

**DISEASES ACCOMPANIED WITH DISORDERS OF THE HYPOTHALAMO-  
PITUITARY SYSTEM**

**Adusalomov Asadbek Xasanboy o'g'li**  
Student of Andijan State Medical Institute

Research advisor: **Madumarova Makhfuza Maksimovna**

Senior teacher of the Department of Pathological Physiology

Student of Andijan State Medical Institute

**ABSTRACT:** In this article, we will learn about hypothalamo-pituitary system diseases and their clinical concepts. it contains the centers of the vegetative nervous system; hypophysis and are connected organically.

**KEY WORDS:** Hypothalamus, pituitary gland, endocrine systems, Cushing's disease, Diabetes insipidus, Etiology, chromosome, somatotrope and gonadotrope, testosterone, free cortisol, hypopituitarism, antidiuretic hormone.

There are more than 30 pairs of nuclei in the hypothalamus. These nerve cells produce neurohormones such as vasopressin and oxytocin, released from the pituitary gland, as well as releasing factors that increase or decrease the secretion of hormones under the influence of the pituitary gland. They affect the pituitary gland and force it to secrete various complex hormones. A slight change in the hypothalamus, sugar, salt, water, hormones, etc. in the body. provided with receptors that sense changes in the amount of substances. The hypothalamus controls metabolism, cardiovascular, digestive, excretory and endocrine glands, sleep, alertness, and excitement mechanisms. Connects between the nervous and endocrine systems. The hypothalamus is an important "control center" in our body, an area of the brain in the diencephalon, located below the thalamus (=hypo). As a higher center, the hypothalamus coordinates water and salt balance and blood. It maintains a constant body temperature and regulates food intake. The hypothalamus affects our emotional and sexual behavior and determines when we sleep and when we wake up. It is also an important control organ in the endocrine system because it regulates when and when . how many hormones are produced. The hypothalamus is the highest center of the vegetative nervous system under the cortex. It controls the vegetative activities that ensure the stability of the internal environment of the body, and the centers that control the metabolism of proteins, fats, carbohydrates and salt are also located in the hypothalamus. cannot maintain. Diabetes insipidus, Sheehan's syndrome, Itsenko-Cushing's disease (etiology, pathogenesis, clinical appearance and treatment) Diabetes insipidus is a clinical symptom, expressed by a decrease in the concentration function of the kidney. This is antidiuretic hormone (ADH) deficiency - m. Diabetes insipidus, Sheehan's syndrome, Itsenko-Cushing's disease (etiology, pathogenesis, clinical appearance and treatment) Diabetes insipidus is a clinical symptom, expressed by a decrease in the concentration function of the kidney. This is due to deficiency of antidiuretic hormone (ADG) - central diabetes insipidus or insensitivity of renal tubules to ADG effect - peripheral (renal) diabetes insipidus. Classification according to the pathogenetic principle 1. Central (hypothalamo-pituitary diabetes insipidus) idiopathic (in 1/3 cases) symptomatic (in 2/3 cases of all diabetes insipidus) 2. Kidney form. Etiology is idiopathic (in 1/3 cases) Wolfram syndrome (DIDMOAD syndrome), diabetes, optic nerve atrophy, deafness accompanied by bladder atony, acute or chronic infections: influenza, angina, meningoenzephalitis, scarlet fever, pertussis, all forms of typhoid, septic conditions, tuberculosis,

