It is important to note that 85% of amputations can be prevented with early detection of diabetic foot syndrome and simple preventive measures to prevent the formation of ulcers, as well as with proper treatment of the ulcer that has already occurred [12].

Epidemiological studies of recent years leave no doubt about the need to develop measures to prevent and reduce the frequency of amputations in patients with diabetes mellitus and to study new methods of local treatment of ulcerative defects.

Over the past three decades, a great deal of clinical and scientific experience has been accumulated, which has made it possible to shed new light on the pathophysiological mechanisms of the formation of diabetic foot syndrome, to carry out pathogenetically based treatment and prevention. A study conducted by a European author showed that in the presence of diabetic polyneuropathy, even without impaired blood flow and other risk factors for diabetic foot syndrome, the likelihood of amputation increases by 1.7 times. In the presence of foot deformity, this risk increases 12 times, and in the presence of ulcerative foot defects in the anamnesis, it increases 36 times.

Risk factors for diabetic foot syndrome, according to leading scientists (from selected analytical and experimental studies), are presented as follows:

- Peripheral sensorimotor and autonomic neuropathy;
- Chronic arterial insufficiency of the lower extremities;
- Presence of high-pressure zones on foot;
- Trauma;
- Limitation of joint mobility;
- Poor control and duration of diabetes;
- Previous ulcers and/or amputations;
- Other Complications of Diabetes;
- Improperly fitted shoes;
- Socio-psychological factors;
- Orthopedic problems;
- Race.

In most cases, the risk factors associated with developing ulcer defects are similar to those that lead to ulcer recurrence. Using history and clinical examination, the following risk factors for ulcer recurrence can be identified:

1. History of ulcers/amputations
2. Loneliness
3. Impaired protective sensitivity
4. Impaired vibration sensitivity
5. Lack of Achilles reflexes
6. Hyperkeratosis
7. Foot deformities
8. Poorly fitting shoes
9. No pulsation on the arteries of the feet.

In 1987, the WHO Diabetes Research Group pointed out that "damage to nerve fibers that conduct sensory excitation and innervate muscles, blood vessels, and internal organs is the most common complication of diabetes." The incidence of diabetic neuropathy ranges from 0% to 93% [13]. This indicator depends on the duration of the disease, the age of the patients and concomitant microangiopathy. Using electrophysiological research methods, peripheral nerve dysfunction is detected in almost 100% of patients, and therefore many authors suggest that diabetic neuropathy should be considered a manifestation of this disease rather than a late complication of diabetes mellitus [14].

The most common cause of neuropathy followed by ulceration is diabetic foot syndrome [15]. In a prospective study, it was shown that within 3 years, patients with established neuropathy, confirmed by an increased threshold of vibration sensitivity, developed ulcer defects 7 times more often [16].

The presence of certain signs of neuropathy ranges from 8% in newly diagnosed patients to 50% with a disease duration of more than 25 years [17], significantly affecting the quality of life, ability to work, and social activity of patients [18].

Sensorimotor disorders result in a decrease and subsequently a loss of all types of sensation and a characteristic deformity of the foot.

According to many authors, a foot devoid of defense mechanisms (lack of response to injury) has a high risk of developing ulcers. Patients with peripheral neuropathy are 7 times more likely to develop ulcers than patients without neuropathy [19].

Motor neuropathy leads to muscle weakness, atrophy, and paresis of the foot muscles. The structure of the foot is deformed due to the instability of the metatarsophalangeal joints. Loss of function of the small internal muscles of the foot and the anterolateral muscle group of the lower extremities leads to the predominance of the influence of long flexors of the fingers, plantar flexors and ankle extensors. "Intrinsik minus" of the foot is a classic deformity with an increase in transverse size, transverse and longitudinal flat feet, deformity of the ankle joints, hammer or claw-like curvature of the toes, hypertrophy of the heads of the metatarsal bones with the formation of corns [20].

Sensory neuropathy leads to the loss of pain, tactile, temperature, vibration, and proprioceptive types of sensitivity. In this way, a second minor injury is not felt by the patient and the skin lesions go unnoticed for some time. Of great importance is the loss of proprioception - the sense of the position of the feet in space and relative to each other. According to a number of authors, it is due to the presence of sensorimotor disorders that an abnormal distribution of zones of increased pressure on the foot is formed. Maximum plantar pressure values in patients with diabetes mellitus are higher than in the general population or in patients with diabetes mellitus without signs of neuropathy [21].

