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Morphological Characteristics of the Thyroid Gland in Polypharmacy with Anti-Inflammatory Drugs

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Abstract: Thyroid gland pathology is considered a marker of environmental distress. The most significant morphophysiological structure of the thyroid gland is a tissue microregion that combines a group of follicles and interfollicular space with an autonomous system of blood and lymph circulation. It is the structures of the tissue microregion that suffer the most under the action of pathogenic factors on the thyroid gland, diminishing its role in facilitating morphological and metabolic changes in tissues and organs [Borodin Yu.I., et al., 2018]. However, the morphological and morphometric changes in the thyroid gland during polypharmacy with anti-inflammatory drugs are poorly understood. This article presents a review of the literature on structural changes in the thyroid gland during polypharmacy with anti-inflammatory drugs.

Keywords: polypharmacy, thyroid gland, morphology, anti-inflammatory drugs.

Relevance

According to the World Health Organization (WHO), thyroid diseases rank second among endocrine disorders after diabetes mellitus. Statistics show that up to one-third of the world's population suffers from thyroid disorders. More than 740 million people worldwide have endemic goiter or suffer from other thyroid pathologies; 1.5 billion people face the risk of developing iodine deficiency diseases. Moreover, according to statistics, the incidence of thyroid diseases globally is increasing by 5% annually. In recent decades, researchers have been actively studying the functional state of the thyroid gland in patients with various conditions. It is known that thyroid hormones regulate the state of all organs and systems in the human body, primarily the processes of cell growth, maturation, and differentiation. Thyroid morphology is receiving significant attention due to the increasing prevalence of endocrine pathologies worldwide. Thyroid pathology ranks second after diabetes mellitus and is considered a marker of environmental degradation. The most significant morphophysiological structure of the thyroid gland is the tissue microdistrict, which encompasses a group of follicles and the interfollicular space with an autonomous system of blood and lymph circulation. It is precisely the structures of the tissue microdistrict that suffer most when pathogenic factors affect the thyroid gland, reducing its role in facilitating morphological and metabolic changes in tissues and organs. Essentially, the thyroid parenchyma is formed by a system of thyrocytes, among which two main types are distinguished - follicular and interfollicular cells. The former create follicles capable of extracellular accumulation of hormonally active substances. The latter participate in the proliferation of the thyroid parenchyma, forming interfollicular islands between the follicles. The morphogenetic potential of stromal-parenchymal relationships is determined by the ratio of follicular epithelial tissue, colloid, and interstitium. The importance of the thyroid gland for the

body's vital functions cannot be overstated. The morphogenetic potentials of stromal-parenchymal relationships are determined by the ratio of follicular epithelial tissue, colloid, and interstitium.

In addition to thyrocytes - the main cellular population comprising the follicular compartment of the gland - it contains the second largest cellular group - calcitonin-producing cells (parafollicular or C-cells) [Solyannikova D.R., Bryukhin G.V., 2009]. They have a neurogenic origin and belong to the so-called APUD system [Smirnova T.S., 2009], which represents cell populations scattered in various organs and producing diverse biologically active substances, considered as a diffuse neuroendocrine system.

Parafollicular cells are located in small groups within the thyroid gland interstitium and/or lie on the basal membrane between thyrocytes (intraepithelially), but never border the follicle lumen. Their highest concentration is found in the central sections of each thyroid lobe, known as the "C-cell region." Parafollicular cells constitute no more than 1% of the thyroid gland epithelium. They are 2-3 times larger than thyrocytes, polygonal or slightly elongated in shape, with larger and lighter nuclei containing 1-2 dense nucleoli and pale cytoplasm with small argyrophilic granules [Volkov V. P., 2014]. The term "polypragmasia" (from poly - many and pragma - object, thing) refers to the simultaneous and often unjustified prescription of multiple medications or therapeutic procedures [E.A. Panova, et al., 2019]. Polypragmasia is essentially pharmaceutical pressure exerted on the patient due to an irrational comprehensive approach. In outpatient and inpatient settings, patients are frequently prescribed more than two medications simultaneously. Moreover, doctors don't always know what the patient is actually taking and in what doses, and non-compliance often occurs. Polypharmacotherapy can result not only from numerous comorbidities and conditions in the patient but also from incorrect medication choices, when the patient takes drugs with similar effects, contradictory actions, or unnecessary medications. There may be insufficient or distorted effects of prescribed drugs due to metabolic changes in the aging body. This often leads to incorrect adjustments in treatment strategy, increasing the number of medications or replacing them with stronger ones. The consequences of polypragmasia include reduced or absent treatment effects, unwanted side effects, frequent hospitalizations, and high costs for both patients and the healthcare system. The scientific medical community offers evidence-based methods to combat polypragmasia through various analytical algorithms for pharmacotherapy prescriptions. These include the medication appropriateness index, Beers criteria (American Geriatric Society, 2003, 2012), STOPP/START criteria (UK National Health Service recommendations, 2013, 2015), FORTA (Germany, 2011), and PINCER criteria (UK, 2012) [Guthrie B., Yu N., Murphy D., 2015]. Unfortunately, the frequency and consequences of irrational polypragmasia in outpatient clinics in our country remain insufficiently studied [E.A. Panova, 2019]. Thyroid diseases are among the most common types of endocrine pathology, caused by many factors, including iodine deficiency, elevated radiation exposure, unfavorable environmental conditions, and psycho-emotional stress. Acute and chronic stress can disrupt thyroid hormone secretion and significantly alter the gland's morphology, causing changes of varying severity and direction.

