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DIABETES MELLITUS AND DIABETES INSIPIDUS: A COMPARATIVE ANALYSIS OF THE MOLECULAR BASIS OF PATHOGENESIS, HORMONAL IMBALANCE, AND METABOLIC CONSEQUENCES

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Abstract:
This article provides a comparative analysis of the etiological factors, developmental mechanisms, molecular and biochemical aspects, and clinical features of diabetes mellitus and diabetes insipidus. Although both conditions share common symptoms such as polyuria and polydipsia, their pathophysiological mechanisms differ fundamentally. In diabetes mellitus, carbohydrate metabolism is disrupted as a result of impaired insulin production or biological activity. In diabetes insipidus, water balance is disturbed due to a deficiency of antidiuretic hormone (vasopressin) or reduced renal sensitivity to it. The article discusses the insulin signaling system, the vasopressin–V2

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receptor–aquaporin-2 mechanism, diagnostic criteria, and modern treatment approaches.

Keywords: diabetes mellitus, diabetes insipidus, insulin, ADH, vasopressin, hyperglycemia, insulin resistance, aquaporin-2, water-electrolyte balance, pathogenesis.

Main text: The endocrine system plays an important role in maintaining metabolism, water-electrolyte balance, energy supply, and the stability of the internal environment in the human body. When the function of this system deviates from normal, various pathological conditions may develop. Diabetes mellitus and diabetes insipidus are examples of such disorders.

Diabetes mellitus is a metabolic disease characterized by chronic hyperglycemia and caused by a deficiency of the hormone insulin or impaired insulin action. Its development is associated with insufficient insulin production or decreased sensitivity of tissues and cells to insulin. Insulin is produced by the β-cells of the islets of Langerhans in the pancreas. It facilitates glucose uptake by muscle and adipose tissues, reduces excessive glucose production in the liver, and stimulates glycogen synthesis. There are two main types of diabetes mellitus:

- Type 1: In this form of diabetes mellitus, an autoimmune reaction of the immune system damages the insulin-producing β-cells of the pancreas. As a result, insulin production is markedly reduced or stops completely, leading to insulin deficiency in the body.
- Type 2: In this form, no autoimmune reaction against β-cells is observed. Instead, the sensitivity of tissues and cells to insulin decreases. Initially, β-cells attempt to compensate for this condition by producing more insulin. Nevertheless, over time, the functional capacity of β-cells begins to decline, and hyperglycemia develops.

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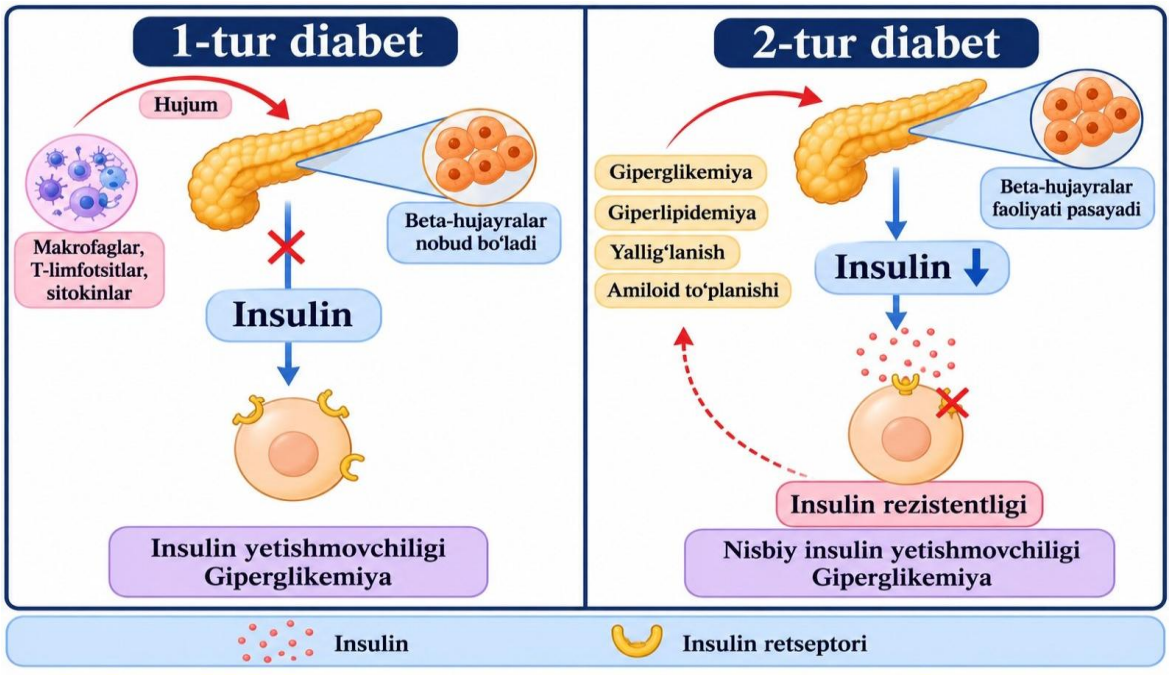
Insulin binds to specific insulin receptors located on the cell membrane. This receptor has tyrosine kinase activity, and its binding to insulin activates intracellular signal-transduction processes. The main mechanism is as follows:

