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THE ORIGIN, DIAGNOSIS, AND MODERN CLINICAL DIAGNOSTIC METHODS OF ADDISON'S DISEASE

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Abstract: Addison's disease is a primary adrenal insufficiency (PAI) caused by the destruction of the glandular tissue of the adrenal glands and a decrease in the synthesis of hormones. The disease occurs against the background of autoimmune pathologies, with damage to the endocrine glands by the tuberculous process. Classic symptoms include skin hyperpigmentation, general and muscle weakness, dyspeptic disorders. Immunological and hormonal blood tests and radiation diagnostic methods are used to make a diagnosis. Treatment of Addison's disease includes hormone replacement therapy, diet therapy, and correction of the underlying cause of the pathology.

Key words: Causes, Pathogenesis, Symptoms of Addison's disease, Complications, Diagnosis, Differential diagnosis, Treatment of Addison's disease, Diet, Pharmacotherapy, Prognosis and prevention.

Introduction

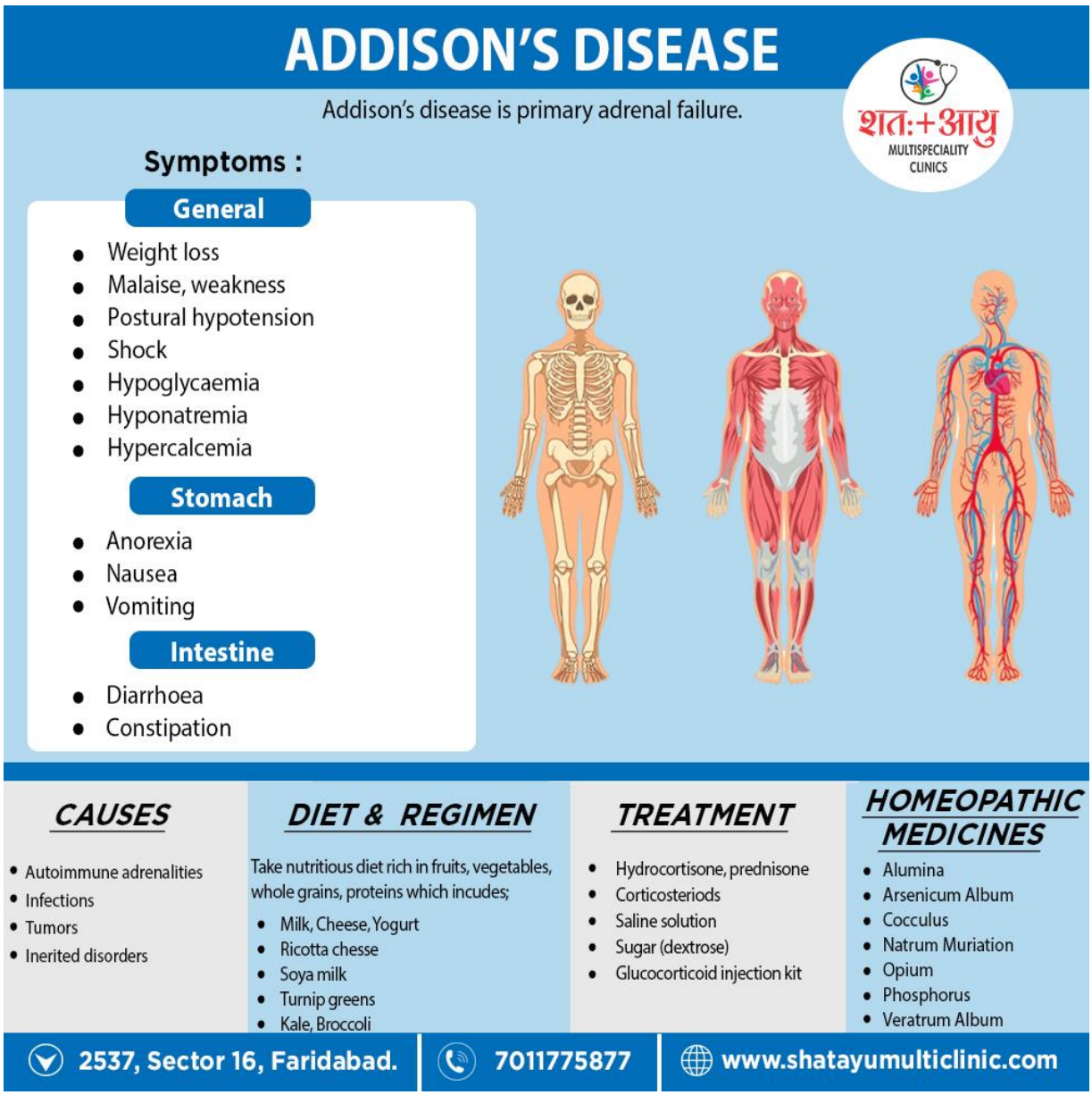
The clinical syndrome associated with damage to the adrenal glands was first described in 1855 by the English physician Thomas Addison. Later, the connection between the characteristic symptom complex and adrenal insufficiency was confirmed by the French physician A. Trousseau, who called it “Addison's disease”. ”. In practical endocrinology, the disease is found with a frequency of 35 to 140 cases per 1 million population. The peak of diagnosis occurs at the age of 20-50 years, the pathology develops more often in women.