syphilis, malaria, rheumatism, brain injuries (accidental or surgical), mine additions to GGS, birth trauma, primary tumor or metastases, hypothalamic adenoma, pituitary adenoma, teratomas, gliomas, craniopharynomas, every 5th case is associated with neurosurgical intervention. Women and men are affected equally 1:1 at the age of 20-40. Pathogenesis. 2 ADG deficiency leads to impaired concentration of urine in the distal tubules of the nephron, resulting in an increase in urine volume and a decrease in specific gravity. In order to prevent dehydration, the thirst center is stimulated and polydipsia develops. Hyperosmolar dehydration develops if you refuse to drink water. When the gastrointestinal tract is strained with fluid, the stomach descends, dyskinesia of the gall bladder, and the syndrome of "sensitive bowel" develops. If there is a sudden injury, the course of the disease will be aggravated. Kidney form is more common in children. It is associated with anatomical incompleteness of kidney nephrons or impaired production of tsAMF (in renal distal tubule cells or collecting tubules) with an enzymatic defect. Renal form can also develop in amyloidosis sarcoidosis chronic hypokalemia tubulopathy chronic hypercalcemia X-linked recessive. Clinic. The severity of the disease, the expression of polyuria and polydipsia depends on the degree of deficiency of ADG. Polyuria 10-18-40o/day and many children have a lot of evening diuresis (nocturia). Urine is colorless, without pathological elements, specific gravity 1000-1005. The disease starts acutely, suddenly, rarely slowly. Physical and mental development lags behind due to polyuria and polydipsia. Appetite is reduced, body weight is reduced, and in some cases obesity occurs. Skin and mucous membranes are dry. Children lag behind in growth, lag behind in physical and sexual development. If the liquid enters in sufficient quantity, the cardiovascular system will not suffer. As dehydration increases, patients experience headache, nausea, vomiting, increased body temperature, blood clotting, increased Na, erythrocytes, hemoglobin, residual nitrogen, tremors, and psychomotor agitation. Diagnostic diabetes insipidus develops after childbirth complicated by typical symptoms: low specific gravity of urine (1000-1005), hyperosmolarity of blood serum (>290 mosm/l), hypoosmolarity of urine (290 mosm/l, hypoosmolarity of urine 1l). Again, frequent pregnancies and childbirths predispose the pituitary gland to functional stress. In very rare cases, it can develop as a result of ischemic changes in the pituitary gland, sometimes even with gastrointestinal bleeding. Relatively rare causes: pituitary adenoma with hemorrhage or infarction, metastases (lung cancer, breast cancer), granulomatous diseases, inflammatory diseases, brain box injury, hypophysectomy, congenital aplasia or hypoplasia. Pathogenesis. Pangipopituitarism is based on the deficiency of growth hormones and growth hormone. As a result, secondary hypofunction of the adrenal, thyroid and reproductive (sex) glands develops. In rare cases, as a result of the addition of the leg or the posterior lobe of the pituitary gland to the process at the same time, it leads to a decrease in the amount of vasopressin and the development of diabetes insipidus. Growth hormone deficiency causes atrophy of internal organs and smooth and skeletal muscles with a universal effect on protein synthesis. Decreased production of prolactin causes agalactia. Clinic. The clinic of pangipopituitarism is determined depending on the extent of destruction of the adenohypophysis and the rate of development. More young women (20-40 years) are affected, but it can also develop in younger and older people. First, somatotropic and gonadotropic activity decreases, then thyrotropin and adrenocorticotrophic function decreases. Decreased size of internal organs, muscle atrophy is observed. Weight loss can be satisfactory - 2-6 kg per month and severe - 25-30 kg per month. Swelling is not bothersome. It is characteristic that the skin becomes white-yellow, waxy, dries, wrinkles, and changes. Hair loss is observed in the armpits and groin area. The general appearance is unique. Sweating and secretion of sebaceous glands decrease, melanin synthesis (MSG deficiency) decreases, resulting in depigmentation of the inguinal skin and teats. Loss of teeth, decalcification of bones, premature graying of hair, brittleness and shedding are observed.

Marasmus and the involution of old age develop rapidly. Acute weakness, apathy, adynamia leading to immobility, hypothermia, collapse (orthostatic) comatose states are characteristic and lead to the death of the patient without special treatment. A decrease in thyroid stimulating hormone (TTG) leads to hypothyroidism either quickly or slowly. Drowsiness, chills, adynamia, weakness, bradycardia occur. constipation and atony develop in the gastrointestinal system. Hypothyroidism and hypogonadism can cause swelling. Violation of the sexual environment takes the main place in the clinic. Sexual disorders often allow all other symptoms to develop. External and internal genitals atrophy. Menstruation stops in women, breast size decreases. When it develops after childbirth, agalactia and amenorrhea are observed. In men, loss of secondary sexual characteristics (grow, axillary hair, mustache, beard), atrophy of the prostate gland, testicles, penis, and decrease in testosterone are observed. Diagnosis. In typical cases, the diagnosis of pangipopituitarism does not cause complications. Symptoms of gonadal, thyroid, and adrenal insufficiency after a complicated birth or for other reasons indicate hypothalamic-pituitary insufficiency. In Simmonds-Sheehan disease, laboratory tests often reveal hypochromic and normochromic anemia, mainly in severe hypothyroidism, leukopenia with eosinophilia, and lymphocytosis. low blood glucose, high cholesterol. Hormonal tests reveal a low level of hormones of the peripheral endocrine glands (T4, testosterone, free cortisol, daily excretion with urine) or a decrease in the amount of tropism and growth hormones. To determine the reserve of pituitary hormones, it is recommended to conduct a test with releasing hormone (thyroliberin, gonadotropin-releasing hormone). Comparative diagnosis: Simmonds-Sheehan disease is compared with diseases that cause weight loss (malignant tumor, tuberculosis, enterocolitis, etc.). In these diseases, weakness and weight loss develop slowly, and anemia prevails, which is the consequence of the disease. Acute anemia creates a basis for comparative diagnosis with blood diseases. Hypoglycemia can stimulate organic hyperinsulinism (insulinoma) in hypopituitarism. 5 Pituitary cachexia in clinical practice is often compared with psychogenic (nervous) anorexia, as it occurs as a result of refusing food in order to lose weight in young girls. decisive in the comparative diagnosis - anamnesis, weight loss, preservation of mental activity and physical activity even at the last stage, preservation of secondary sexual characteristics along with genital atrophy and tendency to hypertrichosis. The amount of tropic hormones can be normal, high or low, but when their normal production is determined using hormonal stimulation tests, functional disorders are observed in anorexia nervosa. Treatment. The treatment of hypopituitarism is replacement therapy in case of hormonal deficiency and elimination of the cause of the disease, if possible.

