SPECIFICITY OF CUSHING'S SYNDROME

Kholmatova. Yu.A.
Tashkent Pediatric Medical Institute
5A510102 Master of degree Endocrinology

Abstract: The article analyzes the specificity of carbohydrate metabolism in Cushing's syndrome from a scientific point of view. Thus, the article draws conclusions about carbohydrate metabolism in Cushing's syndrome.

Key words: Cushing's syndrome, steroid diabetes mellitus, hyperglycemia, carbohydrate metabolism, hypercortisolism.

Cushing's syndrome or hypercorticism develops due to increased synthesis of adrenocorticotropic hormone from pituitary adenoma and chronic cortisol secretion as a result of its continued effect on the adrenal glands.

Among neuroendocrine diseases, Cushing's syndrome is the most common, with the development of pituitary adenoma occurring in 2-3 cases per 1 million population, and adrenal tumor-related type in 0.6 cases per 1 million population. The disease is prevalent among women and men in a 3: 1 ratio. Cushing's syndrome is one of the leading causes of disability after diabetes and osteoporosis among endocrine diseases.

Carbohydrate metabolism disorders are observed in most neuroendocrine diseases, and in almost 70% of patients with Cushing's syndrome, carbohydrate metabolism disorders are detected to varying degrees. While 20-50% of patients with hypercorticism are diagnosed with diabetes, impaired glucose tolerance occurs in 30-65% of patients.

Hypercorticism is manifested by clinical signs such as weight gain, increased blood pressure, depression, purple streaks, hyperpigmentation, muscle weakness, decreased libido, hirsutism, menstrual cycle disorders. In turn, the disease increases the risk of arterial hypertension, diabetes, central obesity, hyperlipidemia and hypercoagulability.

The relevance of the study of carbohydrate metabolism disorders in patients with Cushing's syndrome is explained by the prevalence of glucose metabolism disorders in the disease and the difficulty in choosing hypoglycemic therapy in these patients. In this group of patients, the effectiveness of treatment of hyperglycemia is reduced due to the difficulty of achieving remission of the underlying disease or complete cure of the disease, and special drugs used in the disease, in turn, cause hyperglycemia. The basis of these disorders is insulin resistance, which occurs due to hypersensitivity of counterinsulatory hormones. In such a situation, it is difficult to compensate for carbohydrate metabolism with both medical and surgical procedures. It is important to look for ways to reduce carbohydrate metabolism disorders among patients with neuroendocrine diseases and to develop effective hypoglycemic therapy regimens.

Disorders of carbohydrate metabolism are a frequent symptom of Itsenko-Cushing's disease Patients with this pathology may experience en-precrinologists, family doctors diagnosed with type 2 diabetes, hypertension, dyslipidemia, osteoporosis. At the same time, the secondary genesis of the clinical symptoms of the disease often remains unrecognized for a long time, which contributes to the progression of the disease and the development of complications. A cohort of patients with Itsenko-Cushing's disease has a high risk of atherosclerotic cardiovascular disease, which can continue despite remission of the disease and normalization of cortisol levels [1].

We have presented the patient's case history, which was observed by primary care specialists for type 2 diabetes and hypertension. A thorough examination of the patient, in which attention was drawn to the rapid weight gain, dysplastic structure, matronism, made it possible to suspect the secondary nature of the disorders and establish the correct diagnosis of Itsenko-Cushing's disease. The performed surgical treatment made it possible to eliminate the clinical manifestations of hypercortisolism, namely, secondary diabetes mellitus, dysplastic obesity, hypertension. We draw the attention of various specialists to the problem of

hypercortisolism and the possibility of developing atypical, erased forms of this pathology, without characteristic skin symptoms of the disease. Endogenous hypercortisolism, in our opinion, should be inappropriate in patients control of diabetes in combination with hypertension, dysplastic obesity or rapid weight gain. Such patients require a comprehensive endocrinological examination, including a study of cortisol, a small dexamethasone test, an assessment of the content of thyroid-stimulating hormone, lipid profile, as well as the state of bone tissue [2].

Steroid diabetes mellitus is an endocrine pathology that develops as a result of a high content of adrenal cortex hormones in the blood plasma and a violation of carbohydrate metabolism. It is manifested by symptoms of hyperglycemia: rapid fatigue, increased thirst, increased profuse urination, dehydration, increased appetite. Specific diagnosis is based on laboratory detection of hyperglycemia, assessment of the level of steroids and their metabolites (urine, blood). Treatment for steroidal diabetes includes withdrawal or reduction of glucocorticoid dosage, surgery to reduce the production of corticosteroid hormones, and antidiabetic therapy.

Steroid diabetes mellitus (SD) can be triggered by a prolonged increase in the secretion of corticosteroids or by taking them in the form of drugs. In the second case, the disease has a synonymous name - drug diabetes. Initially, it is not associated with the functional state of the pancreas, develops against the background of hormonal treatment and can go away on its own after drug withdrawal. SJS, provoked by an increase in natural hormones, is most often observed in Itsenko-Cushing's disease. In this group of patients, epidemiological indicators reach 10-12%. There is no exact information on the prevalence of SJS in he general population.

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