

Original Article

Subclinical Hypothyroidism and Functional Hemodynamics of the Heart

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Abstract

Subclinical hyperthyroidism (SHyper), a laboratory decrease in thyroid-stimulating hormone levels with peripheral thyroid hormone levels within the reference interval, is considered a medical condition, especially its impact on cardiac hemodynamics among young women. To determine the effects of subclinical thyroid dysfunction on hemodynamics in young women. clinical observation included two groups of women: (1) the main group consisted of 30 patients with subclinical hyperthyroidism, which was examined at the Family Medicine Clinic of the West Kazakhstan Marat Ospanov Medical University (Aktobe, Kazakhstan) and (2) the control group consisted of 30 practically healthy women. The groups were comparable in age (30–42), mean age 36.2 ± 7.0 years. Blood pressure (BP) was measured, heart rate (HR) was counted, and thyroid hormones were determined. in the examined patients, clinical symptoms of pathology were not observed; laboratory tests determined a decrease in thyroid-stimulating hormone (TSH) in the main group of patients to 2.48 ± 0.23 mIU/L in comparison with the control group 3.65 ± 1.3 mIU/L, which was significant ($p < 0.05$) increase in blood pressure SBP/DBP from $122.34 \pm 2.7/78.3 \pm 2.7$ to $127.8 \pm 3.1/83.2 \pm 2.1$ mmHg and HR from 77.7 ± 1.9 to 82.2 ± 2.6 in 1 minute. A significant ($r^2 = 0.482$) interaction was determined between TSH -0.236 mIU/L and HP $+20.3$ in 1 minute, this could be a predictor of an increase in heart rate greater than the reference value (60–80 per minute), and an increased heart rate is considered a predictor of an increase in blood pressure. Subclinical hyperthyroidism may produce a significant laboratory decrease in TSH, without significant clinical symptoms of increased blood pressure. In conclusion, current observational experience suggests that small decreases in laboratory values of thyroid-stimulating hormone may affect blood pressure and heart rate.

Keywords: subclinical thyrotoxicosis, thyroid-stimulating hormone, triiodothyronine, thyroxine, blood pressure

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1. Introduction

Subclinical hyperthyroidism is a biochemical condition characterized by low or suppressed levels of thyroid-stimulating hormone (TSH <0.4 mIU/L) in the blood serum within the normal reference range of thyroxine T4 (T4) and free triiodothyronine (FT3) [1–3]. The biological role of thyroid hormones is carried out through protein metabolism. The mechanism of action of thyroid hormones (TH) is realized through receptors: α -1, α -2, β -1 on the surface of cardiomyocyte membranes. Changes in the function of the cardiovascular system depend on the dose of thyroid hormones (TG). Under physiological conditions, small doses of hormones have anabolic effects, large doses have catabolic effects, and the heart reacts even to minor fluctuations in plasma TG concentrations [4,5].

The main effects on cardiomyocytes occur at nuclear and extranuclear levels. At the level of the cell nucleus, the active form of triiodothyronine (T3), enhancing the synthesis of cardiac-specific proteins, increases the contractile function of the heart. The extranuclear actions of hormones are realized through the cytoplasmic membrane and cell organelles. T3 and to a lesser extent T4 can increase the density of β 1 receptors on the surface of cardiac muscle cells [6–8]. The pathophysiological mechanism for the development of cardiac dysfunction is carried out by oxidative phosphorylation. This in turn leads to inhibition of anabolic metabolism and a decrease in protein synthesis in the myocardium. Oxygen absorption and free oxidation by tissues increase, resulting in energy deficiency. Thus, cardiac function is impaired by TG metabolism, energy resources are reduced due to protein catabolism, and subclinical hyperthyroidism is a risk for developing increased blood pressure and increased heart rate. This study aimed to determine the effects of subclinical thyroid dysfunction on hemodynamics in young women.

2. Materials and Methods

2.1. Study design

Clinical, descriptive, cross-sectional case-control study at the Family Medicine Clinic of the West Kazakhstan Marat Ospanov Medical university (Aktobe, Kazakhstan). Informed consent for the study was obtained. The recorded data was compiled and entered in a spreadsheet (Microsoft Excel) and then exported to data editor of SPSS Version 20.0 (SPSS Inc., Chicago, Illinois, USA).

2.2. Characteristics of patients

The groups were comparable in age (30–42), mean age 36.2 ± 7.0 years. All subjects were measured according to the generally accepted method of blood pressure (BP) using a mechanical tonometer according to the Korotkov method, counting the number of heartbeats (HR). Thyroid hormones were determined in the Olymp laboratory, Aktobe.

2.3. Statistical analysis

The results were analyzed using $M \pm SD$ methods, nonparametric statistics using the Mann-Whitney test to determine differences in value for small groups, and linear regression analysis for interactions between the main parameters. The difference was considered statistically significant at $P > 0.05$.

3. Results

Thyroid hormones were predetermined for the diagnosis of SHyper. No clinical symptoms of the disease were observed in the patients. The increase in blood pressure SBP/DBP in the main group of patients was $127.8 \pm 3.1 / 83.2 \pm 2.1$ mmHg and differed in a significant P-level ($p < 0.000$) from the normative intervals (120/129–80/84 mmHg) and the control group $122.3 \pm 2.7 / 78.3 \pm 2.7$ mmHg. The increase in heart rate 82.2 ± 2.6 per 1 minute in comparison with the normal interval (80–90 per 1 minute) and with the control group 77.7 ± 1.9 per minute was significant ($p < 0.000$). The decrease in TSH level in the main group - 2.48 ± 0.23 mIU/L with a value ($p < 0.000$) differed from the reference interval (0.27-4.2 mIU/L) and from the control group (3.65 ± 1.3 mIU/L) (Table 1).