Pharmacological effects on the immune system often lead to the development of undesirable phenomena such as autoimmune diseases. Among all organs of the endocrine system, the thyroid gland is most frequently affected, as its embryonic development features predispose it to both spontaneous and induced lesions under various autoimmune influences. The term "polypragmasia" is often used in medical literature; however, there is no universally accepted definition. In Russian literature sources, polypragmasia is defined as the simultaneous prescription of a large number of medications, including their unjustified use. In foreign literature, the term "polypharmacy" (from Greek poly- and pharmacy - medicine) is used. Other literature sources provide a qualitative definition of polypragmasia as prescribing more drugs to a patient than the clinical situation requires, and a quantitative definition as prescribing 5 or more drugs to a patient. The reason for simultaneously prescribing several medications may be the presence of comorbidities (multimorbidity), drug availability, as well as clinical

recommendations, guidelines from professional medical societies, and treatment standards that in some cases contain recommendations for complex therapy with more than 5 drugs for a single indication, the effectiveness of which corresponds to high levels of evidence. Literature analysis shows that today, combating polypragmasia with anti-inflammatory drugs is one of the important tasks in providing medical care to patients of any age. This highlights the need to develop strategies that improve the quality of medical care and reduce adverse drug reactions [Shekunova E.V., Kovaleva M.A., et al. 2020 Annuar Fazalda, Adam Quraisiah, Mohd Fahami Nur Azlina. 2018, Arthur J. Kast, Natalie A. Terry, Gaary D. Albenberg, 2019]. At the end of the 20th century, P.J. Davis et al. discovered and subsequently studied non-genomic mechanisms of thyroid hormone action, initiated by plasma membrane receptors for T3 and T4 located on the αVβ3 integrin [Davis P.J., Glinsky G.V., Lin H.Y. et al., 2015]. This integrin is expressed on the surface of leukocytes, platelets, and epithelial and endothelial cells, facilitating interaction between cells and leukocytes with biological surfaces. Non-genomic mechanisms include the stimulation of thyroid hormones without involving the transcription of mitogen-activated protein kinase, phosphatidylinositol-3-kinase, and serine-threonine kinase genes, thereby contributing to tumor progression: angiogenesis, cell proliferation, and cell migration. Additionally, iodothyronines, in a dose-dependent manner, can stimulate the expression of tissue-specific pro-inflammatory genes, providing a systemic pro-inflammatory effect, which leads to an immunopathological process at the tissue and organ level. Considering the systemic pro-inflammatory effect of thyroid hormones, it is suggested that prolonged hyperthyroidism can contribute to the occurrence of a chronic inflammatory reaction, making cells more susceptible to malignancy [Glushakov R.I., Vlasyeva O.V., Sobolev I.V. et al. 2015]. Cytokine-induced thyroiditis (in 50-70% of cases proceeding as a destructive variant) and thyroid damage when using immune checkpoint inhibitors (with CTLA-4 + PD-1/PD-L1 combined treatment regimen leading to an increase in hypothyroidism frequency up to 20%) are known. Some researchers see an effect on immune processes even in iatrogenic thyroid pathologies such as lithium-induced gland dysfunction. Authors describing these lesions note that the predisposition to autoimmune thyroid lesions or the nature of the disease being treated (lithium in bipolar affective disorders; interferon α in hepatitis C) also involves autoimmune mechanisms [Melnichenko G.A., Glibka A.A., Demicheva O.Yu., 2019]. Conclusion From the above literature review, it becomes clear that research on polypragmasia and its impact on the endocrine system, especially the thyroid gland, is insufficient. Among the available data, there are some discrepancies that require further morphological and morphometric studies.

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