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TREATMENT AND DIAGNOSIS OF ALDESTEROMA

Xo'jakov Murodjon Oybekovich

Andijan State Medical Institute, Uzbekistan

Annotation: Aldosteroma is a hormone-active tumor of the glomerular epithelium of the adrenal cortex, leading to the development of clinical Conn's syndrome-primary aldosteronism. Among the symptoms of aldosteroma are cardiovascular (arterial hypertension, headaches, cardialgia, visual impairment), neuromuscular (myopathy, myalgia, paresthesia, convulsions), renal (polyuria, polydipsia, isostenuria). The diagnosis of aldosteroma is established by the characteristic clinical picture of the disease, the results of laboratory tests and instrumental studies: ultrasound, scintigraphy, CT (MRI), angiography and selective venography of the adrenal glands. With aldosteroma, radical removal of a tumor with an affected adrenal gland (adrenalectomy) is indicated.

Key words: Aldosteroma, myopathy, myalgia, paresthesia, convulsions.

The symptom complex caused by increased production of the mineralocorticoid hormone aldosterone was first described by D. Conn, and was called "primary aldosteronism" or Conn's syndrome. In 70-85% of cases, primary hyperaldosteronism is caused by adrenal adenomas, in other cases-hyperplasia of the adrenal cortex, thyroid or ovarian tumors with hormonal activity.

In clinical endocrinology, aldosteroma is understood as an aldosterone-secreting adrenal adenoma, the development of which is accompanied by signs of primary aldosteronism. Aldosteromas in most cases are benign, in less than 5% of cases – malignant. Aldosteroma, as a rule, is detected at the age of 30 to 50 years, and in women 3 times more often than in men. Cases of aldosteroma development in childhood are described. The causes of aldosteroma, as well as many other tumor formations of the adrenal cortex, are reliably unknown. Presumably, heredity plays a certain role in its development.

Pathogenesis

Aldosteroma is manifested by autonomous excessive (increased by 40-100 times) secretion of the mineralocorticoid hormone-aldosterone, which regulates water and electrolyte metabolism in the body. High levels of aldosterone lead to increased reabsorption of sodium ions in the renal tubules and increased excretion of potassium, magnesium and hydrogen ions in the urine, which contributes to fluid retention, hypervolemia, hypokalemia and metabolic alkalosis, pathological changes in various organs and systems. A feature of primary aldosteronism in aldosteroma is the low activity of renin in blood plasma.

A benign aldosteroma is a small (no more than 1-3 cm) yellow-brown adrenal gland tumor surrounded by a thin connective tissue capsule. Benign aldosteroma can be combined with atrophy or hyperplasia of the surrounding areas of the adrenal cortex. Primary malignant aldosteroma, which develops from its own elements of the adrenal cortex, is characterized by rapid growth, large size and mass; sometimes with a small size of the formation, there may already be signs of metastasis.

Aldosteromas are more often single (up to 70-90% of cases), in 6% of cases – multiple with bilateral localization. Morphologically, aldosteromas have a heterogeneous structure: they can consist of cells similar to cells of the bundle or reticular zone.

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Symptoms of aldosteroma

Clinical manifestations of aldosteroma are caused by disorders associated with primary aldosteronism, and are represented by three main syndromes - cardiovascular, neuromuscular and renal.

Cardiovascular syndrome in aldosteroma is mainly caused by sodium and water retention, hypervolemia, development of edema of the inner lining of the vascular wall (intima) and narrowing of the vascular lumen, increased peripheral resistance, increased vascular reactivity to the action of pressor factors, in particular, aldosterone. The clinical picture of aldosteroma is characterized by constant moderate or severe arterial hypertension, headache, development of fundus changes (hypertensive angiopathy, angiosclerosis, retinopathy and neuroretinopathy), cardialgia, hypertrophy, and later - left ventricular myocardial dystrophy.