#### **REFERENCES:**

1. Ismailov S.I. "Thyroid diseases". Tashkent 2022. p. 116
2. Ismailov S.I., Ubaidullaeva N.B. Treatment of thyroid dysfunction in pregnancy and postpartum period // Rukovodstvo po klinicheskoi praktike Endokrinologicheskogo Obshchestva Tashkent. - 2008.- 26 p.
3. Abashova, E.I. External genital endometriosis and hormonal lack of ovaries: avtoreferat.dis.... kand. Med. nauk / E.I. Abashova.-SPb., 1999. - 23s.
4. Anolyan A.N. "Function of the reproductive system and hormonal contraception in women with autoimmune thyroiditis" Journal of Gynecology #5. 2008 S 167169.
5. Adamyan, L.V. Genital endometriosis. Sovremennyy vzglyad na problemu / L.V. Adamyan, S.A. Gasparyan.- Stavropol: SGMA, 2004

6. Vorobeva, A.A. Immunology and allergology: uchebnoe posobie dlya vuzov / A.A. Vorobeva, A.S. Bykova, A.V. Karaulova. - M.: Prakticheskaya meditsina, 2006. 288 p
7. Blatt AJ, Nakamoto JM, Kaufman HW. National status of testing for hypothyroidism during pregnancy and postpartum // JI of Clinical Endocrinology and Metabolism. 2012. Vol. 97. R. 777-784.
8. Lechebnye aspekty estrogen-gestagenov i gestagenov kontratseptivov u zhenshin s autoimunnym thyroiditom / A.N. Mgeryan, V.N. Prilepskaya, E.A. Mejevitinova, A.V. Tagieva., E.S. Chernysheva // Mat i ditya: materialy X Ros. Nauch. forum. - M., 2009. - S. 362
9. Altomare M., La Vignera S., Acero P. et al. High prevalence of thyroid dysfunction in pregnant women // Journal of Endocrinological Investigation. 2013. Vol. 36. R. 407-411.
10. Stagnaro-Green D., Abalovich M., Alexander E. et al. Guidelines of the American Thyroid Association for the Diagnosis and Management of Thyroid Disease During Pregnancy and Postpartum // Thyroid. 2011. Vol. 21. doi: 10.1089/thy.2011.0087.
11. Li C., Shan Z., Mao J., Wang W. et al. Assessment of thyroid function during first trimester pregnancy: what is the rational upper limit of serum TSH during the first trimester in Chinese pregnant women? // J Clin Endocrinol Metab. 2014. Vol. 99. R. 7379.
12. Bestwick JP, John R., Maina A. et al. Thyroid stimulating hormone and free thyroxine in pregnancy: expressing concentrations as multiples of the median (MoMs) // Clin Chim Acta. 2014. Vol. 430. R. 33-37.
13. La'ulu SL, Roberts WL. Ethnic differences in first trimester thyroid reference intervals // Clin Chem. 2011. Vol. 57. R. 913-915.
14. Medici M., Korevaar T.I., Visser W.E. et al. Thyroid function in pregnancy: what is normal? // ClinChem. 2015. Vol. 61(5). P. 704-713.
15. Moon H.W., Chung H.J., Park C.M. et al. Establishment of trimester-specific reference intervals for thyroid hormones in Korean pregnant women // Ann LabMed.2015.Vol.35(2).P.198-204.
16. Springer D., Zima T., Limanova Z. Reference intervals in evaluation of maternal thyroid function during the first trimester of pregnancy // Eur J Endocrinol.2009.Vol.160.P.791-797.
17. Springer D., Zima T., Limanova Z. Reference intervals in evaluation of maternal thyroid function during the first trimester of pregnancy // Eur J Endocrinol.2009.Vol.160.P.791-797.