SYMPTOMS OF ERECTILE DYSFUNCTION IN PATIENTS WITH DIABETES

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Abstract. Erectile dysfunction (ED), which significantly reduces the quality of life of men, according to foreign researchers, occurs in more than 50% of patients with diabetes, remaining undetected for a long time due to the fact that that patients often do not complain to doctors about sexual dysfunction, and doctors do not conduct sufficiently active questioning of the patient. At the same time, ED may be an early indirect clinical sign of the occurrence and progression of atherosclerosis and neuropathy in diabetes, which demonstrates the need to study various forms of ED to identify risk groups and improve preventive measures. To date, no epidemiological studies have been conducted to study the prevalence and clinical characteristics of ED in patients with diabetes in the Republic of Uzbekistan.

The incidence of erectile disorders in patients with diabetes depends not only on the patient's age, but also on the duration of the underlying disease and the duration of the period of decompensation of carbohydrate metabolism [7, 8]. The development of ED is influenced by the presence of concomitant diseases, complications of diabetes, and the effectiveness of the therapy [9]. Thus, several studies have examined the relationship between the presence of ED and late diabetic complications and have shown that ED was detected almost 2 times more often in patients with diabetic nephropathy or retinopathy [10].

Diagnosed erectile dysfunction in patients with diabetes may be an indirect sign of the development or progression of the atherosclerotic process and coronary heart disease, and may also be the first manifestation of diabetic neuropathy [11, 12].

This study made it possible not only to study the epidemiology and clinical characteristics of ED in patients with diabetes, but also demonstrated the prognostic value of ED in some complications of diabetes.

Erectile dysfunction (ED) is widespread among men with diabetes mellitus (DM) and occurs in 50-60% of patients [10]. In a study by Brunner et al . ED was detected in 49% of patients with type 1 diabetes [3], while among men with type 2 diabetes this figure reached 89.2% [4]. A higher prevalence of ED has been shown among patients with diabetes than among patients with arterial hypertension or among a healthy group of men [4].

The risk of ED in diabetes is 3 times higher than in a healthy population [6|. In a study by Bacon et al . it was shown that, according to regression analysis, men with

type 2 diabetes compared with men without diabetes had a significantly higher risk of developed ED (1.3 for men with type 2 diabetes) [2].

In more than 50% of diabetic patients, ED occurs in the first 10 years of the disease and may precede other complications of diabetes or be their first manifestation [10]. It is known that the incidence of ED is directly dependent on the age of the patient [5], as well as the duration of diabetes [8, 10]. The likely reason for the increase in the frequency of ED in patients with type 2 diabetes with age is the development of concomitant pathology, especially coronary heart disease [9] and arterial hypertension, as well as more frequent use by patients of medications that negatively affect sexual function [7].

We have undertaken work to assess the prevalence and characteristics of ED in patients with short duration of type 2 diabetes in order to establish the dominant factor in its etiology.

The incidence of diabetes mellitus in the world is rapidly increasing. According to the International Diabetes Federation, today more than 5.3% of the world's population suffers from diabetes [4, 8]. The problem is acquiring serious economic importance, since as a result of the severe course of the disease, disability occurs in people at the most working age [2, 7, 9, 11].

According to leading specialized centers, diabetic polyneuropathy ranges from 2 to 15% without special selection of patients, up to 77% with special selection of the total number of patients with elevated sugar levels [1, 5, 13]. At the same time, according to various sources, the incidence of erectile dysfunction (ED) among patients with diabetes reaches 35-55% [3, 6, 10, 12].

The pathogenesis of diabetes mellitus and erectile dysfunction is based on their common process of endothelial dysfunction, identified by an imbalance in the production of endothelial signaling molecules during the transformation of the adaptive response of the endothelium into a maladaptive one [2, 6].

The incidence of diabetes mellitus in the world is rapidly increasing. According to the International Diabetes Federation, today more than 371 million people suffer from diabetes mellitus (DM). Approximately 10% of the total number of patients with diabetes are type 1 diabetes [1].

Sexual disorders, characterized by a decrease in the patient’s quality of life, leading to infertility and social problems, are observed in more than 40% of patients with diabetes [2, 3]. It is important to note that sexual disorders in patients with type 1 diabetes begin at a younger age compared to the population without diabetes [4].

The prevailing sexual dysfunction in patients with diabetes is erectile dysfunction (ED). Numerous studies have shown that ED affects up to 35-55% of patients with type 1 diabetes [5], and the risk of ED in patients with diabetes is 3 times higher compared to the population without diabetes [6].

