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STRUCTURE, FUNCTION, PATHOLOGICAL CHANGES OF ENDOCRINE GLANDS, PATHOGENESIS AND MORPHOLOGICAL BASIS OF DISEASES

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Abstract: The endocrine system is a network of glands without excretory ducts that secrete hormones directly into the bloodstream and regulate metabolism, growth, reproduction, and homeostasis.The endocrine system includes the pituitary gland, thyroid gland, parathyroid glands, adrenal glands, pancreas,and sex glands (gonads), as well as specialized tissues of complex organs that perform other function.For example,the islets of Langerhans of the pancreas, ovarian egg cells, and Leydig cells of the testes also produce hormones.There is also the APUD system,which unites endocrine cells of neuroectodermal origin that produce hormones of polypeptide or protein nature. These include the alpha cells of the islets of Langerhans that produce glucagon, the beta cells that produce insulin, and the parafollicular cells of the thyroid gland that produce calcitonin.Pathological changes are most often manifested as a result of autoimmune processes,neoplasms (tumors), or developmental defects, leading to excessive or insufficient hormone production.Morphological changes of the endocrine glands include atrophy, hyperplasia, hypertrophy, neoplastic nodules, inflammatory infiltration, as well as conditions such as fibrosis and necrosis.Endocrine changes play an important role in human pathology.They may be associated with primary damage to the endocrine glands or may occur in tumors developing outside the endocrine system.Endocrine changes that is ,endocrineopathy phenomena,when associated with tumors,are considered paraneoplastic syndromes.

The purpose of this article is to study and analyze the pathomorphological changes that occur as a result of the functions and pathological alterations of the endocrine glands.

Keywords: endocrine glands, hormones, endocrine system, homeostasis, target organs, huperthyroidism, hypothyroidism, necrosis, fibrosis.

INTRODUCTION

The endocrine system consists of several organs located in different places, closely related to each other, which ensure homeostasis. Signal transmission through molecules released into the extracellular space is divided into autocrine, paracrine and endocrine, depending on the distance over which these molecules act. In endocrine signaling, molecules called hormones act on target cells located far from the site of synthesis, hormones are usually transported from the site of synthesis to their targets by the bloodstream. In response to the action of a hormone, target tissues often secrete factors that reduce the activity of the gland that produces the stimulating hormone. This mechanism is called negative feedback. A number of conditions can disrupt

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endocrine function, such as impaired hormone synthesis or secretion, abnormal interactions between hormones and target organs, and abnormal target organ responses. Endocrine diseases are divided into: 1. disorders of hormone synthesis or secretion, accompanied by insufficient or excessive production or secretion of hormones, associated with biochemical and diseases leading to clinical manifestations; 2. neoplasms of the endocrine glands. Such neoplasms may be dysfunctional or accompanied by insufficient or excessive production of hormones. In the study of endocrine diseases, it is necessary to comprehensively evaluate morphological data, taking into account changes in hormone levels, their regulatory factors and other metabolites. Endocrine hormones are characterized by three main features: 1) their effect is directed to a specific target: hormones affect a relatively limited number of tissues (the "target tissues" of this hormone); 2) their effect is only specific, that is, specific: ("one hormone - one target tissue - the same effect"); 3) their effect is potent, that is, a very small amount of hormone is needed to produce a typical response.

Relevance of the problem. The structure, function of the endocrine glands and their pathological changes are one of the most important and relevant areas of modern medical science. Because the endocrine system controls the activity of almost all organs and systems of the body hormonally. Hormones play the role of the main regulator in metabolism, growth and development, reproductive function, stress adaptation, and ensuring the stability of the internal environment (homeostasis).

In recent years, the number of endocrine pathologies such as diabetes mellitus, thyroid diseases (hypothyroidism, hyperthyroidism, autoimmune thyroiditis), adrenal dysfunction, and disorders of the pituitary and gonadal functions has been increasing significantly. The study of the morphological basis of pathological changes in the endocrine glands is of particular importance. Because changes at the cellular and tissue level (atrophy, hyperplasia, dystrophic and inflammatory processes, autoimmune destruction, tumor transformations) determine the clinical manifestations of diseases. For example, the destruction of the follicular epithelium of the thyroid gland leads to a decrease in hormone synthesis, and the growth of pituitary cells in the form of adenomas leads to the development of many hormonal syndromes. In addition, a deep study of the pathogenesis of endocrine system pathologies serves as an important scientific basis for early diagnosis, differential diagnosis and the development of effective treatment methods in modern medicine. In particular, the identification of processes associated with immunological mechanisms, genetic mutations and disorders of neuroendocrine regulation is of great relevance today. Therefore, a comprehensive study of the structure, functional activity, pathological changes and morphological foundations of endocrine glands remains one of the important scientific and practical tasks of modern medicine. This allows for the prevention, early detection and effective treatment of endocrine diseases. It is the main scientific foundation for developing strategies.

