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LATENT AUTOIMMUNE DIABETES IN ADULTS: DIAGNOSIS, TREATMENT AND CLINICAL FEATURES

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ABSTRACT

Latent autoimmune diabetes in adults (LADA) is a special type of diabetes that combines features of both type 1 and type 2 diabetes. The development and progression of LADA is due to multiple factors, including immunological, genetic and environmental. An autoimmune attack on pancreatic beta cells, leading to a decrease in their function, is the central pathogenetic mechanism. In this case, the presence of specific autoantibodies, such as antibodies to glutamate decarboxylase (GADA), serves as an important diagnostic marker. Genetic factors, particularly variations in HLA genes, also play a significant role, indicating a hereditary predisposition to the condition. In addition, environmental factors such as viral infections and lifestyle changes can act as triggers for the onset of the autoimmune process.

ЛАТЕНТНЫЙ АУТОИММУННЫЙ ДИАБЕТ У ВЗРОСЛЫХ: ДИАГНОСТИКА, ЛЕЧЕНИЕ И КЛИНИЧЕСКИЕ ОСОБЕННОСТИ

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Сахарный диабет 1-го типа, LADA диабет, бета-клетки, инсулин, сахар.

ABSTRACT

Латентный аутоиммунный диабет у взрослых (LADA) - это особый тип диабета, который сочетает в себе черты как сахарного диабета 1-го типа, так и диабета 2-го типа. Развитие и прогрессирование LADA зависят от множества факторов, включая иммунологические, генетические и внешние. В основе патогенеза лежит аутоиммунная атака на бета-клетки поджелудочной железы, которая приводит к снижению их функции. Наличие специфических аутоантител, таких как антитела к глутаматдекарбоксилазе (GADA), является важным

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диагностическим признаком. Генетические факторы, в частности вариации в генах HLA, также играют значительную роль, указывая на наследственную предрасположенность к этому состоянию. Кроме того, внешние факторы, такие как вирусные инфекции и изменения в образе жизни, могут стать пусковым механизмом для начала аутоиммунного процесса.

KATTALARDAGI LATENT AUTOIMMUN DIABET: TASHXIS, DAVOLASH VA KLINIK XUSUSIYATLAR

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ABSTRACT

Kattalardagi latent autoimmun diabet (LADA) - bu 1-toifa va 2-toifa qandli diabetning xususiyatlarini birlashtirgan diabetning maxsus turidir. LADA ning rivojlanishi ko'plab omillarga, jumladan immunologik, genetik va atrof-muhitga bog'liq bo'ladi. Pankreatik β- hujayralariga autoimmun hujum, ularning funksiyasini pasayishiga olib keladi. Bu asosiy markaziy patogenetik mexanizmdir. Bunday holda, glutamat dekarboksilaza (GADA) ga o'ziga xos autoantitelalarning mavjudligi muhim diagnostika belgisi bo'lib xizmat qiladi. Genetik omillar, xususan, HLA genlarining o'zgarishi ham muhim rol o'ynaydi, bu holatga irsiy moyillikni ko'rsatadi. Bundan tashqari, virusli infektsiyalar va turmush tarzini o'zgartirish kabi atrof-muhit omillari autoimmun jarayonning boshlanishi uchun triggeri sifatida harakat qilishi mumkin.

Introduction. LADA diabetes (latent autoimmune diabetes of adults) is a type of diabetes characterized by an autoimmune attack on the insulin-producing cells of the pancreas. Unlike type 1 diabetes, which usually develops in childhood or adolescence, LADA diabetes most often affects adults. LADA diabetes usually develops in adults between 30 and 50 years of age, while type 1 diabetes is more common in children and adolescents. LADA diabetes is an autoimmune disease in which the body's immune system attacks and destroys the cells of the pancreas that produce insulin. Type 2 diabetes, on the other hand, is not an autoimmune disease and is most often associated with insulin resistance and impaired insulin secretion.

Symptoms of LADA Diabetes usually develop gradually over several months or years. Type 1 diabetes typically has a more acute onset with severe symptoms. Patients with LADA

diabetes eventually require insulin therapy as their body gradually loses the ability to produce insulin. Patients with type 2 diabetes may initially be treated with oral medications, but over time they may also require insulin. Understanding these differences is critical to the diagnosis and treatment of LADA diabetes. Early detection and treatment can help slow the progression of the disease and prevent complications.

LADA diabetes develops as a result of an autoimmune process that gradually destroys the pancreatic cells that produce insulin. The progression of the disease can be divided into several stages:

Stage 1: In this early stage, the immune system begins to attack pancreatic cells but has not yet caused significant damage to pancreatic function.

During this stage, blood sugar levels may be normal or slightly elevated, and patients may not experience any symptoms.

Stage 2: As the disease progresses, the immune system continues to destroy pancreatic cells, resulting in decreased insulin production. Blood sugar levels begin to rise, and patients may develop symptoms such as increased thirst, frequent urination, and unexplained weight loss.

Stage 3: In this stage, insulin production from the pancreas is significantly reduced and patients become dependent on insulin therapy. Blood sugar levels become more unstable, and patients may experience more severe diabetes symptoms, such as fatigue, blurred vision, and slower wound healing.

The rate of progression of LADA Diabetes varies from person to person. Some patients may develop symptoms within a few months, while others may take several years to progress. Early diagnosis and treatment are critical to slow the progression of the disease and prevent complications. LADA diabetes is an autoimmune disease in which the body's immune system attacks and destroys the cells of the pancreas that produce insulin. This autoimmune process plays a key role in the development and progression of LADA diabetes.