· Insulin binds to the insulin receptor.
· The receptor's tyrosine kinase activity is activated.
· Adaptor proteins that transmit the signal from the insulin receptor into the cell, along with the PI3K–Akt signaling pathway, are activated.
· GLUT4 transporters move to the membranes of muscle and adipose cells.
· Glucose uptake by cells increases.
· Glycogen synthesis is stimulated.



In insulin resistance, the efficiency of this signal-transduction process decreases. As a result, cells cannot take up glucose adequately, and its concentration in the blood rises above the normal range.

Insulin deficiency or reduced insulin action affects not only carbohydrate metabolism but also lipid and protein metabolism. Diabetes mellitus reduces

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glucose uptake by cells and increases gluconeogenesis and glycogenolysis in the liver. Lipolysis becomes activated in the body, increasing the amount of free fatty acids. In type 1 diabetes, severe insulin deficiency may increase the production of ketone bodies. Protein metabolism is also affected: proteolysis increases, and amino acids are used for gluconeogenesis. Chronic hyperglycemia increases the non-enzymatic glycation of proteins. This leads to the formation of advanced glycation end products, which contribute to damage to blood vessels and other tissues. Therefore, if left untreated, long-standing diabetes mellitus increases the risk of diabetic retinopathy, nephropathy, neuropathy, and cardiovascular diseases.

Diabetes insipidus is an endocrine disorder characterized by impaired mechanisms for retaining water in the body. Its principal regulator is antidiuretic hormone (ADH), also known as vasopressin. This hormone is synthesized in the supraoptic and paraventricular nuclei of the hypothalamus. It is then stored in the posterior lobe of the pituitary gland and released into the bloodstream.

An important function of vasopressin is to ensure water reabsorption in the collecting ducts of the kidneys. The main forms of diabetes insipidus are:

➢ Central diabetes insipidus (AVP deficiency).

As a result of impaired function of the hypothalamus or the posterior pituitary lobe, insufficient vasopressin is produced. Possible causes include traumatic brain injuries, certain tumors, neurosurgical procedures, and other diseases.

➢ Nephrogenic diabetes insipidus (AVP resistance).

In this form of diabetes insipidus, vasopressin is present in the body, but the kidneys do not respond to it adequately. In other words, the problem is not with vasopressin itself, but with the receptors or signal-transduction system of renal cells.

In diabetes insipidus, any stage of this pathway may be impaired. As a result, water reabsorption decreases, large volumes of dilute urine are produced, and the