The incidence of erectile disorders in patients with diabetes depends not only on the age of the patient, but also on the duration of the underlying disease and the duration of the period of decompensation of carbohydrate metabolism [7, 8]. The development of ED is influenced by the presence of concomitant diseases, complications of diabetes, and the effectiveness of the therapy [9]. Thus, several studies have examined the relationship between the presence of ED and late diabetic complications and have shown that ED was detected almost 2 times more often in patients with diabetic nephropathy or retinopathy [10].

Diagnosed erectile dysfunction in patients with diabetes may be an indirect sign of the development or progression of the atherosclerotic process and coronary heart disease, and may also be the first manifestation of diabetic neuropathy [11, 12]. A similar study conducted by Rozhivanov R.V. (2005) on the basis of the Federal State Budgetary Institution "Endocrinological Scientific Center" of the Ministry of Health of the Russian Federation, shows comparable with foreign data on the prevalence of ED among patients with type 1 and type 2 diabetes, the dependence of the prevalence on the age of patients, the duration of the disease, as well as the relationship with the level of compensation of carbohydrate metabolism and the presence of complications of diabetes [13].

Considering the prevalence of ED in type 1 diabetes, as well as the understanding that this condition not only reduces the quality of life of young patients, but can also be one of the symptoms of such complications of diabetes as diabetic neuropathy, coronary artery disease, atherosclerosis, timely, individualized and comprehensive approach to the diagnosis and treatment of ED in this category of patients.

Classification of ED

ED forms:

- Organic (vasculogenic , neogenic , endocrine)
- Psychogenic
- Mixed (organic pathology and psychological factor)
- Medication

The functional state of the penis is regulated by the tone of the smooth muscles of the arterial vessels and trabeculae of the cavernous bodies. After sexual stimulation, nitric oxide (NO), synthesized by the endothelium, increases the concentration of guanylate cyclase (GMP). Increasing concentrations of cyclic GMP (cGMP) lead to relaxation of smooth muscle fibers, increased arterial inflow and venous occlusion in the penis. The rate of cGMP breakdown depends on the activity of the enzyme 5-phosphodiesterase [14].

The development of ED in diabetes may be based on several factors simultaneously (atherosclerosis + neuropathy, neuropathy + psychogenic factor, etc.[15].

Penile erection is regulated by various isoforms of NO- ein thetase of neuronal , endothelial and smooth muscle origin. [16]. Several biochemical mechanisms explain the occurrence of erectile dysfunction in diabetes. The vascular and neurogenic components together are the causes of ED in diabetes, since it is known that endothelial dysfunction leads to the development of ischemic neuropathy, which, in turn, has a negative effect on NO synthesis. Many studies have shown impaired endothelial - dependent and neurogenic relaxation in the corpora cavernosa in patients with diabetes and ED. This finding is associated with NO deficiency. Moreover, some foreign studies have shown a significant increase in the number of NO- synthetase -binding sites in the tissues of the corpora cavernosa of rats 2 months after the induction of diabetes mellitus [17]. This process is similar to those found in other vascular beds, where endothelium-dependent relaxation of the vascular wall was altered as a result of impaired NO synthesis due to high glucose concentrations. Thus, a defect in NO synthetase activity plays a role in the etiology of ED in patients with diabetes, due to diffuse endothelial dysfunction [18]. It was also shown that the relaxation of smooth muscle cells in the corpora cavernosa in patients with diabetes during electrical stimulation was weak due to a decrease in the production of nitric oxide by NO synthetase. It is important to note that long-term hyperglycemia induces an increase in the consumption of nicotinamide adenine dinucleotide phosphate (NADPH), a cofactor in NO production, consequently reducing nitric oxide levels [19].

Excessive generation of free radicals impairs NO- induced relaxation and through the accumulation of advanced glycation end products (advanced glycation end-products (AGE)), circulating in the blood, which are also responsible for the development of diabetic vascular complications [20].

AGE products, accumulating in patients with diabetes, interact with specific receptors of tissues that are exposed to vascular damage, and also increase the expression of mediators of vascular damage, the release of which is also stimulated by glucose. [3].

All of the above factors are involved in the pathophysiology of cardiovascular diseases, characterized by high mortality (painless myocardial ischemia, sudden cardiac death , etc.), which are highly associated with ED [4].

Neuropathy is an important component in the development of diabetic ED. Morphological damage to autonomic nerve fibers in the tissues of the cavernous bodies in patients with diabetes and ED has been shown [25]. The presence of peripheral polyneuropathy is considered characteristic of patients with ED, however, a decrease in the speed of nerve impulse conduction along the nerve fiber and heart rate variability are still recorded slightly more often in patients with diabetes and ED than in patients with ED and polyneuropathies of other origins [6].

Numerous studies devoted to pathological changes in the nervous system in

patients with diabetes refer to independent primary damage to peripheral nerve fibers [17].