Reasons

The main provoking factor of Addison's disease is autoimmune damage to the adrenal glands, which accounts for up to 90% of all causes of the disease. Adrenal insufficiency is often accompanied by other autoimmune processes: vitiligo, type 1 diabetes, hypothyroidism and thyroiditis. When combining such pathologies, a diagnosis is made of polyglandular syndrome. Other causes of adrenal insufficiency:

Tuberculosis. In patients infected with Koch's bacillus, hematogenous spread of infection to the cortex and medulla of the adrenal glands is observed, which leads to the development of specific inflammation. Tuberculosis causes about 10% of cases of Addison's disease.

Adrenal gland damage. In addition to tuberculosis, there is a possibility of developing a nonspecific infectious process in the cortex of the organ. Less common in clinical practice are adrenoleukodystrophy, bilateral hemorrhage in the adrenal glands in Waterhouse-Friderichsen syndrome.



Pituitary gland disorders. When the regulatory effects of adrenocorticotropic hormone (ACTH) are suppressed, adrenal function is reduced. This problem occurs with pituitary tumors, postpartum gland necrosis (Sheehan syndrome), and craniopharyngioma.

Iatrogenic causes. The disease occurs as a result of bilateral total adrenalectomy in the absence of appropriate replacement therapy. Less common causes are hypophysectomy or radiation damage to the pituitary gland. Adrenal insufficiency is provoked by steroidogenesis blockers and some anticoagulants.

At the present stage, the possibility of a genetic predisposition to the occurrence of the disease has been identified. Scientists have been able to establish the association of Addison's disease with HLA monotypes DQ2/DR4-DQ8. Other studies have shown the role of single nucleotide polymorphisms in the CTLA-4 gene region. The development of pathology is associated with a violation of negative selection of lymphocytes in the thymus, resulting in autoimmune reactions.

All of the above causes cause primary adrenal insufficiency, which has a similar pathogenesis and clinical presentation. To avoid confusion in terminology, endocrinologists now use the name "Addison's disease" to refer only to PUI of autoimmune and tuberculous origin. Thomas Addison described these variants of the disease in his works.

Methods

The adrenal glands are a paired endocrine organ, consisting of a cortex and a medulla. The cortex produces a number of hormones: mineralocorticoids (aldosterone), glucocorticoids (cortisol), sex steroids (androgens and estrogens). The mechanism of development of Addison's disease is associated with an insufficient amount of aldosterone and cortisol, which are involved in metabolic processes and affect almost all internal organs.

With a deficiency of the main adaptogenic hormone cortisol, resistance to endo- and exogenous stress factors decreases, with the concentration of glucocorticoids usually increasing tenfold. In addition, patients experience circulatory disorders, dehydration, inhibition of gluconeogenesis, and a decrease in glycogen stores in the liver.

Aldosterone deficiency leads to an increase in potassium levels and a decrease in sodium levels in the blood. With a lack of this hormone, water loss through the kidneys and gastrointestinal tract increases, as a result of which the volume of circulating blood decreases and arterial hypotension develops. Excess potassium disrupts cardiac activity and disrupts intraventricular conduction.

