ETHIOPIAN INTERNATIONAL JOURNAL OF MULTIDISCIPLINARY RESEARCH

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 11, Issue 05, May-2024 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

CLINICAL COURSE, DIAGNOSIS AND TREATMENT METHODS OF ITSENKO-CUSHING SYNDROME

Khamidova Lola, Murtozoyeva Ugiloy, Erkinboyev Jahongir Dilmurodivich

jahongiralimatov08@gmail.com

+998996755099/+998934040014

4th grade students of the Faculty of Pediatrics, Samarkand State Medical University.

Kurbanova Nozima Sobirjanovna

Scientific leader: Samarkand State Medical University,

assistant of the Department of Endocrinology

Abstract

This scientific article provides detailed information about the clinical course of Itsenko-Cushing's syndrome, diagnosis and treatment methods, the causes of the Itsenko-Cushing's syndrome, the basis of the disease.

Key words

Itsenko-Cushing syndrome, pituitary microadenoma, adrenal cortex, Primary and secondary hypercortisolism, Primary hyperfunction, Secondary hypercortisolism, Subclinical form, Iatrogenic hypercortisolism, Functional hypercortisolism, obesity, diabetes, liver disease, hypothalamus syndrome, depression.

Introduction

ITSENKO - CUSHING'S DISEASE [named after neuropathologist N. M. Itsenko (1889-1954) and American neurosurgeon H. U. Cushing (1869-1939)] is a disease caused by excessive production of pituitary adrenocorticotropic hormone (ACTG). Excessive production of ACTG can be caused by pituitary microadenoma. In this case, the function of the adrenal glands increases, the patient becomes obese, blood pressure increases, hirsutism is observed in women, diabetes occurs, the function of the gonads decreases, the skin becomes dry, etc.

It is known that the hormones produced by the adrenal cortex perform very important functions in the body. With insufficient or excessive secretion of hormones of the cortical substance, various diseases. Itsenko-Cushing syndrome is a set of symptoms caused by excessive synthesis of cortical hormones in the adrenal glands. There are several types of this disease and they all have the same symptoms. a group of states given the reasons. This swelling can be hormonal regulation and other malfunctions in the body. Subclinical hypercortisolism can be completely asymptomatic.

Itsenko-Cushing syndrome or hypercortisolism syndrome occurs due to the hypothalamus releasing large amounts of corticoliberins - substances that stimulate the pituitary gland to increase the production of adrenocorticotropic hormone (ACTH), which, in turn, causes excess synthesis. hormones of the adrenal cortex.

There are primary and secondary hypercortisolism:

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 11, Issue 05, May-2024 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

The causes of primary hyperfunction are mainly tumors of the adrenal glands - corticosteromas. They are mainly hormonally active, that is, they produce hormones. A lot of glucocorticoids and a certain amount of androgens and mineralocorticoids are released into the blood. Also, the cause of this type of disorder can be tumors located in other organs that synthesize ACTH-like compounds.

Secondary hypercortisolism occurs due to disturbances in the work of the hypothalamus-pituitary-adrenal system. A short description of the development mechanism of this country is found above. Another cause of secondary hypercortisolism can be a pituitary adenoma, which stimulates an increase in the production of ACTH, which in turn leads to a large release of cortical hormones into the blood. According to the forms of the course, the syndrome of hyperfunction of the adrenal glands is divided into 3 types:

The subclinical form occurs when there are inactive tumors of the adrenal gland. In 10 out of 100 patients, a corticosteroma is detected, which releases cortisol in small amounts, which is not the cause. clinical manifestations. But according to some signs, it can be concluded that there is hypercortisolism syndrome. The patient may have increased blood pressure, disorders in the work of the reproductive system, diabetes.

Iatrogenic hypercortisolism is also called drug-induced or exogenous. Such patients are treated with synthetic corticosteroids for a long time. What is it and in what cases is it used? Long-term corticosteroids are indicated for inflammatory diseases such as rheumatism, various pathologies, kidney diseases, blood diseases and chronic connective tissue diseases. This group of drugs is also given to people who have undergone organ transplantation. Exogenous hypercortisolism occurs in 70% of cases. Functional hypercortisolism obesity, diabetes, liver disease, hypothalamus syndrome, depression. Also, this condition is diagnosed with disputatism in adulthood and youth, pregnancy and alcoholism.