Endocrine diseases are conditions in which the production of one or more hormones is impaired. The causes can be various: from autoimmune processes to heredity, from chronic stress to tumors.

The pituitary gland is located in the lower part of the brain, in the Turkish saddle at the base of the skull, and is connected to the brain through a special structure called the pituitary stalk.

Clinical manifestations of pituitary diseases:

Hyperpituitarism. It is observed with excessive secretion of tropic hormones. The causes may be pituitary adenoma, hyperplasia and carcinoma of the anterior pituitary gland, hormone production by tumors located outside the pituitary gland, and some lesions of the hypothalamus.

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Hypopituitarism. It is caused by a deficiency of tropic hormones. This can be caused by various destructive processes, such as ischemic injury, surgery, radiation exposure, inflammation, and non-functioning pituitary adenoma.

Lactotropic adenomas of the pituitary gland

Lactotropic adenomas (prolactinomas) are the most common type of hyperfunctional pituitary adenoma, accounting for 30% of all clinically significant pituitary adenomas. These tumors may be microadenomas or large, diffuse tumors with a pronounced mass effect. Microscopically, most lactotropic adenomas consist of weakly acidophilic or chromophobic cells (slightly granular lactotropic adenoma). Lactotropic adenomas are usually diagnosed in women aged 20-40 years, because hyperprolactinemia causes menstrual disorders, which is why they require medical attention. In contrast, hormonal changes may be minimal in men and older women.

Somatotrophic adenomas of the pituitary gland:

GH-secreting tumors are the second most common type of pituitary adenoma. Somatotrophic adenomas may be very large at diagnosis, as the increased GH secretion and associated symptoms may be minor. Histologically, adenomas composed exclusively of GH-producing cells are divided into 2 types: richly granular and poorly granular. Richly granular adenomas consist of monomorphic and acidophilic cells with a clear cytoplasmic immunoreactivity with antibodies to GH, as well as a perinuclear cytoplasmic immunoreactivity with antibodies to cytokeratins. Poorly granular variants consist of chromophobic cells with clear nuclear and cytological pleomorphism, as well as a focal weak immunoreactivity with antibodies to GH. Persistently elevated GH levels stimulate the secretion of IGF-1 (or somatomedin C) in the liver, which causes many of the clinical manifestations of the disease. If a child develops a somatotropic adenoma before the growth plates in the epiphyses of the long bones have closed, high GH (and IGF-1) levels lead to gigantism.

Gigantism is characterized by a general increase in body size and disproportionately long limbs. If GH levels increase after the growth plates have closed, acromegaly develops. This condition is characterized by a particularly pronounced increase in skin and soft tissue cells, an increase in the size of internal organs (thyroid, heart, liver, and adrenal glands), as well as an increase in the size of the facial skull, arms, and legs.

Hypersecretion of GH also leads to other diseases (gonadal dysfunction, diabetes, generalized muscle weakness, hypertension, arthritis, chronic heart failure) and increases the risk of developing gastrointestinal cancer. Diagnosis of hypersecretion of a pituitary tumor is based on the detection of high levels of IGF-1 concentrations. One of the most sensitive tests for diagnosing acromegaly is the suppression of growth hormone production during a glucose tolerance test. Treatment of pituitary adenomas can be surgical (resection) or drug therapy. Drug therapy uses somatostatin analogs.

Corticotropic tumors of the pituitary gland:

Corticotropic adenomas are usually microadenomas. These tumors are often basophilic, markedly granular, and sometimes chromophobic, weakly granular. Tumor cells of both types of adenomas stain with PAS, as they contain carbohydrates.

Hypofunction of the pituitary gland: adenohypophysis hormones are present in 3 different states: 1) when the pituitary gland is damaged and the secretory cells are destroyed; 2) when the pituitary stalks are damaged and the transfer of releasing factors from the hypothalamus to the pituitary gland is stopped; 3) when the hypothalamus itself is damaged and the production of releasing factors that control the activity of the adenohypophysis is reduced. The last 2 factors cause hyperprolactinemia due to the lack of inhibitors. When the adenohypophysis itself is

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damaged, a deficiency of two or three hormones occurs. Most often, the secretion of growth hormone, adrenocorticotropic hormone, and gonadotropin is reduced. However, there may be cases where only one hormone, for example, growth hormone, is deficient.