Symptoms of Addison's disease

The clinical picture develops gradually, often patients do not remember the time of manifestation of the disease. The first symptoms of Addison's disease include unexplained weakness, decreased performance and decreased muscle strength. In severe cases, it becomes difficult for the patient to perform ordinary daily activities, the person does not get out of bed, refuses to eat and talk; Fainting occurs against the background of arterial hypotension.

Many patients have dyspeptic disorders: diffuse abdominal pain, nausea and vomiting, stool instability. Symptoms are not clearly related to the nature of the diet, their intensity varies from mild disorders to severe disorders of the digestive function. Due to the large loss of sodium, a craving for salty foods appears. Weight loss is observed against the background of general weakness, decreased appetite, and suppression of anabolic processes.

In patients with Addison's disease, the skin darkens over time and acquires a characteristic bronze color. This symptom has given rise to the unofficial name of the pathology "bronze disease". Hyperpigmentation first appears on open areas of the body exposed to sunlight, as well as in the area of the external genitalia and nipples. Then darkening of the skin is observed in places of constant friction with clothing and natural folds of the body.

Complexities

The most dangerous consequence of Addison's disease is the development of acute adrenal insufficiency (Addisonian crisis). This condition occurs against the background of injury, surgery, or severe infection. Patients develop hypotensive shock, severe dehydration, and hypoglycemia within a few hours. In the absence of timely intensive therapy, Addisonian crisis is fatal.

More than 50% of patients eventually develop mental disorders and neurosis-like states. This is especially true for patients with an undiagnosed illness against the background of increased health anxiety and cancer phobia. With a progressive form of Addison's disease, hallucinations may occur. Hyperkalemia is fraught with the development of myocardial dystrophy and intraventricular blockade.

Diagnostics

Examination of the patient by an endocrinologist begins with an assessment of the clinical manifestations. To make a diagnosis, a set of laboratory tests is required, the scope of which is determined taking into account the clinical situation, the general condition of the patient and the presence of symptoms of other autoimmune syndromes. Diagnosis of Addison's disease requires the following types of studies:

Results: The main methods include assessing the level of cortisol in the blood and urine, if the clinical picture is absent, a test with 1-24-ACTH is recommended. If the level of cortisol according to the results of pharmacological testing is below 500 nmol / l, the presence of primary hypocortisolism can be suspected.

Immunological tests. Antibodies specific to adrenal antigens (21-hydroxylase, desmolase, 17-hydroxylase) are detected in the blood. In polyglandular syndrome, antibodies to antigens of the thyroid gland and other organs are detected.