When central hypercorticism is detected, drug treatment with inhibitors of corticotropin and corticoliberin production is prescribed. Patients are usually prescribed Peritol, a serotonin inhibitor, for a 4-week course. It reduces the production of corticoliberin in the hypothalamus. Also, dopamine agonists such as Abergine and Bromergon (Parlodel) for 6 to 10 months.

How is Cushing syndrome diagnosed?

Cushing syndrome is often diagnosed after a thorough medical examination investigating the individual's medical history and conducting a physical exam. Subsequently, multiple laboratory tests (e.g., 24-hour urinary free-cortisol test, overnight low-dose dexamethasone suppression test, or late-night salivary cortisol test) are typically performed, which require samples of bodily fluids, such as urine, saliva, or blood. Oftentimes, the diagnosis of Cushing syndrome is only confirmed after two or more tests demonstrate elevated levels of ACTH and cortisol.

Further testing, including imaging tests (e.g., CT or MRI), may be used to assess whether a tumor is the underlying cause of the individual's Cushing syndrome. Some pituitary tumors are too small to appear on imaging and, instead, require petrosal sinus (i.e. small sinus veins that drain from the pituitary) blood samples.

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 11, Issue 05, May-2024 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

How is Cushing syndrome treated?

The treatment of Cushing syndrome varies depending on the underlying cause. If the disease was caused by the use of a glucocorticoid medication, treatment typically involves lowering the glucocorticoid dose or changing the medication to another steroid class or anti-inflammatory. If the medication is lowered or changed, it will be slowly tapered to avoid adrenal crisis caused by a sudden reduction of cortisol. When Cushing syndrome results from a tumor, the tumor is often surgically removed. If surgery is not a viable option (e.g., the tumor has metastasized) or does not relieve symptoms, radiation therapy may be utilized. After a pituitary tumor is removed, about 6-18 months of cortisol supplementation will typically follow the surgery. If both adrenal glands are removed, cortisol and other hormone supplementation will be needed for the duration of the individual's life.

They reduce the release of corticotropin. In addition, inhibitors of the formation of corticosteroids in the cortical substance of the adrenal glands are prescribed. These are Mamomit and Metapyron drugs. Do not assume that a disorder such as adrenal hyperfunction will be cured quickly. It will take time for all processes to return to normal. Treatment is usually delayed for 6 months or more.

Symptomatic therapy is aimed at correcting carbohydrate, electrolyte and protein metabolism. Prescribe drugs that lower blood pressure and normalize the work of the heart and blood vessels. A frequent complication of adrenal hyperfunction is osteoporosis, so prevention of bone fractures is important in the treatment of this syndrome. To normalize protein metabolism, doctors prescribe anabolic steroids such as Nerobolil and Retabolil. Carbohydrate metabolism If carbohydrate tolerance is broken, I regulate it by correcting the diet, as well as by prescribing insulin or sulfonylurea derivatives.

Because the syndrome of hyperfunction of the adrenal cortex is accompanied by hypoxia, patients should not take biguanides. electrolyte exchange is carried out with potassium preparations. High blood pressure is prescribed antihypertensive therapy, and in case of cardiovascular system disorders - cardiac glycosides and diuretics. To prevent osteoporosis, it is recommended to take drugs that accelerate the absorption of calcium in the intestines: vitamin D derivatives. Calcitonin and Calcitrin are prescribed to fix calcium in bones. To prevent fractures and other unpleasant complications of osteoporosis, therapy should be carried out for at least 1 year.

Itsenko-Cushing's disease is a disease of the hypothalamus-pituitary system. Itsenko-Cushing syndrome - adrenal cortex disease (KN) or malignant tumors of non-adrenal localization, produce ACTH or corticoliberin (bronchi, thymus, pancreas, liver cancer), which leads to hypercortisolism. Hypercortisolism can also be iatrogenic and functional.