Pituitary hyperfunction: The most common cause of pituitary hyperfunction is pituitary adenomas, which have the property of enhancing the production of pituitary hormones. Adenomas are divided into 3 types:

1. Formed from lactotroph cells that produce prolactin
2. Formed from somatotroph cells that produce growth hormone
3. Formed from corticotroph cells that produce adrenocorticotropic hormone. All adenomas are monoclonal and produce only one hormone. Macroscopically, large adenomas are soft and have a reddish-brown color when cut. They may contain foci of necrosis (because the adenoma compresses the vessels supplying the pituitary with blood). Areas of hemorrhage are also found (pituitary apoplexy). The interstitial tissue of the pituitary gland is compressed by the tumor and atrophies. When examined under a microscope, the adenoma consists of monomorphic epithelial cells. These cells are arranged in nests or drawers, sometimes forming glandular or papillary structures. In terms of structure, they resemble the epithelial cells of the pituitary gland. Adenoma cells are round, ovoid, or polygonal in shape and have granules in their cytoplasm. The nucleus is smaller, located in the center of the cell or off-center.

Gigantism usually begins in puberty or prepuberty. In this case, the body and limbs grow excessively, exceeding the norms for a person of this gender for their age, and an imbalance occurs in the bone skeleton. Internal organs also enlarge, arrhythmias, and neuromuscular changes begin. Patients develop diabetes insipidus and cognitive decline.

Acromegaly is seen in middle-aged people. The head, eyebrows and cheekbones, occipital and temporal lobes, and lower jaw also enlarge. Kyphosis occurs in the thoracic spine or lordosis in the lumbar spine. Patients develop impaired glucose absorption, diabetes mellitus, gynecomastia, osteoporosis, and hypertension in men. Such changes are also observed in hypothalamic tumors, medullary cancer, thyroid carcinoid, and tumors of the islets of Langerhans, as hypersecretion of releasing factors that enhance the production of somatotroph hormone occurs.

Pituitary Cushing's disease is associated with the formation of corticotrophic adenomas, occurs in both sexes, and is more common in women aged 20-40. It is characterized by the accumulation of fat cells under the skin of the face, neck, and upper half of the body, causing disproportionate fat deposition. The skin of the chest, abdomen, thighs, and buttocks stretches, and red lines (striae) appear. Hirsutism, hypertension, and mental changes are also observed.

Hypofunction of the pituitary gland is a syndrome of diseases.

Shikhen syndrome - pituitary necrosis that begins after childbirth - also causes pituitary insufficiency. During pregnancy, the pituitary gland doubles in size, which leads to disruption of the blood supply to this gland due to compression of the vessels. If there is bleeding during childbirth or during the postpartum period, posthemorrhagic anemia or hemorrhagic shock may occur. Under such conditions, necrosis may begin in the adenohypophysis. If pituitary insufficiency is observed after childbirth, the first symptoms are involution of the mammary gland, failure to produce milk, and failure to lactate. Later, the menstrual cycle is disrupted. As the process progresses, atrophy of the internal organs, along with the gonads, and cachexia (SIMMONDS cachexia) become more severe. Pituitary infarction is less common in sickle cell anemia, vasculitis, and intravascular coagulation (IVC) syndrome. Large necrosis and hemorrhages in the pituitary gland can cause a sudden rise in temperature and convulsions.

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The syndrome of the Turkish saddle is a rare pathology, which can be detected only by pneumoencephalography in patients with reduced pituitary secretory activity. The disappearance of the pituitary gland can be explained by diffuse necrosis, the formation of fibrous tissue after purulent inflammation, infarction, hypophysectomy (hypophysectomy performed using radiation or surgically).

Pathological processes occurring in the thyroid gland are very diverse. Three main pathological processes are clinically most striking:

1) Increased size and weight of the thyroid gland (goiter, stroma);
2) Hyperthyroidism, characterized by excessive production of thyroxine hormone - Graves' disease or Graves' goiter;
3) Decreased thyroid activity - hypothyroidism. In hypothyroidism, the thyroid gland may become completely inactive (athyroid) or its function may decrease (dysthyroid);

Thyroiditis: a pathological process characterized by infiltration of the thyroid gland with leukocytes or the formation of fibrosis, and both processes in the gland sometimes occur together. In clinical practice, three types of thyroiditis are most important:

1. Hashimoto's thyroiditis, subacute granulomatous, and chronic painless thyroiditis.
2. Fibrous RIDEL thyroiditis is very rare, in which the gland parenchyma is covered with dense fibrous tissue. This process is considered an idiopathic process. Riedel's thyroiditis sometimes proceeds with mediastinal and retroperitoneal fibrosis.