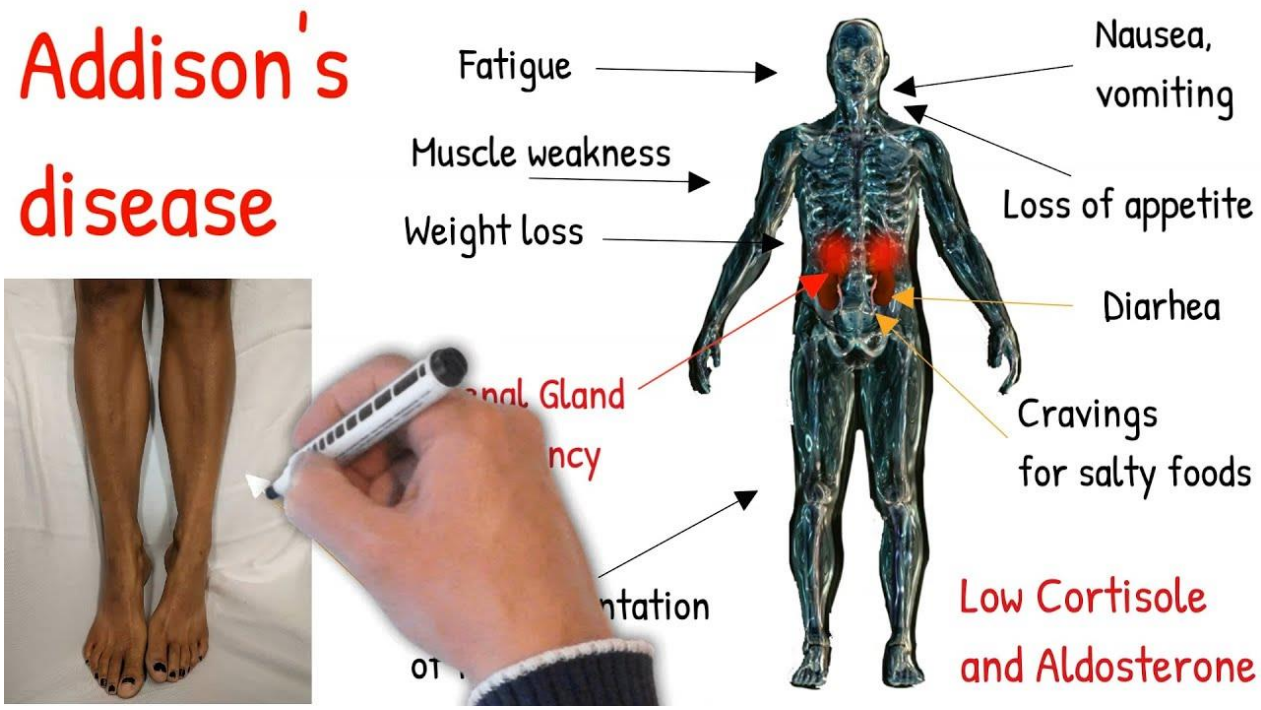
Clinical and biochemical tests. When assessing hemogram parameters, normochromic or hypochromic anemia, moderate leukopenia, relative lymphocytosis, and eosinophilia are detected. According to a biochemical blood test, hyperkalemia and hyponatremia are diagnosed.

Radiological diagnostics. If there is suspicion of a tuberculous etiology of Addison's disease, a chest X-ray is performed. X-ray and computed tomography of the adrenal glands have limited diagnostic value, they can detect an increase in the size of the organ and individual calcifications.

Differential diagnosis

The diagnosis of hypocortisolism is not difficult with a detailed clinical picture and the presence of bronze pigmentation. In patients with subtle manifestations of Addison's disease, differential diagnosis should be made with pernicious anemia, oncopathology, and diseases of the central nervous system. Gastrointestinal symptoms are described as "acute abdomen",

a. It is necessary to distinguish the clinical manifestations of chronic cholecystitis and enterocolitis.
b. Ultrasound examination of the adrenal glands
c. Ultrasound examination of the adrenal glands
d. Treatment of Addison's disease



