PATHOPHYSIOLOGICAL MECHANISMS AND CLINICAL SIGNIFICANCE OF HYPOTHYROIDISM

Sharofiddinov Javlon Komoliddinovich
Ikromova Dilnura Dilmurodovna
Asia International University, Bukhara, Uzbekistan
Email: sharofiddinovjavlon180@gmail.com
di9761477@gmail.com
mobile phone number: +998991582079
+998930081382

Abstract: Hypothyroidism is a common endocrine disorder characterized by a deficiency of thyroid hormones. According to current data, overt hypothyroidism occurs in approximately 0.3–5% of the general population, while subclinical hypothyroidism is found in about 4–10%, reaching up to 12% in some populations. It is observed more frequently among women and elderly individuals.This condition affects the body by disrupting metabolism, cardiovascular function, nervous system activity, digestive processes, and reproductive function. Hypothyroidism often develops slowly and insidiously, which may delay its early diagnosis. If not identified and treated in time, it can lead to serious complications such as myxedema, infertility, atherosclerosis, heart failure, and cognitive impairment. Therefore, studying the pathophysiological mechanisms and clinical significance of hypothyroidism is of great importance in modern medicine.

Keywords: Hypothyroidism, thyroid gland, thyroxine, myxedema, hypometabolism, basal metabolic rate (BMR), glycosaminoglycans, TSH (thyroid-stimulating hormone), cardiohemodynamics, levothyroxine.

INTRODUCTION

Hypothyroidism is a systemic clinical-pathophysiological syndrome characterized by a deficiency of thyroid hormones (thyroxine – T_4 and triiodothyronine – T_3) or a reduction in their biological effect at the cellular receptor level. In modern endocrinology, this condition is interpreted as a "metabolic crisis" involving the deceleration of all types of metabolism and a disruption of organizational homeostasis.

ETIOLOGY AND CLASSIFICATION

From a pathophysiological perspective, hypothyroidism is classified based on the level of the primary lesion:

Primary Hypothyroidism (Thyrogenous): Destruction of the thyroid parenchyma (Hashimoto's thyroiditis, subtotal thyroidectomy, Radioiodine-131 therapy) or impaired hormone biosynthesis due to iodine deficiency.

Secondary Hypothyroidism (Pituitary): Deficiency in the secretion of Thyroid-Stimulating Hormone (TSH) by the adenohypophysis (Simmonds-Sheehan syndrome, pituitary adenoma).

Tertiary Hypothyroidism (Hypothalamic): Decreased synthesis of Thyrotropin-Releasing Hormone (TRH) in the paraventricular nuclei of the hypothalamus.

PATHOPHYSIOLOGICAL MECHANISMS

1. Deceleration of Energetic and Basal Metabolism

Thyroid hormones bind to nuclear receptors to regulate mitochondrial biogenesis and Na^+/K^+-ATPase activity. Their deficiency leads to a slowdown in redox processes and a decrease in ATP synthesis. This manifests clinically as hypothermia and generalized adynamia.

2. Glycosaminoglycan Metabolism and Myxedema

The most pathognomonic sign of hypothyroidism is myxedematous edema. In the absence of sufficient hormones, glycosaminoglycans (hyaluronic acid and chondroitin sulfate) accumulate in the dermis and interstitial spaces. These substances are highly hydrophilic and bind large amounts of water, resulting in "non-pitting" edema (hard edema that does not leave an indentation when pressed).

3. Dysfunction in Lipid and Carbohydrate Metabolism

Dyslipidemia: Due to the inhibition of lipolysis and decreased activity of Low-Density Lipoprotein (LDL) receptors, serum cholesterol levels rise sharply, creating a predisposition for accelerated atherosclerosis. Hypoglycemic Tendency: Slowed glucose absorption from the intestines and inhibited glycogenolysis alter insulin sensitivity.

CLINICAL SIGNIFICANCE OF SYSTEMIC DISORDERS

Cardiohemodynamics: Bradycardia, decreased myocardial contractility (Negative\ Inotropic\ Effect), and reduced cardiac output. Myxedematous cardiomyopathy is often accompanied by transudate accumulation in the pericardial cavity. Hematological Changes: Chronic hypochromic anemia develops due to the loss of hormonal stimulation of erythropoiesis and impaired iron absorption. Neuropsychic State: Cognitive dysfunction, psychomotor retardation, memory loss, and depressive states, stemming from the weakening of interneuronal synaptic connections in the Central Nervous System.

DIAGNOSTIC STANDARDS

Modern laboratory assays allow for precise localization of the pathology: Primary: TSH \uparrow , fT_4 \downarrow. Secondary/Tertiary: TSH \downarrow (or inappropriately normal), fT_4 \downarrow

CONCLUSION

Hypothyroidism is not merely a localized glandular dysfunction but a multi-organ pathology affecting all physiological systems. A profound understanding of its pathophysiological mechanisms, especially in subclinical forms, is crucial for early diagnosis and adequate levothyroxine substitution therapy to maintain the patient's quality of life.

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