Hashimoto's thyroiditis (lymphomatous stroma) is an autoimmune disease. It can occur in people of any age. It is observed 10-12 times more often in women. Its characteristic feature is an increase in the size of the thyroid gland and a decrease in its function. The onset of the disease begins with a euthyroid state. During the exacerbation of the disease, hyperthyroidism, which is clinically somewhat similar to Graves' goiter, may begin. However, while NLA-DR5 increases in Hashimoto's goiter, NLA-DR3 increases in Graves' goiter. Immunoglobulins in patients with Hashimoto's thyroiditis cause an increase in the size of the thyroid gland and increase the secretion of thyroxine. It is also important that people with the NLA-DR5 genotype have an organ-specific defect in the functional activity of T-suppressors.

Chronic thyroiditis is characterized by transient thyrotoxicosis and lymphocytic infiltration of the gland tissue, the cause of which is unknown. The thyroid gland may be of normal size or moderately enlarged. Both lobes are hypertrophied to the same extent. The disease is initially asymptomatic, then thyrotoxicosis begins and the gland enlarges. The thyrotoxic phase usually lasts several months (several years), alternating with euthyroidism. The thyroid gland can remain enlarged for a long time. Thyroid hormones. They are divided into malignant and benign tumors. They can occur in the form of solitary or nodular structures. Among benign tumors, adenoma is more common, fibroma, teratoma, paraganglioma, hemangioma, lipoma, myoma are less common. They are more often observed in endemic goiter foci and in populations exposed to radioactive radiation. The most common type of thyroid tumor is nodular adenoma. Thyroid cancer is extremely rare.

Adenoma is a benign tumor arising from the epithelium of the follicles and can occur in people of any age. Solitary adenomas are most often found in non-endemic regions. The nodular form of the goiter is characterized by the presence of many nodes in the gland.

Thyroid cancer is the most common of all malignant tumors, and is mainly divided into 2 categories:

1) Well-differentiated carcinomas: papillary and follicular carcinoma;

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2)Poorly differentiated carcinomas, which include medullary and undifferentiated carcinoma.

Thyroid cancer is 2 times more common in women, and can occur at any age. However, it should be noted that papillary and follicular cancer mainly occur in people under 30 years of age, especially in people who have previously been exposed to radiation on the head and neck. The role of generalized radiation in the development of thyroid cancer is of great importance, as evidenced by the sad experience of Hiroshima, Nagasaki and Chernobyl. In recent years, mutated ras genes have been found in some thyroid tumors. In addition, the expression of the c-erb B and c-erb B/2 oncogenes is observed. Interestingly, the c-erb B oncogene encodes epidermal growth factor receptors. Anaplastic cancer differs from all tumors in its severe course and malignancy. It occurs in people aged 60-80 years. Patients die within 2 years. It appears as a large structure that rapidly grows into the capsule of the gland. When examined under a microscope, it is revealed that the tumor is completely undifferentiated and consists of small, hairy cells similar to sarcoma cells. Multinucleated giant cells and multilayered squamous epithelial cells are found. It metastasizes to many places and early.

Graves' disease describes a disease characterized by an enlargement of the thyroid gland. This disease is the most common cause of hyperthyroidism;

Hyperthyroidism due to diffuse hyperfunctional enlargement of the thyroid gland

Damage to the infiltrative ophthalmopathy with the development of exophthalmos

Focal infiltrative dermopathy, sometimes called pretibial myxedema, is observed in a small number of patients. Graves' disease (hyperthyroidism) and Hashimoto's thyroiditis (hypothyroidism) are autoimmune diseases of the same thyroid gland, with completely opposite manifestations. Genetic factors play an important role in the development of Graves' disease: the concordance between monozygotic twins reaches 30-40%, while in dizygotic twins it does not exceed 5%. The genetic predisposition to Graves' disease is due to polymorphisms in immune-functional genes, such as the CTLAA and PTPN22 genes, and is associated with the HLA-DR3 allele. Graves' disease is characterized by impaired tolerance to thyroid autoantigens, mainly TSH receptors.

Congenital anomalies. The most common and clinically significant congenital anomalies of the thyroid gland are the thyroglossal duct and its cyst. In addition, a fistula tract can form from the rudimentary remnant of the thyroid gland during the tubular stage of development. Some areas of this duct may be absent, forming small closed segments that later develop into cysts. These cysts may remain clinically asymptomatic until adulthood. The segments of the thyroglossal duct and the cysts located above in the scrotal region are covered with stratified squamous epithelium, similar to the epithelium covering the cecum of the tongue. Characteristically, there is a pronounced lymphocytic infiltration under the covering epithelium. Secondary infection can lead to abscess formation in these formations.

