MODERN VIEW OF TYPE 2 DIABETES MELLITUS

Safoeva Zebo Farkhotovna
Samarkand State
Medical University.
Samarkand city

Annotation. *As the United States has entered the 21st century, the epidemic of obesity and T2DM continues unabated, affecting more young adults and children than in the past. They will spend longer periods of their lives with the disease. Perhaps partly under the pressure of commercial interests, we as a nation have learned to eat too quickly, too much and the wrong foods. However, the problem of obesity and its consequences is widespread throughout the world, affecting both developing and economically developed countries. In people with energy-saving insulin resistance syndrome (IRS) genes, this excess food and especially insulin-inducing carbohydrates leads to obesity, the IRS phenotype, and T2DM. Almost half of new cases of diabetes in adolescents can be classified as type 2 diabetes.*

Key words*: type 2 diabetes mellitus (T2DM), insulin, autoimmune response, pancreas, β-cells, antigen-presenting cells.*

Currently, in some US states with a large number of ethnic groups predisposed to IRS and T2DM (Hispanics, American Indians, Asian Indians, African Americans), the number of children with T2DM is beginning to rival, if not exceed, the number of children with T1DM. It is estimated that one in three people born in the United States in 2000 will develop T2DM at some point in their lives (5). The rising incidence of type 2 diabetes is associated with an increasing prevalence of obesity worldwide. Approximately 3,700 youth are diagnosed with T2DM each year in the United States, and the number of youth with T2DM is expected to nearly quadruple from 22,820 in 2010 to approximately 85,000

adolescents with T2DM by 2050 (10). The number of young adults is growing at a similar rate. with T2DM have been reported in the UK, India, China and Japan (10). The pathophysiology of type 2 diabetes mellitus is characterized by T2DM. insulin resistance in peripheral tissues (muscle, adipose tissue, liver) with progressive β-cell deficiency, which is especially manifested by impaired insulin secretion in response to glucose stimulation, increased glucose production by the liver, and the absence of markers of the pancreatic autoimmune response. (7). The progressive decline in β-cell function occurs more rapidly in young adults, at a rate of 20–30% per year, compared with a decline of 7–11% per year in adults, even with aggressive drug therapy.

Obesity/insulin resistance (IR) See IRS Intrauterine environment

Epidemiological studies have shown a strong association between poor intrauterine growth and subsequent development of metabolic syndrome. It has been suggested that the effects of poor nutrition in early life impair pancreatic development and lead to irreversible changes in glucose and insulin metabolism (8).

Diabetes mellitus during pregnancy

Studies conducted in Indian Pima women showed a significantly increased risk of developing T2DM in the offspring of women with diabetes during pregnancy compared with mothers without diabetes (9).

Ethnicity

There is a significantly increased risk in some ethnic/racial groups (5). Sex and Puberty Puberty is a state of IR caused by increased secretion of GH during the process. There is a decrease in insulin sensitivity by 30-50% and a compensatory increase in insulin secretion. Individuals with a congenital defect in insulin secretion and an inadequate response to resistance develop diabetes. The average age at diagnosis of type 2 diabetes in children is 13.5 years, which is the time of peak adolescent growth and development.

Glutamate decarboxylase (GAD)

The discovery of Kauffman et al. The striking homology of the 18 amino acid peptide sequence between human GAD 65 and the Coxsackievirus p2-C protein strengthened the evidence for a specific model of molecular mimicry involving GAD. In addition, this specific region of GAD 65 contains a T cell epitope involved in the GAD cellular autoimmune response in humans with immune-mediated diseases, and this region is an early target of cellular immunity in NOD mice (7) glutamic acid decarboxylase (GAD) catalyzes the formation of an inhibitory neurotransmitter γ-aminobutyric acid (GABA) from glutamine (14) There are two forms of GAD (GAD 65 and GAD 67): glutamic acid decarboxylase (GAD) 65 is the predominant form in human pancreatic islet cells, while glutamic acid decarboxylase (GAD 67) predominant in mouse islets. Within the islets, guided tissue regeneration (GTR) is predominantly observed in β-cells, although it has also been proposed to play a role in the inhibition of somatostatin and glucagon secretion, as well as in the regulation of proinsulin synthesis and insulin secretion (8).

Another study also supports the association between Coxsackievirus and T1DM by binding IgM antibodies to Coxsackievirus B as a marker of recent exposure to the virus in newly diagnosed IMD patients and age/sex-matched controls (19). In this report, humoral immunity to coxsackievirus and GAD clustered even in people without diabetes. A series of overlapping synthetic glutamic acid decarboxylase (GAD 65) peptides have been used to study the most reactive T cell determinants in individuals at increased risk of developing T1DM, that is, autoantibody-positive first-degree relatives of patients with T1DM. Increased in vitro T cell responses to glutamic acid decarboxylase peptides GAD 65 (amino acids 247–266 and 260–279) have been observed in patients with newly diagnosed T1DM and in at-risk individuals with autoantibodies (140). The sequence of this region of glutamic acid decarboxylase GAD 65 (amino acids 250–273) is significantly similar to the p2-C protein of Coxsackie B virus (3). However, not all published reports have demonstrated an association between immunity to directed tissue regeneration of GAD and coxsackievirus. For

example, one study identified the non-Coxsackie homologous region of GAD 65 as the predominant cellular immune epitope in a study of human polyclonal T cell responses (5).

Second insulinoma antigen (IA-2).

Tyrosine phosphatase IA-2 is another molecular target of the pancreatic islet autoimmune response in T1DM. In one recent study, an epitope spanning amino acids 805–820 induced the greatest T cell response in all at-risk relatives to 68 overlapping synthetic peptides spanning the intracytoplasmic domain of IA-2 (14). This epitope was found to have 56% identity and 100% similarity in 9 amino acids to the sequence of VP7, the main immunogenic protein of human rotavirus. This dominant epitope also has 75-45% identity and 88-64% similarity in 8-14 amino acids with dengue, cytomegalovirus, measles, hepatitis C and canine distemper viruses, as well as with the bacterium Haemophilus influenzae.

In addition, three other IA-2 epitope peptides have 71-100% similarity in 7-12 amino acids with herpes, rhinoceros, hanta and flaviviruses. The other two have 80-82% similarity to the dietary proteins of milk, wheat and legumes. These molecular mimicries may trigger or exacerbate β-cell autoimmunity.

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