Autonomic neuropathy appears to be the main pathogenetic factor of ED in patients with diabetes. Patients with manifestations of peripheral neuropathy more often suffer from ED than patients with diabetes without polyneuropathy [8]. The most substantiated metabolic hypothesis is the theory of polyol metabolism, according to which excess glucose in diabetes is metabolized according to the polyol type, ultimately turning into sorbitol and fructose, the accumulation of which in nerve cells triggers the development of neuropathy [9]. The significance of hyperglycemia in the development of diabetic neuropathy is clinically confirmed by the fact that, provided that compensation of carbohydrate metabolism is achieved, the progression of diabetic neuropathy is reduced by 40-60% [3].

The vasculogenic hypothesis for the development of neuropathy, based on depletion of endoneurial blood flow, increased endoneurial vascular resistance and decreased oxygenation of the nerve, is also important . According to this theory , the primary pathological changes in endoneurial vessels and associated hypoxia and ischemia [1].

All of the above indicates the important role of peripheral neuropathy in the development of ED in patients with diabetes. Many authors characterize this condition as “neurogenic ED,” thereby drawing attention to the leading role of diabetic neuropathy in erectile disorders in such patients [1, 2].

Along with vasculogenic and neurogenic forms of ED in diabetes, endocrine ED associated with androgen deficiency is common [13].

Recent studies have demonstrated that NO synthetase is an androgen-dependent enzyme [14]. The androgen dependence of NO synthetase is evidenced by the fact that androgen receptors were found in the nerve cells of the pelvic parasympathetic ganglia, in which NO and vasoactive intestinal peptide are synthesized [15], as well as stimulation of NO synthesis in the ganglia under the influence of androgens [16]. Moreover, hypogonadism is a common symptom in patients with diabetes [17]. The causes of androgen deficiency in men with diabetes are different. These reasons may be overweight or obesity, as well as an age-related decrease in testosterone secretion. [18].

References

1. Prevalence of erectile dysfunction and its correlates: a population-based study in Morocco / S. Berrada , N. Kadri, S. Mechakra-Tahiri , C. Nejjari // Int J Impot Res. - 2003. - Vol.15, Suppl 1. - P.3-7.
2. Artykova M. A., Nabieva N. A. Radiated semiotics of perfusion brain disorders in epilepsy in children cerebral paralysis. – 2020.

3. Artikova M. A., Nabiyeva N. A. Complicated symptomatic epilepsy, content and distribution of haptoglobin phenotypes in children with cerebral palsy //Turkish Journal of Physiotherapy and Rehabilitation. – Т. 32. – С. 3.
4. Prevalence and correlates of erectile dysfunction in a population-based study in Belgium / R. Mak , G. De Backer, M. Kornitzer , JM De Meyer // Eur Urol-Vol.41(2). - P.132-138.
5. Rozhivanov , R.V. Erectiledysfunction in patients with diabetes mellitus according to control and epidemiological studies / R.V. Rozhivanov , Yu.I. Suntsov,D.G. Kurbatov // Diabetes mellitus. - No. 2. - P.51-54.
6. Ходжиева Д. Т., Ахмедова Д. Б. Турли генезли бош оғриқларида беморларни олиб боришга дифференциал ёндашув ва реабилитация усулларини такомиллаштириш //журнал неврологии и нейрохирургических исследований. – 2022. – Т. 3. – №. 4.
7. Bakhodirovna A. D. Analysis of Side Effects of Drugs Used in the Treatment of Chronic Tension-Type Headache //international journal of inclusive and sustainable education. – 2022. – Т. 1. – №. 4. – С. 54-55.
8. Ахмедова Д. Бирламчи бош оғриқлари фармакотерапияси //Прикладные науки в современном мире: проблемы и решения. – 2022. – Т. 1. – №. 27. – С. 9-13.
9. Ахмедова Д. Б. Сурункали зўриқишдаги бош оғриғи ва мигренда олий нерв фаолияти бузилиши //журнал неврологии и нейрохирургических исследований.–2021.–№. SPECIAL. – Т. 1.
10. Bahodirovna A. D. Effect of Treatment Methods for Chronic Stress Headaches on Electroencephalographic Parameters //Central Asian Journal of Medical and Natural Science. – 2021. – Т. 2. – №. 5. – С. 315-318.
11. Bahodirovna A. D. Tension headaches and psychovegetative disorders //Web of Medicine: Journal of Medicine, Practice and Nursing. – 2023. – Т. 1. – №. 1. – С. 27-29.
12. Ахмедова Д. Б. Бирламчи бош оғриқларини амбулатор шароитда ташхислашдаги хатоликлар //Неврология ва нейрохирургия тадқиқотлар журнали.-Тошкент. – 2020. – Т. 4. – №. 4. – С. 6-8.