Adrenal glands. The adrenal glands are a pair of endocrine organs consisting of a cortex and a medulla, which differ in embryogenesis, structure and function. Beneath the adrenal capsule is a narrow layer called the zona glomerulosa, which borders the medulla with a similar narrow zona reticularis. The adrenal gland consists of chromaffin cells that synthesize and secrete catecholamines.

Hyperfunction of the adrenal cortex

Since there are three main types of corticosteroids produced by the adrenal cortex, there are three syndromes of hyperfunction:

1)Hypercortisolism or Cushing's syndrome, characterized by excess cortisol;

2)Hyperaldosteronism (excessive secretion of aldosterone);

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3) Adrenal cortex syndrome, caused by excess cortisol;

Hypercortisolism. Hypercortisolism is caused by any condition that is accompanied by an increase in glucocorticosteroid levels. The adrenal form of hypercortisolism is called Cushing's syndrome. The causes of Cushing's syndrome can be exogenous and endogenous. Endogenous causes can be divided into ACTH-dependent and non-dependent. Pituitary adenomas that secrete ACTH are the cause of 70% of cases of endogenous hypercortisolism. In most cases, the cause of hypercortisolism is a microadenoma of the pituitary gland that secretes ACTH. Less often, areas of hyperplasia of corticotroph cells without adenoma formation are found in the anterior pituitary gland. Hyperplasia of the adrenal cortex leads to the development of hypercortisolism.

The term hyperaldosteronism is characterized by prolonged excessive secretion of aldosterone, which can be primary or secondary, that is, caused by causes not related to the adrenal cortex. Suppression of ACTH synthesis by hyperaldosterone is a rare cause of primary hyperaldosteronism. In this case, the fusion of the CYP11B1 (tenal 11B - hydroxylase) and CYP1182 (aldosterone synthase) genes results in a hybrid gene that causes the continuous production of hybrid steroids in addition to cortisol and aldosterone. In secondary hyperaldosteronism, aldosterone is secreted in response to activation of the renin-angiotensin-aldosterone system. Secondary hyperaldosteronism is characterized by increased plasma renin levels and occurs in the following cases:

- reduced renal perfusion (arteriolar sclerosis, renal artery stenosis)

Purpose of the study: The purpose of the presentation of this article is to provide a deep analysis of the structure and functional properties of the endocrine glands, to shed light on their role and importance in maintaining homeostasis in the body on a scientific basis. Also, special attention is paid to studying the pathogenesis of endocrine system diseases, that is, the mechanisms of disease development based on modern scientific views, and to determining the morphological basis of these processes (changes at the tissue and cellular levels).

The article reveals the relationship of pathological changes occurring in the endocrine glands at the molecular, cellular and systemic levels, and explains the formation of clinical symptoms based on them. In addition, modern diagnostic methods, early detection of hormonal imbalances and prevention of their complications are also covered.

The presentation also analyzes the structural basis of morphological changes occurring in the endocrine glands - hypertrophy, hyperplasia, atrophy, dysplasia and neoplastic processes in an inextricable link with their functional disorders. Based on the correlation of these changes with clinical symptoms, a morphofunctional model of diseases is formed.

In addition, modern scientific approaches - molecular endocrinology, the theory of receptor-ligand interaction, and the role of oxidative stress and inflammatory factors in endocrine pathology are also conceptually covered.

The main goal of this presentation is to integrate theoretical knowledge with practical medicine, to create a scientific basis for improving early diagnosis and effective treatment strategies through a deep understanding of the mechanisms of development of endocrine diseases. In this work, the consequences of imbalances in the activity of the hypothalamic-pituitary-endocrine axes at the systemic organism level and their relationship with clinical manifestations are substantiated on the basis of modern scientific views.

In addition, the results of the study are of significant theoretical and practical importance in the early detection, prognosis and development of treatment strategies for endocrine pathologies based on an individual approach.

CONCLUSION. The endocrine system is a complex and multi-level regulatory system that provides homeostasis in the body, and plays an important role in ensuring the stability of the

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internal environment, metabolism, growth and development processes in the body. Pathological changes occurring in these glands are inextricably linked with morphological disorders at the cellular and tissue levels, which determine the mechanisms of disease development.

The pathogenesis of endocrine pathologies is multifactorial, in which genetic, immunological and environmental factors play an important role. In general, a comprehensive study of the structure, function and pathological changes of endocrine glands is of great importance in medical practice for the early detection, prevention and development of effective treatment strategies for diseases.

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