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Thyroid diseases as an important medical and social problem

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Abstract: In our article we analyzed literary sources on thyroid diseases.

Keywords: Population, gland, form, complications, death.

Introduction: The name of the thyroid gland (English: thyroid, German: Schilddruse) is difficult to associate with its shape, since in fact it consists of two lobes connected by an isthmus and bears little resemblance to a shield.

Thomas Wharton (1614-1673), who followed Vesalius in describing this gland in humans, gave it this name, apparently based on its close location to one of the cartilages of the trachea, which in shape really does resemble a Greek shield (thyreos).

The thyroid gland produces hormones that regulate many physiological and biochemical processes in the body in almost all tissues. Therefore, it is considered the "first violin" in the endocrine ensemble, controlled by the higher centers of the brain, the hypothalamus and the pituitary gland.

In addition to iodinated thyronines (actually thyroid hormones), this gland in humans also produces the polypeptide hormone calcitonin, the synthesis and mechanism of action of which are not discussed in this review.

This review presents current data on the study of thyroid gland regulation mechanisms, as well as the biosynthesis, secretion and mechanism of action of thyroid hormones. Particular attention is paid to the possibility of explaining the effects of thyroid hormones in vivo from the standpoint of the interaction of these hormones with their intra- and extranuclear cellular

receptors in vitro.

Thyroid hormones determine the development of the brain and the development of all tissues where protein synthesis occurs. This is most important for the growing organism of the fetus and infant. In adults, they also play the role of conductor of the functions of almost every organ system.

Thyroid hormones must be constantly available to perform these functions. To maintain their availability, the thyroid gland contains large reserves of thyroid hormones.

Moreover, the synthesis and secretion of thyroid hormones are maintained within narrow limits by a regulatory mechanism that is very sensitive to small changes in circulating hormone concentrations.

Thyroid gland diseases (TG) are an important medical and social problem worldwide due to the high prevalence of the pathology, which affects the most able-bodied group of the population and children. And if the medical issues of the problem are studied intensively, its general biological aspects are poorly considered in the literature.

One of the pressing problems of modern thyroidology is goiter endemicity. And it should be noted that due to the destruction of the system of preventive measures in the 90s, our country has seen an increase in the number of regions with this pathology.

The main form of goiter in goiter endemic regions is diffuse nontoxic goiter, which is characterized by diffuse enlargement of the gland, and clinically – a euthyroid state. The cause of goiter formation in goiter endemic regions is traditionally considered to be iodine deficiency in the environment.

The structural unit of the thyroid gland is the follicle. Each follicle is surrounded by a basal membrane. Outside the basal membrane is a dense network of blood capillaries. It should be noted that in terms of vascularization, i.e. the amount of blood that passes through the organ per unit of time, the thyroid gland is in first place, which has its own functional significance.

The follicle wall contains: A cells (follicular epithelial cells) that synthesize thyroid hormones, B cells (modified A cells) that appear in some diseases, and C cells (parafollicular cells) that synthesize calcitonin. (poorly differentiated cells)

However, some authors also distinguish the source of basal extra- and intrafollicular proliferation, and therefore, as a source of formation of the corresponding follicles. These variants of formation of new follicles, apparently, are manifested in cases of thyroid pathology, accompanied by thyrotoxicosis syndrome.

But there is a slightly different opinion. In particular, a number of researchers believe that B-cells are mast cells and they, together with C cells, provide homeostasis of biogenic amines in the thyroid gland. The authors believe that they can perform the function of carriers of catecholamines and serotonin to the functionally active zones of the organ.

Biogenic amines stimulate A-cells to endocytosis of colloid and release of thyroid hormones into the blood, and they also regulate the function of the gland's microcirculation. And, according to the authors, when the number of C-cells decreases, the ability of A-cells to produce thyroid hormones decreases, as well as to absorb inorganic iodine, which is necessary for hormone synthesis.

It is believed that iodine is not only a substrate for the synthesis of thyroid hormones, but also a factor regulating the function of the thyroid gland, since the proliferation of thyrocytes depends on the quantitative content of its atoms. Thyroid hormones have a relatively simple chemical formula - amino acid tyrosine + iodine atoms.

The process of their synthesis has features related to the features of the thyroid gland structure. The latter consist in the fact that the structural unit of the organ - follicles - have a cavity. It, to some extent, resembles the external environment for the organ, although this is a storage place, i.e. temporary location, of ready hormones.

And as the authors believe, the thyroid gland is the only endocrine organ where there is a two-way movement of products during the synthesis and secretion of hormones into the blood. So, the main components involved in the biosynthesis of thyroid hormones are: thyroglobulin protein, iodine atoms, and the enzyme thyroid peroxidase (TPO).

Thyroglobulin is a glycoprotein. It is synthesized in the cisterns of the endoplasmic reticulum, and then “packed” into the secretory vesicles of the Golgi apparatus and transferred to the apical part of the cell, which faces the colloid, then the contents of the vesicles are released into the follicle cavity. TPO is a transmembrane glycoprotein, an enzyme of the oxidoreductase class with a molecular weight of 103 kDa.

The polypeptide chain of TPO consists of 933 amino acid residues. The protein is a dimer, the parts of which are linked by disulfide bonds. Iodine in inorganic compounds enters thyrocytes with the participation of sodium pumps. Thyroglobulin molecules are formed in the cisterns of the rough endoplasmic reticulum.

The synthesized thyroglobulin molecules are

concentrated in the vesicles of the Golgi apparatus, and then, breaking away, merge with the apical membrane. Subsequently, the protein, by exocytosis, enters the colloid. It should be noted that they also contain a system for generating hydrogen peroxide.