Itsenko-Cushing's disease was first described in 1924 by the Soviet neuropathologist Itsenko and in 1932 by the American surgeon Cushing.

The basis of the disease and Itsenko-Cushing syndrome is a tumor process (benign adenomas or malignant) in the hypothalamus-pituitary region or in the adrenal cortex. Itsenko-Cushing's disease is detected in 70-80% of patients, syndrome in 20-30%. In some cases, the disease develops after brain damage or neuroinfection.

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 11, Issue 05, May-2024 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

With Itsenko-Cushing's disease, ACTH secretion control mechanisms are disturbed. In the central nervous system, there is a decrease in dopamine and an increase in serotonergic activity, as a result of which the synthesis of KRG (corticotropin-releasing hormone, corticoliberin) by the hypothalamus increases. Under the influence of CRH, ACTH secretion by the pituitary gland increases, its hyperplasia or adenoma develops. ACTH increases the secretion of corticosteroids - cortisol, corticosterone, aldosterone, and androgens by the adrenal cortex. Chronic long-term cortisolemia leads to the development of the symptom complex hypercortisolism- Itsenko-Cushing's disease.

With Itsenko-Cushing syndrome, tumors of the adrenal cortex (adenoma, adenocarcinoma) contain an excess of steroid hormones, the "negative feedback" mechanism stops working, and there is a simultaneous increase in the content of corticosteroids and ACTH in the blood.

Patients note a change in appearance, the development of obesity with red stretch marks on the skin of the abdomen, chest and hips, the appearance of headaches and bone pain, general and muscle weakness, sexual dysfunction, changes in hairline.

Arterial hypertension is often complicated by damage to eye vessels: narrowing of vessels, bleeding and decreased vision are detected in the fundus. In every 4th patient, intraocular pressure increases, in some cases it turns into glaucoma with visual impairment. Cataracts develop more often than usual.

Menstrual disorders in women develop in the form of oligo-amenorrhea, secondary infertility, atrophic changes in the uterine mucosa and ovaries are observed. In men, there is a decrease in potential, sexual desire.

Astheno-vegetative syndrome. Mood disorders from fatigue, euphoria to depression are characteristic. Acute psychoses, visual hallucinations, epileptoid seizures, convulsions sometimes develop.

Muscle weakness syndrome. Hypokalemia develops due to hypercortisolism and increased muscle protein breakdown. Patients complain of severe weakness, which is sometimes pronounced like this, patients cannot get up from a chair without external help. Examination reveals atrophy of the muscles of the limbs and the front wall of the abdomen.

Syndrome of impaired carbohydrate metabolism. It varies from impaired glucose tolerance to the development of diabetes mellitus ("steroid diabetes"). Glucocorticoids enhance gluconeogenesis in the liver, reduce the utilization of glucose in the periphery (opposition to the action of insulin), enhance the effect of adrenaline and glucagon on glycogenolysis. Steroid diabetes is characterized by insulin resistance, very little development of ketoacidosis, and is well controlled by diet and oral glucose-lowering drugs.

LIST OF REFERENCES:

1. Абдуллаев Н. Каримов Ё. Патофизиология. - Т.: Абу Али ибн Сино номидаги тиббиёт нашриёти, 1998.
2. Патологическая физиология: Под ред. А. Д. Адо, JI. М. Ишимовой. 2-е изд., перераб. и доп. - М.: «Медицина», 1980

eISSN: 2349-5715 pISSN: 2349-5707

Volume: 11, Issue 05, May-2024 SJIF 2019: 4.702 2020: 4.737 2021: 5.071 2022: 4.919 2023: 6.980 2024: 7,662

3. Патологическая физиология. Под ред. чл.-корр. АМН СССР Н. Н. Зайко. 2 -е изд., перераб. и доп. -Киев: «Вища школа», 1985.

4. Патологик физиология. Под редакцией Н.Н. Зайко и Ю.В.Быця. - 4-е изд. Москва. «Мед-пресс» - ияформ».- 2007 г.