STATUS AND PROSPECTS FOR THE FIGHT AGAINST DIABETES MELLITUS

Scientific adviser: **Daminov Abdurasul Taxirovich**
Assistant of the Department of Endocrinology, Samarkand State Medical University

[1]Rabbimova Nilufar Ulugbekovna, [2]Abduxalimova Yulduz Jaxongirovna
Students of the Samarkand state Medical University

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Abstract: Diabetes mellitus remains one of the important medical and social health problems in almost all countries of the world. The prevalence of diabetes mellitus in industrialized countries is 5-6% and tends to increase. This is mainly due to the increase in patients suffering from non-insulin-dependent diabetes mellitus (NIDDM, type II diabetes). Thus, according to S. R. Kahn (1995), in the USA, about 6-7% of the general population are patients with NIDDM. Calculations have shown that if the average life expectancy increases to 80 years, the number of patients with NIDDM will exceed 17% of the total population[8-10]. The great social significance of diabetes mellitus is that it leads to early disability and mortality, which is caused by the presence of late vascular complications of diabetes: microangiopathy (retinopathy and nephropathy), macroangiopathy (myocardial infarction, stroke, gangrene of the lower extremities), neuropathy. Diabetes mellitus is a very common cause of blindness and death from uremia. People with diabetes are at greatest risk of developing cardiovascular disease. More than 40% of all non-traumatic lower extremity amputations are performed due to diabetic foot syndrome and gangrene of the lower extremities[1-7]. The main goals of the Diabetes Mellitus program are to radically increase the duration and improve the quality of life by providing specialized medical care to people with diabetes; carrying out the prevention of diabetes mellitus and its late complications using sufficient material and technical resources[11-14]. The program includes the following sections:

1) organization of diabetes service in the country;

2) organization of a system for the prevention of diabetes mellitus and its complications;

3) providing modern effective treatment for people with diabetes;

4) development of domestic production of medicines and diagnostics, food products for patients with diabetes[15-20].

The implementation of the activities provided for by the Diabetes Mellitus program will help achieve the results recommended by the World Health Organization and the St. Vincent Declaration, including a 33% reduction in the number of patients with late stages of renal failure; a 33% reduction in the number of patients with blindness resulting from diabetic retinopathy; a 50% reduction in the number of lower limb amputations in patients with diabetes mellitus; ensuring the same level of successful pregnancy outcome in women with diabetes as in healthy women; increasing active life expectancy and reducing early disability in people with diabetes[21-25]. Since the first studies, which showed that IDDM is characterized by the presence of autoantibodies to pancreatic islet antigens and, in particular, cytoplasmic (ICA) and cell-surface (ICSA), characteristic polyclonal activation of their formation has been established. In addition to these antibodies, in type I diabetes, other antibodies to pancreatic islet antigens are detected in the blood serum of patients: cytotoxic, to insulin, proinsulin and glutamate decarboxylase. In patients suffering from IDDM, organ-specific autoantibodies to thyroglobulin, thyroid peroxidase, gastric parietal cells, intrinsic Castle factor, adrenal cortex cells, antilymphocytotoxic, to tubulin, activin, immunoglobulins (IgG, Ab) and non-organ-specific autoantibodies are also detected in the blood serum : antinuclear, to smooth muscle fibers, fibroblasts, reticular and mitochondrial, and when treated with insulin - antibodies to exogenous insulin, glucagon, somatostatin, pancreatic polypeptide. All of these antibodies are currently considered as an epiphenomenon or as markers of IDDM. They are not involved in the mechanisms of β-cell destruction[26-31]. The following antigens were isolated in pure form from the islet and beta cells of the pancreas and identified: cytoplasmic, or ICA antigen; glutamate decarboxylase (GAD 65 and GAD 67); 38 kDa protein, secretory granule membranes; antigen 37/40 kDa, not related to GAD; carboxypeptidase H with mol. weighing 52 kDa; periphery with pier weighing 58 kDa; glucose transporter type II (GLUT-2), ICA 69 (Rgp-1); temperature shock protein (65 kDa); protein with mol. weighing 69 kDa with a cross-reaction to the ABBOS protein (a fragment of bovine albumin).A genetic predisposition to IDDM is associated (linked) with certain genes of the HLA system, whereas in NIDDM this connection is not observed. Inheritance of susceptibility to IDDM is a rather complex process, and several HLA genes are involved in the transmission of susceptibility or resistance to diabetes. Moreover, different alleles of the same gene are combined with different pathogenetic mechanisms and different haplotypes are associated with different susceptibility to IDDM. Predisposition to IDDM is combined with the following haplotypes: HLA-DR3, DQw2 (or DQBl*0201) and HLA-DR4, DQw8 (or DQB 1*0302). The clearest association of IDDM is observed with the genes of the DQAl*0501 - DQBl*0302 locus). Allele DQBl*0302 - in the 57th position of the P-

chain of the DQ locus there is no aspartic acid (Asp); allele DQAl*0501 - in the 52nd position of the CC chain of the DQ locus there is an arginine residue (Arg) [32-35]. However, there is still no consensus regarding the exclusive role of HLA genes in the predisposition to the development of diabetes mellitus. This is evidenced by numerous data that in different ethnic groups, predisposition to IDDM is combined with different haplotypes of HLA genes. For example, many authors indicate that the DR4 allele, combined with DQA 1*0301, is significantly positively associated with IDDM in many, but not all races. And in the Chinese population, this allele completely neutralizes the predisposition to diabetes. Another allele (DQB 1*0201) is positively combined in almost all populations, with the exception of the Japanese. This suggests that the predisposition to IDDM, combined with certain HLA genes, is complex[35-37].

Purpose: to study common complications and preventive measures for diabetes mellitus in people of different ages.

Materials and methods: studying complications of the disease, data were collected from patients Endocrinological center for Samarkand the years. The data of 982 patients were studied, of which 563 were men and 419 women. The following antigens were isolated in pure form from the islet and beta cells of the pancreas and identified: cytoplasmic, or ICA antigen; glutamate decarboxylase (GAD 65 and GAD 67); 38 kDa protein, secretory granule membranes.

Study results: An important discovery in recent years has been the identification of mitochondrial gene mutations as a cause of diabetes. Mitochondrial DNA, consisting of 16,569 base pairs, encodes 13 oxidative phosphorylation enzymes. Its mutation usually concerns leucine tRNA - the so-called tRNALeu<UUR\ mutation. For the first time, such a point mutation of mitochondrial DNA was described in MELAS syndrome (mitochondrial myopathy, lactic acidosis, encephalopathy and stroke-like episodes), which often occurs in Japan. A component of this syndrome is the presence of IDDM or NIDDM with or without sensory loss, which usually develops after the clinical manifestation of diabetes.

Conclusion: Already today, based on the achievements of diabetology, it is possible to carry out a set of organizational and therapeutic measures aimed at preventing diabetes and its late complications. It is safe to say that the approved Federal program “Diabetes Mellitus” will help achieve the goals set.

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