The decrease in vasoconstrictor and the prevalence of vasodilating influences leads to the opening of arteriovenous shunts, leading to an increase in skin blood flow several times compared to normal [23]. Autonomic nerve neuropathy leads to an autosympathectomy condition that causes a loss of vasomotor tone [24]. As a result, the pressure on the venous capillaries increases and neuropathic edema occurs [25]. This may explain the clinical manifestations of the neuropathic form of diabetic foot syndrome – warm skin of the feet, a well-palpable pulse on the foot, and the expression of veins. In addition, sympathetic denervation of arteries and arterioles is accompanied by degeneration of the muscle layer with its subsequent calcification, which was previously regarded as a manifestation of atherosclerosis.

According to almost all researchers, the pathology of large vessels is one of the leading risk factors for diabetic foot syndrome. Atherosclerotic lesions (macroangiopathies) in patients with diabetes mellitus develop 10 years earlier, progress faster and are much more severe [26]. Atherosclerotic lesions occur in the popliteal region, tibia, and in the arteries of the foot. Pathogenetically, the atherosclerotic process of the tibial arteries does not differ from that in people who do not suffer from diabetes mellitus.

There are several classifications of vascular lesions of the extremities. The classification of ischemic lesions of the feet according to Fontaine-Pokrovsky allows us to distinguish 5 stages of the lesion, which is more based on the subjective sensation of pain:

Stage I – pain in the limb occurs after a long walk (about 1 km);

Stage IIa – a distance without pain walking (average step at a speed of about 3 km/h) of more than 200 m;

Stage IIb – the patient walks less than 200 m;

Stage III a – "pain at rest", i.e. in a horizontal position, which forces the patient to periodically lower the leg down (up to 3-4 times a night);

Stage III b (critical ischemia) – swelling of the lower leg and foot;

Stage IV a – (critical ischemia) – necrosis in the toes;

Stage IVb – gangrene of the foot and lower leg (high amputation required).

Vascular problems in patients with diabetes mellitus can manifest themselves in different forms and often due to the lack of complaints and signs, the initial stages may remain undiagnosed.

It should be noted that asymptomatic chronic arterial insufficiency of the lower extremities is a serious problem in the majority of DM patients. In this situation, the diagnosis can be made only on the basis of the results of an instrumental examination.

A special form of vascular lesion is a state of critical limb ischemia. It occurs in diabetes mellitus 5 times more often than in patients without diabetes mellitus. Critical limb ischemia, both in patients with and without diabetes mellitus, is a combination of the following signs.

1. Persistent pain in the limb at rest, requiring the use of analgesics, lasting more than 2 weeks, with systolic

blood pressure in the ankle area less than 50 mmHg and/or BP on the big toe ≤ 30 mmHg;

2. Ulcer or gangrene in the area of the foot, toes in combination with objective findings.

The rationale for distinguishing this group of patients is that without timely restoration of blood flow, ischemia will almost inevitably lead to extensive tissue necrosis and amputation [27].

According to the 1997 definition of critical limb ischemia [28], this diagnosis is made in the presence of one of the following manifestations:

1. Ischemic pain at rest in combination with blood pressure on the tibial artery in the supine position < 40 mm Hg, on the artery of the big toe < 30 mm Hg, absence of pulse wave or its smoothness on ultrasound examination.

2. Limited tissue necrosis - ulcer or local gangrene on the background of diffuse ischemia of the foot in combination with blood pressure on the tibial artery in the supine position < 60 mmHg. On the artery of the big toe <40 mmHg. Absence of pulse wave or its smoothness on ultrasound examination.

3. Tissue necrosis extending to the metatarsal area. This makes it impossible to preserve the foot, in combination with the arterial pressure on the tibial artery in the supine position < 60 mmHg, on the artery< big toe < 40 mmHg. The absence of a pulse wave or its smoothness on ultrasound examination.

Macroangiopathy is atherosclerosis that has a number of features in patients with diabetes mellitus, according to the authors [29]:

- more distal lesion (more often popliteal artery and lower leg arteries),
- bilateral and multiple localization of stenosis,
- development of the process at a younger age,
- comparable incidence of men and women, etc.

Diabetes mellitus is a powerful factor that stimulates the development of atherosclerosis [30].