The trigger for the autoimmune reaction is thought to be a combination of genetic and environmental factors. The exact mechanism is unknown, but certain genetic variations are thought to make individuals more susceptible to developing autoimmunity against pancreatic cells. When these susceptible individuals are exposed to certain environmental triggers, such as viral infections, the immune system may mistakenly attack pancreatic cells, perceiving them as foreign.

The attack by the immune system leads to the destruction of the beta cells of the pancreas, responsible for producing insulin. As beta cells are destroyed, the body's ability to produce insulin decreases, leading to elevated blood sugar levels and the development of diabetes symptoms. Unlike type 1 diabetes, in which the autoimmune attack usually occurs quickly and leads to rapid destruction of beta cells, in LADA diabetes the autoimmune process occurs more gradually. This results in slower disease progression and a later onset of symptoms in adults.

The diagnosis of LADA Diabetes is confirmed by the presence of characteristic immunological markers that indicate an autoimmune attack on pancreatic cells:

- ICAs are directed against proteins present in the pancreatic cells that produce insulin. The presence of ICA is a strong indicator of an autoimmune process.

- Autoantibodies to glutamate decarboxylase 65 (GAD65): GAD65 is an enzyme involved in the synthesis of the neurotransmitter GABA. Autoantibodies to GAD65 are common in patients with LADA Diabetes.

-Tyrosine phosphatase autoantibodies (IA-2 and IA-2β): These autoantibodies are directed against intracellular proteins present in the insulin-producing cells of the pancreas. Their presence is also associated with autoimmune diabetes.

The presence of one or more of these autoantibodies, in combination with clinical features of diabetes mellitus and the corresponding age of onset, may help differentiate LADA diabetes from other types of diabetes, such as type 1 diabetes mellitus and type 2 diabetes mellitus.

In addition to autoantibodies, other immunological markers may also be present in LADA Diabetes, such as:

- increased CD4/CD8 T cell ratio
-dysfunction of regulatory T cells
-inflammatory cytokines.

Genetic factors play an important role in the development of autoimmune diseases, including LADA diabetes. Research has identified several genes associated with an increased risk of autoimmune diabetes:

HLA (human leukocyte antigen) genes code for molecules that help the immune system recognize and attack foreign substances. Certain HLA gene variants, such as HLA-DR3 and HLA-DQ2, have been associated with an increased risk of developing LADA diabetes. Non-HLA genes: In addition to HLA genes, other genes associated with autoimmune diabetes have been identified. These include genes involved in the regulation of immune tolerance, T cell activation and function, and cytokine production. These genetic variations can affect the immune response, making individuals more susceptible to developing an autoimmune attack against the insulin-producing cells of the pancreas. However, it should be noted that the presence of these genetic factors does not guarantee the development of LADA diabetes. Other environmental and lifestyle factors also play a role in triggering the autoimmune process.

A family history of diabetes, especially type 1 diabetes, increases the likelihood of developing LADA, as both conditions are characterized by autoimmune destruction of pancreatic beta cells.

However, some patients with LADA have a familial predisposition to type 2 diabetes, highlighting the complex interaction of genetic and immunological factors in the development of the disease. This indicates a unique genetic profile of LADA, where both autoimmune and metabolic predisposition are possible. Although LADA is considered a distinct form of diabetes, its clinical manifestations can vary, creating similarities with either type 1 or type 2 diabetes. In patients with rapid progression and severe loss of beta cell function, LADA often resembles type 1 diabetes mellitus, requiring early initiation of insulin therapy. In cases of slower progression, where partial beta cell function persists for several years, LADA may have features of type 2 diabetes mellitus, with initial glycemic control possible through diet and oral hypoglycemic agents. These features make diagnosis difficult and require careful monitoring for timely adjustment of therapy.

The diagnosis of LADA is established taking into account a number of clinical and laboratory signs. The main diagnostic criteria include age of onset (usually over 30 years), slow progression to insulin dependence, and the presence of autoantibodies such as antibodies to glutamate decarboxylase (GADA) or other beta cell antigens. These antibodies indicate the autoimmune nature of the disease, which makes it possible to distinguish LADA from type 2 diabetes. In addition, C-peptide levels are assessed during diagnosis to determine the degree of beta cell function and the extent of their decline. This comprehensive diagnostic approach helps to accurately classify LADA and develop optimal treatment strategies. The presence of autoantibodies, especially GADA, is the main feature distinguishing LADA from type 2 diabetes mellitus. The presence of these antibodies confirms the autoimmune nature of the disease and helps diagnose LADA in the early stages. Genetic tests can also be useful to help identify genetic predispositions, especially if variations are found in genes associated with autoimmunity, such as HLA. These tests also help assess the risk of disease progression and help individualize your treatment plan.

Conclusion. LADA is a unique form of diabetes characterized by a combination of autoimmune and metabolic mechanisms. Key factors influencing the development and progression of the disease are immunological aspects (autoantibodies and immune attack on beta cells), genetic predisposition (including variations in HLA and other genes associated with autoimmunity), and environmental factors such as diet and viral infections that can serve as triggers. Understanding these factors is critical for early diagnosis, correct classification and effective disease management, thereby slowing the loss of beta cell function and optimizing therapy.

To improve the diagnosis and treatment of LADA, further research is needed aimed at more accurate methods for early detection and prevention of this type of diabetes. Promising directions are the development of screening tests to identify people at high risk of developing LADA, especially taking into account genetic and immunological markers. In addition, it is important to optimize therapeutic approaches that will slow the autoimmune destruction of beta cells and preserve their function as much as possible. This may include both targeted immunomodulatory interventions and personalized insulin therapy and lifestyle regimens tailored to the characteristics of each LADA patient.

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