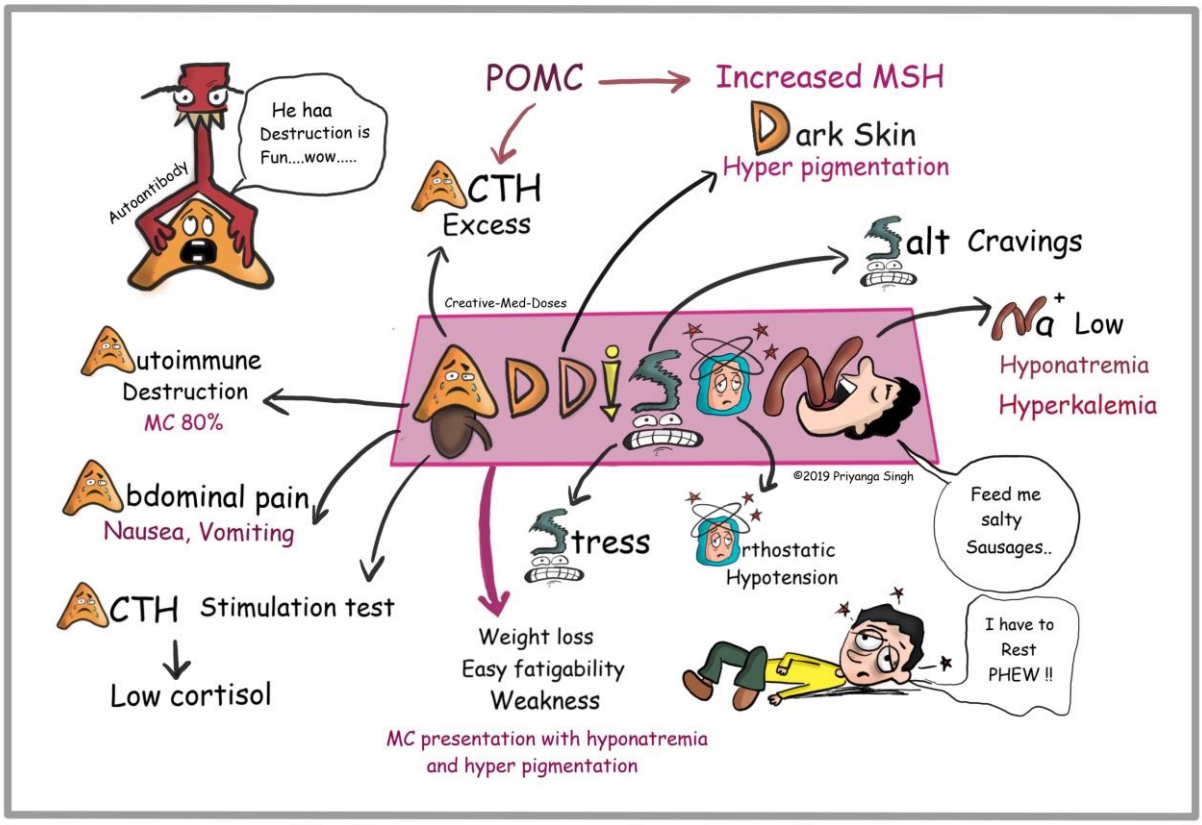
Diet

Diet therapy is important to correct electrolyte imbalances. The diet is enriched with 5-10 g of table salt per day. Since ascorbic acid activates the synthesis of corticosteroids, it is recommended to additionally take vitamin C in high doses. To compensate for the lack of body weight, the diet should contain a high level of protein, a sufficient amount of vegetable and animal fats in the right proportions.

Pharmacotherapy

If possible, etiotropic therapy is prescribed for Addison's disease. When the adrenal glands are affected by tuberculosis, treatment is carried out under the supervision of a phthisiatrician using an individual combination of drugs. To control autoimmune diseases, pathogenetic treatment is recommended: the appointment of levothyroxine, insulin therapy.

The basis of treatment is hormone replacement therapy, which involves the use of glucocorticoids and mineralocorticoids. Correction of mineralocorticoid metabolism is not particularly difficult for this purpose, a single drug is used in standard doses, which shows high effectiveness; The use of corticosteroids has a number of rules and nuances:



the standard regimen involves prescribing short- or medium-acting hormonal drugs in low doses with a dosing frequency of 2 or 3 times a day;

for mild colds and other acute illnesses, the dose of glucocorticoids is increased 2-3 times to adjust the body's need for hormones;

In case of severe illnesses, high fever, or preparation for surgery, it is recommended to switch to intravenous administration of hormones until the condition normalizes.

The use of hormone therapy requires strict clinical and laboratory monitoring to prevent the development of iatrogenic complications. The criteria for successful therapy are the disappearance of subjective symptoms, gradual regression of pigmentation, and maintenance of normal body weight. Patients with Addison's disease are advised to avoid diuretics, which can exacerbate electrolyte disturbances.

Conclusion: With properly selected replacement therapy, the disease does not significantly affect the quality of life and life expectancy of patients. The prognosis is determined by the patient's compliance, the causes and severity of the manifestations of hypocortisolism. Prevention of the disease consists in timely detection and treatment of patients with tuberculosis, dynamic monitoring of patients with other autoimmune pathologies.

It is characterized by nonspecific symptoms: signs of general weakness and attacks of hypoglycemia that develop several hours after eating. The most dangerous complication of chronic hypocortisolism with improper or inadequate treatment is adrenal (Addison's) crisis - an acute decompensation of chronic adrenal insufficiency. More...

Adrenal tumors that occur without signs of hyperaldosteronism, hypercortisolism, feminization or virilization, or vegetative crisis develop asymptomatically. As a rule, they are detected incidentally during MRI, CT scan of the kidneys, or ultrasound of the abdominal and retroperitoneal space. Aldose-formingMore...

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