Diabetic macroangiopathy (as well as peripheral atherosclerosis in patients without diabetes mellitus) can cause necrosis of the skin and subcutaneous tissues without any additional mechanical damage due to a sharply disrupted supply of oxygen and nutrients to the tissues. However, in some patients, it is possible to identify some factors (skin cut during nail treatment, mycotic lesion in the interdigital spaces, etc.) that violate the integrity of the skin. A significant decrease in blood flow, the reparative ability of the skin and leads to the expansion of the necrosis zone.

The presence of macroangiopathy did not depend on the age and duration of diabetes. Smoking has a significant effect on atherosclerotic vascular damage: it occurs in 15.8% of smokers and in 7.3% of non-smokers [31].

According to Greek scientists, macroangiopathy in patients with diabetes mellitus is genetically dependent on the duration of diabetes, abdominal type of fat deposition and the level of triglycerides in the blood, while smoking, the type of glucose-lowering therapy, blood lipids, and the level of HbA1c, do not have a significant impact on the development of macroangiopathy [32].

Patients with chronic arterial insufficiency of the lower extremities are usually bothered by pain in the calf muscles that appear during exertion (walking) and disappear after a few minutes of rest – intermittent claudication syndrome. It is based on insufficient blood supply to the muscles by obstructively altered NK arteries, as a result of which lactic acid accumulates in the muscle mass [33]. With an advanced pathological process, there is pain at rest, especially at night, and ulcers can develop.

Digital pulsation measurement on the 4 ankle arteries (anterior and posterior tibial) is recommended for screening for major artery involvement. If pulsation in these arteries cannot be checked, the saphenous and femoral arteries should be examined. The dorsal artery of the foot may be absent at birth. In the presence of pulsation on the above vessels, the presence of a serious pathology can be ruled out.

Thus, the level of material costs aimed at the treatment of patients with diabetic foot syndrome, both for the state and for each individual patient, is very significant and not always justified, which requires revision and optimization of the treatment strategy. One of the reasons for this is the late diagnosis and start of treatment.

Conflict of Interest – None

Ethical aspect – the article is of a review nature and the information presented has a cited reference to primary sources.

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DIABETIK OYOQ SINDROMI KENG TARQALGANLIGI VA XAVF OMILLARI

F.M. Abduraxmanov
Toshkent tibbiyot akademiyasi

ABSTRAKT

Qandli diabetli bemorlar orasida diabetik oyoq sindromining keng tarqalganligi o'rtacha 4-10% ni tashkil etadi. Turli mamlakatlarda qandli diabetning tarqalishi 1,5 dan 6% gacha bo'lishiga qaramay, diabetik oyoq sindromi bilan og'rigan bemorlar barcha travmatik bo'lmagan pastki oyoq-qo'l amputatsiyalarining 40-60% ni tashkil etadi. Diabetik oyoq diabetning mustaqil asoratlari sifatida, ko'z, buyrak, yurak-qon tomir va asab tizimlarining diabetik sindrom bilan birga, 1987 yilda Jenevadagi JSST tadqiqot guruhining hisobotida aniqlandi.

Ushbu ko‘rib chiqish maqolasida qandli diabet bilan og‘rigan bemorlar orasida nogironlik va o‘limning asosiy sababi sifatida diabetik oyoq sindromining tarqalishi to‘g‘risidagi so‘nggi 10 yil davomida adabiyot ma’lumotlari taqdim etiladi.

Tayanch iboralar: Qandli diabet, diabetik oyoq sindromi, tarqalganlik, etiologiya

РАСПРОСТРАНЕННОСТЬ И ФАКТОРЫ РИСКА РАЗВИТИЯ СИНДРОМА ДИАБЕТИЧЕСКОЙ СТОПЫ

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АБСТРАКТ

Распространенность синдрома диабетической стопы среди больных сахарным диабетом составляет в среднем 4-10%. Не смотря на тот факт, что распространенность сахарным диабетом в различных странах составляет от 1,5 до 6%, на больных синдромом диабетической стопы приходится 40-60% всех ампутаций нижних конечностей нетравматического характера. Диабетическая стопа как самостоятельное осложнение сахарного диабета наряду с диабетическим поражением глаз, почек, сердечно-сосудистой и нервной системы выделена в Докладе исследовательской группы ВОЗ в Женеве, в 1987 году.

В данной обзорной статье приводятся литературные сведения за последний 10 лет о распространённости синдрома диабетической стопы – как основной причины инвалидности и летальности среди больных сахарным диабетом.

Ключевые слова: Сахарный диабет, синдром диабетической стопы, распространенность, этиология