PECULIARITIES WHEN ACCOMPANIED BY HYPOTHYROIDISM AND IODINE DEFICIENCY IN PATIENTS WITH ADRENAL GLAND PATHOLOGY

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Abstract. *Hormones produced by the adrenal glands affect most functional and metabolic processes in the human body, ensuring stability under stress. Since the adrenal glands are a stress-sensitive organ in the endocrine regulation system, the development of adaptation mechanisms under stress conditions can be characterized by their morphological state.*

However, thyroid diseases associated with a persistent lack of hormones cause impaired homeostasis, metabolism, and oxygen exchange. Given that the adrenal glands are vital target organs of the thyroid gland, the study of their functional relationships under normal and pathological conditions is of particular interest.

Keywords: *hypothyroidism; adrenal insufficiency; stress response; iodine deficiency.*

ОСОБЕННОСТИ СОЧЕТАНИЯ ГИПОТИРЕОЗА И ЙОДОДЕФИЦИТА У БОЛЬНЫХ С ПАТОЛОГИЕЙ НАДПОЧЕЧНИКОВ

Аннотация. *Гормоны, вырабатываемые надпочечниками, влияют на большинство функциональных и метаболических процессов в организме человека, обеспечивая устойчивость в условиях стресса. Поскольку надпочечники являются стрессочувствительным органом в системе эндокринной регуляции, то развитие механизмов адаптации в условиях стресса можно охарактеризовать по их морфологическому состоянию. Однако заболевания щитовидной железы, связанные со стойким недостатком гормонов, вызывают нарушения гомеостаза, метаболизма и кислородного обмена. Учитывая, что надпочечники являются жизненно важными органами-мишенями щитовидной железы, особый интерес представляет изучение их функциональных взаимоотношений в норме и патологии.*

Ключевые слова: *гипотиреоз; надпочечниковая недостаточность; реакция на стресс; йододефицит.*

Introduction

Hypothyroidism is a chronic disease characterized by insufficient production of thyroid hormones—thyroxine (T4) and triiodothyronine (T3). The disorder can result from various causes, including iodine deficiency, autoimmune thyroiditis (Hashimoto’s disease), congenital

defects, and post-surgical or post-radiation thyroid damage. The global prevalence of hypothyroidism is significant, with iodine deficiency remaining the leading cause worldwide.

Symptoms of hypothyroidism include fatigue, weight gain, cold intolerance, depression, and cognitive impairments. If left untreated, it can lead to severe complications such as cardiovascular diseases, infertility, and myxedema coma. Iodine, an essential trace element, plays a critical role in thyroid hormone synthesis, and its deficiency directly contributes to thyroid dysfunction.

The adrenal glands, responsible for producing corticosteroids and catecholamines, are crucial in maintaining homeostasis, stress response, and metabolic functions. They are intricately linked with thyroid function, as thyroid hormones regulate adrenal steroidogenesis, while adrenal hormones influence thyroid hormone metabolism. Disruptions in one of these glands can significantly impact the function of the other.

Materials and Methods

This study involved a comprehensive review of scientific literature and clinical studies that analyze the structural and functional relationships between thyroid and adrenal gland pathology. The methodology includes:

- Histopathological examination of adrenal tissue samples;
- Measurement of serum thyroid hormone (T3, T4, TSH) and adrenal hormone (cortisol, aldosterone) levels;
- Assessment of iodine status through urinary iodine concentration and dietary intake evaluation;
- Correlation analysis between hypothyroidism severity and adrenal morphology.

Clinical and experimental studies were reviewed to investigate the extent of adrenal dysfunction in patients with hypothyroidism and iodine deficiency. Data on adrenal gland morphology, histological changes, and hormonal alterations were analyzed.

Results

Findings indicate that chronic hypothyroidism leads to significant morphofunctional changes in the adrenal glands. Key observations include:

- **Adrenal Cortical Atrophy:** Prolonged hypothyroidism was associated with a decrease in adrenal gland size, particularly in the zona fasciculata and zona reticularis, due to reduced ACTH stimulation.
- **Compensatory Adrenal Hyperplasia:** In contrast, some cases of iodine deficiency-induced hypothyroidism resulted in adrenal hyperplasia, particularly in the zona glomerulosa, due to excessive activation of the renin-angiotensin system.

- **Hormonal Imbalance:** Cortisol production was found to be altered in hypothyroid patients, with some exhibiting hypercortisolism due to stress-induced compensatory mechanisms, while others showed hypocortisolism due to impaired adrenal stimulation.
- **Disrupted Catecholamine Synthesis:** Thyroid dysfunction affected the adrenal medulla, leading to altered adrenaline and noradrenaline synthesis, impacting the stress response.

These findings suggest a bidirectional relationship between thyroid and adrenal function, with thyroid hormone insufficiency influencing adrenal steroidogenesis and adrenal hormones modulating thyroid activity.

Discussion

The study highlights the complex interplay between thyroid and adrenal glands in endocrine regulation. Several key mechanisms underpin this relationship:

- **Regulation of Adrenal Function by Thyroid Hormones:** Thyroid hormones enhance adrenal steroidogenesis by increasing ACTH receptor sensitivity and modulating enzyme activity in steroid biosynthesis pathways.
- **Impact of Glucocorticoids on Thyroid Function:** Glucocorticoids suppress thyroid-stimulating hormone (TSH) secretion and reduce peripheral conversion of T4 to T3, leading to a feedback inhibition that further exacerbates hypothyroidism.
- **Iodine Deficiency and Adrenal Dysfunction:** Chronic iodine deficiency not only impairs thyroid hormone synthesis but also disrupts adrenal gland homeostasis, leading to compensatory hormonal alterations and structural changes.

The correlation between stress and thyroid-adrenal interactions is also significant. Chronic stress-induced hypercortisolism can suppress TSH secretion, reducing thyroid hormone levels, while prolonged hypothyroidism can compromise adrenal function, leading to adrenal insufficiency.

Despite extensive research, gaps remain in fully understanding the exact mechanisms linking adrenal pathology to thyroid dysfunction. Further studies are needed to elucidate the long-term consequences of combined thyroid-adrenal dysfunction and the potential benefits of therapeutic interventions targeting both glands.

Conclusion

The study underscores the intricate relationships between adrenal pathology, hypothyroidism, and iodine deficiency. The findings suggest that:

1. Hypothyroidism leads to significant morphological and functional alterations in the adrenal glands, affecting both steroidogenesis and catecholamine synthesis.
2. Iodine deficiency exacerbates thyroid dysfunction, further impacting adrenal gland function through compensatory hyperplasia or atrophy.

3. Chronic stress and adrenal hormone imbalances can contribute to the progression of thyroid disorders, creating a bidirectional feedback loop.

Addressing iodine deficiency and optimizing thyroid function is essential for preventing adrenal dysfunction. Future research should explore targeted therapeutic interventions to improve outcomes for patients with coexisting thyroid and adrenal disorders.

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