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Annotation: *Addison's disease, also called adrenal insufficiency, is an uncommon disorder that occurs when your body doesn't produce enough of certain hormones. In Addison's disease, your adrenal glands, located just above your kidneys, produce too little cortisol and, often, too little aldosterone*

Keywords: *kidneys, chronic insufficiency, hypocorisms, pathological condition.*

Chronic insufficiency of the adrenal cortex, or hypocorisms, Eng. Addison's disease is a rare endocrine disorder that causes the adrenal glands to lose their ability to produce enough hormones, primarily cortisol. This pathological condition was first described by the British physician Thomas Addison in his 1855 publication entitled Constitutional and Local Consequences of Disease of the Adrenal Cortex.

Addison's disease can occur due to primary adrenal insufficiency (in which the adrenal cortex itself is affected or poorly functioning), or secondary adrenal insufficiency, in which the anterior pituitary gland does not produce enough adrenocorticotrophic hormone (ACTH) to adequately stimulate the adrenal cortex.

Addison's disease can occur with any lesion of the adrenal cortex or pituitary gland, leading to a decrease in the production of cortisol or aldosterone. Thus, Addison's disease can occur with tuberculous lesions of the adrenal glands, damage to the adrenal cortex by chemical agents (for example, chlodian), non-hormone-producing tumors of the adrenal glands that destroy healthy tissue, etc.

Addison's disease usually develops slowly, over several months or years, and its symptoms may go unnoticed or not appear until some kind of stress or illness occurs that dramatically increases the body's need for glucocorticoids.

The most common symptoms of Addison's disease are:

- ✓ chronic fatigue, gradually worsening over time;
- ✓ muscle weakness;
- ✓ loss of weight and appetite;
- ✓ nausea, vomiting, diarrhea, abdominal pain;

- ✓ low blood pressure, which decreases even more when standing (orthostatic hypotension);
- ✓ hyperpigmentation of the skin in the form of spots in places exposed to solar radiation, known as "addison's melasma";
- ✓ dysphoria, irritability, irascibility, dissatisfaction with everything;
- ✓ depression;
- ✓ craving for salt and salty foods, thirst, drinking plenty of fluids;
- ✓ hypoglycemia, low blood glucose;
- ✓ in women, menstruation becomes irregular or disappears, men develop impotence;
- ✓ tetany (especially after drinking milk) due to excess phosphate;
- ✓ paresthesia and impaired sensitivity of the limbs, sometimes up to paralysis, due to excess potassium;
- ✓ increased number of eosinophils in the blood;
- ✓ excessive amount of urine;
- ✓ hypovolemia (decrease in the volume of circulating blood);
- ✓ dehydration (dehydration of the body);
- ✓ tremor (trembling of hands, head);
- ✓ tachycardia (rapid heartbeat);
- ✓ anxiety, restlessness, inner tension;
- ✓ dysphagia (swallowing disorders).
- ✓ addisonian crisis

In some cases, the symptoms of Addison's disease can come on unexpectedly quickly. This state of acute insufficiency of the adrenal cortex is called "addisonian crisis" and is an extremely dangerous, life-threatening condition for the patient. Any acute illness, blood loss, trauma, surgery, or infection can exacerbate an existing adrenal insufficiency, which can lead to an Addisonian crisis. Addisonian crises are most common in undiagnosed or untreated, or inappropriately low, underdosed corticosteroids in patients with Addison's disease, or in those in whom the dose of glucocorticoids has not been temporarily increased due to illness, stress, surgery, etc.

In previously diagnosed and adequately treated patients, Addisonian crisis may occur as a result of an abrupt cessation of corticosteroid treatment or a sharp decrease in their dose, or an increase in the body's need for glucocorticoids (surgery, infections, stress, trauma, shock).

An Addisonian crisis can also occur in patients who do not suffer from Addison's disease, but who receive or have received in the recent past long-term treatment with glucocorticoids for other diseases (inflammatory, allergic, autoimmune, etc.) with a sharp decrease in the dose or abrupt withdrawal of glucocorticoids, as well as with an increase in the body's need for glucocorticoids. The reason for this is the inhibition of the secretion of ACTH and endogenous glucocorticoids by exogenous glucocorticoids, the

gradually developing functional atrophy of the adrenal cortex during long-term glucocorticoid treatment, as well as a decrease in the sensitivity of tissue receptors to glucocorticoids (desensitization) during therapy with suprphysiological doses, which leads to the patient's dependence on the intake of exogenous glucocorticoids in the body. (“steroid addiction”).

Hormone replacement therapy is prescribed to treat Addison's disease. With a lack of cortisol, hydrocortisone is prescribed; with a low content of aldosterone - fludrocortisone acetate tablets (Florinef). When taking the hormone aldosterone, it is advised to increase your salt intake. Usually, patients with secondary adrenal insufficiency do not need to take aldosterone because this adrenal function is preserved. The dose of the drug is selected individually for each patient.

During an Addisonian crisis, there is a drop in blood pressure and blood glucose, as well as an increase in potassium, which can threaten the patient's life. Hydrocortisone, normal saline (0.9% NaCl), and dextrose (sugar) are usually given intravenously for Addisonian crisis. This usually results in a dramatic improvement. When the patient is able to drink and take drugs by mouth, the amount of hydrocortisone is reduced, keeping only the maintenance dose. With a lack of aldosterone, maintenance therapy with fludrocortisone acetate is prescribed. There are several different causes that can lead to the development of Addison's disease, and some of them have a hereditary component. The most common cause of Addison's disease in the United States and Western Europe is autoimmune destruction of the adrenal cortex. The tendency to develop this autoimmune aggression against the tissues of one's own adrenal glands is most likely inherited as a complex genetic defect. This means that for the development of such a condition, an “orchestra” of several different genes is needed, interacting with as yet unidentified environmental factors.

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