Subsequently, hydrogen peroxide crosses the apical membrane and becomes available to TPO in the lumen of the follicle. According to the same scheme, TPO molecules are synthesized in the cisterns of the rough endoplasmic reticulum, which also condense in the vesicles of the Golgi apparatus.

The subsequent fate of the contents of the vesicles, in particular TPO, is somewhat different - during their exocytosis, the molecules of enzyme maintain a connection with the apical membrane in the zone of the thyrocyte microvilli. Apparently, in this zone, with the participation of an oxidizing agent, the degradation of iodine-containing compounds occurs with the formation of reactive iodine atoms.

According to the authors, the protein thyroglobulin is iodinated with the participation of TPO in the lumen of the follicle, which raises some doubts, which will be discussed in the section "Discussion of the results of the study". One way or another, thyroglobulin finally matures (iodinated) in the colloid and remains in it until the moment when there is a need for additional supply of hormones to the blood.

The next stage of hormone formation is proteolysis in thyroglobulin molecules. At the same time, thin pseudopodia appear on the surface of follicular cells, which delimit small amounts of colloid; at the same time, colloidal droplets pass into the cytoplasm via the phagocytic mechanism.

The membrane-enclosed droplets in the cytoplasm combine with lysosomes, lysosome enzymes digest them, and then thyroid hormones are separated from the droplets and released. As indicated, thyroid globulin (thyroglobulin) is a glycoprotein secreted by thyrocytes into the lumen of the follicles; it does not normally enter the internal environment (circulating fluids).

Therefore, there is a very low probability that the organism will be immunologically tolerant to the glycoprotein, colloid. Consequently, T-lymphocytes targeting thyroglobulin are preserved (not eliminated during embryonic development), which is one of the factors in the development of autoimmune thyroiditis.

The authors note that changes in thyroglobulin metabolism are biphasic. At the first stage, iodine organification in the thyroid gland increases, as evidenced by an increase in the concentration of TPO in the bloodstream, and then thyroglobulin proteolysis increases, which confirms an increase in T4 concentration against the background of an unchanged concentration of thyroglobulin in the blood serum.

This position should also be considered controversial, since this thyroid protein, as noted, has antigenic properties and should not enter circulation under normal conditions. TPO, which is part of the complex of the so-called microsomal fraction, also has antigenic properties, therefore, when determining microsomal antibodies, antibodies to TPO are actually determined.

And the currently widespread determination of the titer of autoantibodies to TPO is used as a marker of thyroid diseases caused by autoimmune processes. Thyroid hormones, triiodothyronine (T3) and thyroxine (T4), are important regulators of the cardiovascular system.

Thyroid hormones, triiodothyronine (T3) and thyroxine (T4), are important regulators of the cardiovascular system.

Changes in the thyroid status of the human body are often accompanied by disruption of the functioning of the heart and blood vessels and can serve as a risk factor for the development of cardiovascular diseases, which remain one of the leading causes of death in the world.

In this regard, the study of the mechanisms of the influence of thyroid hormones on the cardiovascular system is of high practical importance. It should be noted that at the moment, the effect of thyroid hormones on blood vessels, in particular on resistive arteries, the tone of which determines the level of systemic arterial pressure, remains poorly understood.

Thyroid hormones can cause a decrease in vascular tone by acting through two mechanisms: genomic and non-genomic.

In the classical genomic action, thyroid hormones bind to nuclear receptors, which act as ligand-dependent transcription factors that alter the expression of target genes.

The genomic effects of thyroid hormones are manifested an hour or more after the interaction of the hormone with the receptor and are long-term. Due to the fact that nuclear receptors have a higher affinity for T3 than for T4, T3 is considered the key form of thyroid hormones in their genomic action. Due to its higher content in the blood, T4 can also exert genomic influence, but at the same time acts as a prohormone capable of converting into the more active T3 in accordance with tissue needs under the action of deiodinase enzymes types 1 and 2 (D1 and D2), which catalyze 5'-deiodination.

The main deiodinase that carries out 5'-deiodination of T3 in arteries is deiodinase type 2 (D2), it is expressed in both endothelial and smooth muscle cells. D2 provides

local synthesis of T3, therefore the intensity of the genomic action of thyroid hormones on vessels may depend on the level of expression and activity of D2 in them.

The physiologically significant role of D2 in blood vessels is confirmed by the connection between the inactivating polymorphism of the gene of this enzyme and a higher risk of developing arterial hypertension in people with euthyroid status. It is known that the level of D2 mRNA in tissues can depend on the content of thyroid hormones in the blood, as well as the intensity of sympathetic influences and other physiologically significant stimuli.

However, the mechanisms of regulation of D2 expression in the vessel wall, as well as the effect of the intracellular T3 produced by it on the vasomotor reactions of resistive arteries, have been practically not studied.

The non-genomic mechanism of action of thyroid hormones includes all effects that initiate receptors localized outside the nucleus.

Non-genomic action of thyroid hormones is not necessarily related to transcription, so it manifests itself relatively quickly, within a few minutes. Non-genomic action can be initiated by both T3 and T4 due to the wide variety of receptors that mediate it.

Thyroid hormones are capable of causing a rapid decrease in arterial tone, but the mechanisms of such an effect have been little studied. It should be noted that non-genomic influence of hormones, along with genomic, can contribute to the decrease in peripheral vascular resistance in hyperthyroidism.

Thus, based on the literature analysis, we can say about the relevance of this problem in medicine in general.

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