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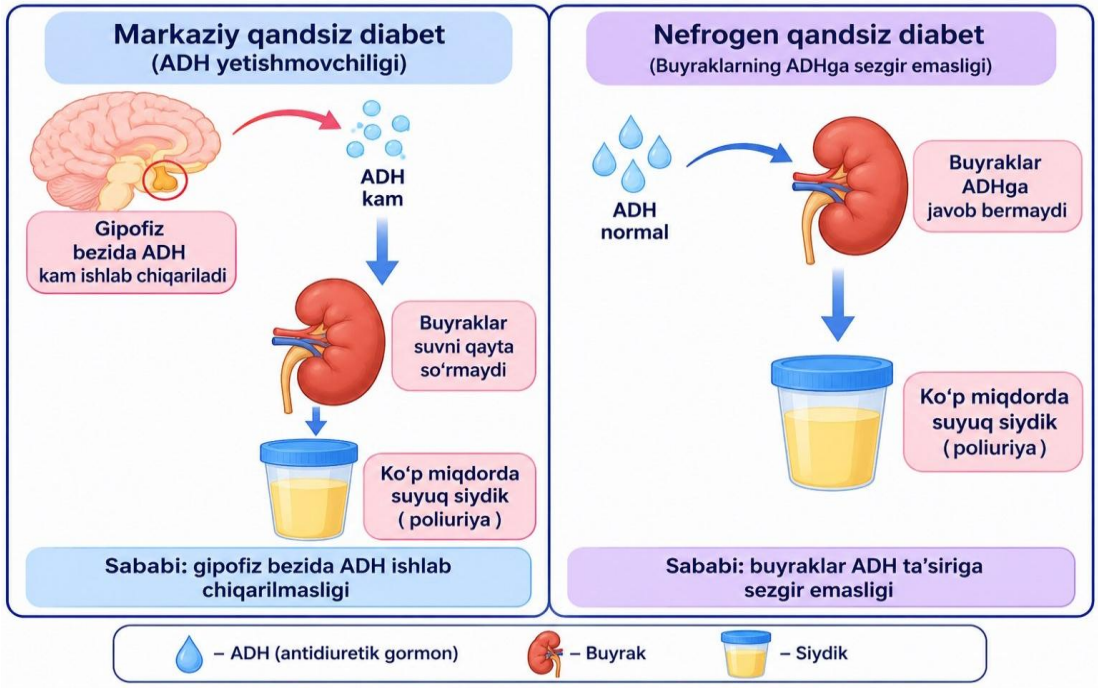
risk of dehydration increases. The main problem in diabetes insipidus is not glucose metabolism, but water-electrolyte balance.



Excessive water loss may lead to the following changes:

- Blood plasma osmolality increases.
- The sodium concentration in the blood may rise (hypernatremia).
- Water shifts from inside the cells to the extracellular space.
- Intense thirst and the need to drink large amounts of water develop.
- Prolonged dehydration may impair blood circulation and kidney function.

In this condition, the body's ability to maintain water balance is significantly reduced.

‣ Clinical Features and Diagnosis

In diabetes mellitus, symptoms may include fatigue, weight loss, blurred vision, and slow wound healing. In diabetes insipidus, the main symptoms are the

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excretion of large volumes of dilute urine, frequent urination at night, intense thirst, and dehydration.

The following parameters are important in diagnosis:

The diagnosis of diabetes mellitus involves measuring blood glucose levels and glycated hemoglobin (HbA1c). A fasting blood glucose level of 7.0 mmol/L or higher is one of the diagnostic criteria for diabetes mellitus. An oral glucose tolerance test (OGTT) and a random blood glucose test are also used.

The diagnosis of diabetes insipidus involves assessing 24-hour urine volume, blood and urine osmolality, and blood sodium levels. Markedly diluted urine, increased plasma osmolality, and, in some cases, hypernatremia are characteristic of this disease. Copeptin testing and a water deprivation test performed under medical supervision are used to clarify the diagnosis.

‣ Modern Principles of Treatment

Treatment of diabetes mellitus depends on the type of disease. Insulin therapy is the mainstay of treatment for type 1 diabetes. In type 2 diabetes, treatment includes dietary regulation, increased physical activity, and medications such as metformin, GLP-1 receptor agonists, and SGLT2 inhibitors.

Central diabetes insipidus is treated with desmopressin. In the nephrogenic form, treatment involves addressing the underlying cause, monitoring fluid balance, and, when necessary, using thiazide diuretics.

Diabetes mellitus and diabetes insipidus develop as a result of disturbances in two different hormonal regulatory mechanisms in the body. In diabetes mellitus, the insulin receptor, PI3K–Akt, and GLUT4 signaling pathways are important, whereas in diabetes insipidus, the vasopressin V2 receptor, cAMP–PKA, and aquaporin-2 system play key roles.

Thus, the first disease involves impaired glucose metabolism, whereas the second involves impaired water reabsorption in the kidneys. Studying these mechanisms is important for understanding the molecular basis of endocrine disorders.

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A comparative study of these diseases at the molecular, biochemical, and clinical levels is important for improving their early diagnosis and developing more effective treatment methods.

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