Neuromuscular syndrome is associated with potassium and magnesium deficiency, hyperchloremic acidosis, and dystrophic changes in muscle and nerve tissue. With aldosteroma, this is manifested by fatigue, muscle weakness of varying degrees of severity, constipation, pain in the fingers and feet, calf muscles, often - paresthesia and convulsions. With aldosteroma, hypokalemic crises can occur, accompanied by acute headache, vomiting, shortness of breath, decreased (loss) vision, myoplegia, sometimes the onset of flaccid paralysis or convulsive attacks, complicated by the development of acute coronary insufficiency, acute cerebrovascular accident (stroke).

With malignant aldosteromas, along with the main symptoms, abdominal pain, fever, and other signs of intoxication may appear. About 10% of aldosteromas are asymptomatic.

Complications

With aldosteroma, kaliepenic nephropathy develops, which is manifested by a violation of the concentration ability of the kidneys, thirst, copious and frequent urination (daily diuresis of up to 10 liters), nocturia, isostenuria. Peripheral edema is not typical for aldosteroma. In severe chronic hypokalemia, myocardial excitability, insulin secretion by pancreatic b cells, and glucose tolerance are impaired.

Diagnostics

Diagnosis of aldosteroma is based on the characteristic clinical manifestations of the syndrome, the results of laboratory tests, functional tests, and instrumental studies. 2 weeks before the examination, the patient should stop taking antihypertensive drugs.

- Instrumental visualization. Ultrasound of the adrenal glands and radioisotope scanning (scintigraphy) of the adrenal glands are used to identify existing pathological changes and clarify their nature (hyperplasia, tumor), CT of the adrenal glands and MRI of the adrenal glands - to determine the localization and size of aldosteroma.
- Angiography. X-ray methods for the diagnosis of aldosteroma-pneumosuprarenography and adrenal angiography can give inaccurate results due to the small size of the tumor and its poor vascularization. Selective adrenal venography with simultaneous determination of the levels of aldosterone and cortisol in the blood of the adrenal veins is the most informative, although it is technically difficult and fraught with complications. Aldosteroma is characterized by a multiple increase in the aldosterone/cortisol ratio.
- Laboratory diagnostics. In the general analysis of urine with aldosteroma, low relative density and alkaline reaction, proteinuria, increased daily excretion of potassium and aldosterone are detected. A biochemical blood test reveals hypernatremia, hypokalemia, high basal serum aldosterone levels, decreased plasma renin activity, and hypochloremic alkalosis.

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- Special samples. In order to diagnose primary aldosteronism with aldosteroma, a test with spironolactone, a test with a load of hydrochlorothiazide, and a "marching" test are performed.

Differential diagnosis

Differential diagnosis of aldosteroma is performed with diffuse small-nodular hyperplasia of the adrenal cortex, arterial hypertension caused by other syndromes (Itsenko-Cushing syndrome, malignant hypertension, renovascular hypertension, imaginary mineralocorticoid excess syndrome, etc.), nephritis with potassium loss, diabetes insipidus, hyperparathyroidism, tetany, secondary aldosteronism.

Treatment of aldosteroma

Treatment of patients with aldosteroma consists in radical removal of the tumor along with the affected adrenal gland – adrenalectomy. If the location of the aldosteroma is known, lumbar or thoracolumbal accesses are used for surgical intervention on the corresponding side, if the localization is not determined, transperitoneal access to both adrenal glands is used.

In the preoperative period (within 7-10 days), a diet with a limited sodium content, potassium preparations and aldosterone antagonists are prescribed. Glucocorticoid therapy is indicated to prevent the development of acute adrenal cortical insufficiency due to surgery for aldosteroma. After surgery, it is necessary to monitor the level of electrolytes and ECG indicators.

Forecast

Removal of aldosteroma in 50-70% of cases contributes to the normalization or significant reduction of blood pressure, if moderate hypertension persists, corrective conservative therapy is performed. With benign aldosteroma and the absence of irreversible changes in the kidneys, the prognosis is favorable. Malignant aldosteromas have an unfavorable course and prognosis.

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