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The Influence of Hypo- and Hyperthyroidism on Morphogenesis and Histophysiology of Adrenal Glands

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Review Article

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Abstract

Survey of literature concerning the influence of thyroid hormones imbalance on morphogenesis and histophysiology of adrenal glands. Elevated levels of thyroid hormones induce an increase in the adrenal cortex and subsequent hypercorticosteronemia due to stimulatory effect of thyroxine on the proliferation rate of adrenocorticocytes accompanied with increased density of vascular bed and increased secretion of catecholamines by chromaffine cells. Hypothyroidism induce significant decrease of adrenal cortex, with subsequent decrease of corticosteron production. Maternal hypothyroidism is associated with the delay in maturation of progeny adrenal glands. Both hypo- and hyperthyroidism claim for alterations in adrenal cells ultrastructure.

Keywords: Hypothyroidism; Hyperthyroidism; Adrenal Glands; Morphogenesis; Histophysiology

Introduction

Thyroid hormones – thyroxin T4 and triiodothyronin T3 – play an essential role in morphogenesis and histophysiology of all organ systems of the body [1,2]. Namely, it is generally accepted that hyperthyroidism stimulates metabolism and proliferation of target cells, while hypothyroidism has opposite effect – inhibition of the same activities [3,4]. The present survey is directed towards the evaluation of impact of thyroid gland disfunction on the adrenals development and of its functional consequences.

Hyperthyroidism

Numerous investigations revealed that elevated levels of thyroid hormones induce an increase in the adrenal cortex and subsequent hypercorticosteronemia [5-13]. The mechanism of this phenomenon is based on thyroxin driven modulation of adrenocorticocytes enzymatic activities [7-9]. In particular, it was determined that hyperthyroidism is accompanied by an increase of tyrosine hydroxylase activity [14] in combination with inhibition of ornithine decarboxylase [12] in adrenocorticocytes. Boler, et al. [15] found that the stimulatory effect of thyroxin on the proliferation rate of adrenocorticocytes is combined with the inhibition of their response to the action of ACTH. Meanwhile, Moore, et al. [16] reported the destructive effects of hyperthyroidism on the mitochondria of zona fasciculata cells.

According to data of Karaca, et al. [17], maternal hyperthyroidism is accompanied with the excess

production of the vascular endothelium growth factor (VEGF) and a significant increase in the density of the vascular bed of the adrenal glands of the offspring. Walter, et al. [18] found a direct positive correlation between the level of the pituitary gland thyroid-stimulating hormone and cortisol in the blood of young healthy people of both sexes. Huang, et al. [19] reported presence of thyroid hormone receptors in a certain population of adrenocorticocytes.

Results depicting the influences of hyperthyroidism on the adrenal medulla are less clear. Namely, Jonek, et al. [7,8] showed that under the influence of hyperthyroidism epinephrocytes and norepinephrocytes (chromaffine cells) increase secretion of catecholamines (adrenalin and noradrenalin). Lau, et al. [20] detected that increased levels of thyroid hormones in postnatal ontogenesis accelerates the formation of preganglionic synaptic contacts in the adrenal medulla, simultaneously inhibiting maturation of chromaffine cells, this option obviously may have a negative effect on the adrenomedullary function.

Investigations on the mechanisms of catecholamines action showed that under the influence of hyperthyroidism there increased content of adrenoreceptors in their target cells resulting with the increased impact of adrenaline, in particular, on the cardiovascular system [21]. Additionally, it was established negative correlation between hyperthyroidism and the level of noradrenaline in blood plasma, urine, and in the cardiac muscle [22]. Schernthaner, et al. [23] published a case report on feetback mechanism: induction a thyrotoxic crisis in a patient with Graves' disease due to stimulation of adrenal medulla. According to experimental data of Helmreich, et al. [24], the surge of adrenaline and noradrenaline under stressfull conditions was accompanied in rats by a decrease of T3, T4 and of pituitary thyroid-stimulating hormone in the peripheral blood flow.

Hypothyroidism

induce significant decrease of adrenal cortex [9,25-28], with subsequent decrease of corticosteron production [12,29]. Simultaneously increased secretion of hypothalamic corticotropin-releasing hormone and of ACTH by the pituitary gland, though its action on adrenocorticocytes deminished; interestingly, dopamine secretion remain unchanged [30,31]. Iranmanesh, et al. [32] reported a supplementation of hypothyroidism with hypercorticism due to a decrease in the metabolic clearance of cortisol and regression of its negative feedback on the hypothalamo-pituitary axis. Gomes Dumm, et al. [33] indicated damage in the ultrastructure of zona fasciculata adrenocorticocytes induced by hypothyroidism.

The decreased level of thyroxin during postnatal morphogenesis claims for a decrease in tyrosine hydroxylase activity of adrenocorticocytes [14], with simultaneous increase of dopamine-beta-hydroxylase activity in chromaffin cells [25]. Studies of Detiuk, et al. [34] revealed that maternal hypothyroidism induce reduction of adrenal cortex in their off springs, accumulation of lipid inclusions and ascorbic acid in within the cytoplasm of adrenocorticocytes, these signs encompassing certain delay in the adrenals maturation.

In clinical observations it was documented development of Addison disease (reduced secretion of adrenal cortex hormones) in hypothyroidism affected patients [35]. In turn, post steroid replacement it was achieved normalization of thyroid-stimulating hormone (TSH) and free thyroxin levels [36]. Moreover, elevated levels of glucocorticoids inhibit pituitary secretion of TSH, which caused the development of secondary hypothyroidism [37]. Combined autoimmune destruction of thyroid and adrenal glands is renowned as Schmidt's syndrome [38].

Pramanik, et al. [39] published a case report claiming empty pituitary syndrome as a missing link in between primary hypothyroid and secondary adrenal insufficiency. The similarities of clinical manifestations of pheochromocytoma (adrenal tumors accompanied by hypersecretion of noradrenaline) and thyrotoxicosis (tachycardia, sweating, weight loss, tremor, fever) also indicate the existence of an intimate correlation between thyroid gland and adrenal medulla [21], although the nature of these interrelations is not as unambiguous as between the thyroid function and the adrenal cortex histophysiology.

Conclusion

Numerous studies demonstrate direct influence of thyroid hormones on the development and function of adrenal cortex: hyperthyroidism accelerates its morphogenesis and stimulates hypertrophy; hypothyroidism, on the contrary, slows morphogenesis and causes hypoplasia [40]. The hormones of thyroid and adrenal glands play a vital role in providing intrauterine homeostasis, differentiation and maturation of the fetal organs in accordance with the time of gestation [41]. An indication of the close functional relationship between the

thyroid and adrenal glands may be the fact that thyroxin and triiodothyronine, as well as the catecholamine produced by adrenal medulla, are derivatives of aminoacid tyrosine [2]. The sympatho-adrenal system, which includes the sympathetic nervous system and the adrenal medulla, interacts with thyroid hormones at many levels. Both systems are evolutionary old, maintaine the temperature homeostasis, and synergistically regulate metabolic processes of the organism [42].

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