TABLE 1: Characteristics of the examined patients.

Parameter	Reference Range	Main Ggroup (n=30)	Control Group (n=30)	P-value
Blood pressure, mmHg				
Systolic	120-129	$127.8 \pm 3.1^*$	$122.34 \pm 2.7^*$	0.000
Diastolic	80-84	$83.2 \pm 2.1^*$	$78.3 \pm 2.7^*$	0.000
Heart rate (HR), per 1 minute	60-80	$82.2 \pm 2.6^*$	77.7 ± 1.9	0.000
Thyroid-stimulating hormone (TSH), mIU/L	0.27-4.2	$2.48 \pm 0.23^*$	3.65 ± 1.3	0.000
Triiodothyronine, (T3), ng/dl	1.2-3.1	2.62 ± 0.26	2.63 ± 0.27	0.290
Thyroxine (T4), ng/dl	66-181	78.06 ± 3.1	75.6 ± 3.8	0.005

Mean (M) \pm standard deviation (SD); Mann-Whitney U test, $p < 0.0500$;

In linear regression analysis, a significant interaction was observed between the heart rate and thyroid-stimulating hormone ($r^2 = 0.48$), that is, a decrease in TSH (-0.236 mIU/L) increased HR ($+20.31$ in 1 minute) (Figure 1). Analysis of observational results shows that patients with subclinical hyperthyroidism have changes in hemodynamic parameters depending on the decrease in TG levels than the group of practical healthy patients.

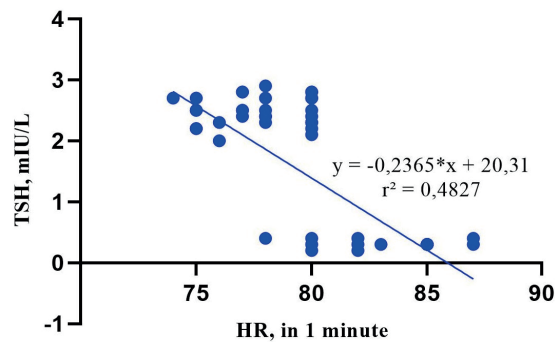


Figure 1: Indicators of interaction between TSH and HP.

4. Discussion

With a TSH level of $2.480.23$ mIU/L and an average age of $36.27.0$ years, patients in the main group were diagnosed with subclinical hyperthyroidism without any overt clinical signs. According to our observations, thyroid hormones caused an increase in heart rate of 82.2 beats per minute and an SBP/DBP elevation of $127.83.1/83.22.1$ mmHg. 35 patients were enrolled in the first investigation on the impact of subclinical hyperthyroidism on blood pressure [9], and 44 patients were enrolled in a subsequent study with 24-hour blood pressure monitoring, which caused the nighttime average SBP/DBP to rise in comparison to the euthyroid state [10]. According to research, SyHyper affects blood pressure levels. With a mean age of 48 ± 17 years and 1240 (78.9%) women, subclinical hyperthyroidism was identified in 86 patients (10.4%) by other researchers [11]. Clinical symptoms have been found to be less frequent in older individuals, although they are more frequently associated with biochemical severity in younger people. Heart rate is a factor in raising blood pressure, and according to our observation, a drop-in thyroid-stimulating hormone (-0.236 mIU/L) raised HR ($+20.31$ in 1 minute). This interaction between the two variables was significant ($r^2 = 0.48$).

In a multivariate logistic study of the incidence of hypertension during a 5-year period, the authors [12] found comparable hypertension in both groups. At the same time, it was 31% in the SHyper group and 19% in the euthyroidism group. Heart rate variability (HRV) is a way that other researchers [13] believe can be used to demonstrate how

thyroid hormones affect the circulatory system; with hyperthyroidism, this indicator will be decreased. These sources for younger patients are insufficient to make reliable conclusions, and experts advise against utilizing medication therapy in young, asymptomatic patients [14]. In contrast, our study included young women. Subclinical hyperthyroidism in cardiovascular disorders frequently affects the elderly.

5. Conclusion

The results obtained during the observation confirm that significant diagnosed subclinical hyperthyroidism can be a harbinger of hemodynamic disturbances in heart parameters, and in all likelihood be a predictor of high blood pressure. According to the current research, TSH and heart rate interact, and TSH probably functions as a statistically significant hemodynamic predictor of young women's hemodynamic cardiac problems and elevated blood pressure without any discernible therapeutic relevance.

References

- [1] Léger J, Carel JC. Diagnosis and management of hyperthyroidism from prenatal life to adolescence. *Best Pract Res Clin Endocrinol Metab.* 2018;32(4):373–386.
- [2] Mooij CF, Cheetham TD, Verburg FA, Eckstein A, Pearce SH, Léger J, et al. 2022 European Thyroid Association Guideline for the management of pediatric Graves' disease. *Eur Thyroid J.* 2022 ;11(1):e210073.
- [3] Karen Tsai MD, Angela M. Leung, MD, MSc. Subclinical Hyperthyroidism: A Review of the Clinical Literature. *Endocr Pract.* 27 (2021) 254e260.
- [4] Paschou SA, Bletsas E, Stampouloglou PK, Tsigkou V, Valatsou A, Stefanaki K, et al. Thyroid disorders and cardiovascular manifestations: an update. *Endocrine.* 2022;75(3):672–683.
- [5] Manolis AA, Manolis TA, Melita H, Manolis AS. Subclinical thyroid dysfunction and cardiovascular consequences: an alarming wake-up call? *Trends Cardiovasc Med.* 2020;30(2):57–69.
- [6] Danzi S, Klein I. Thyroid disease and the cardiovascular system. *Endocrinol Metab Clin North Am.* 2014;43(2):517–528.
- [7] Fazio S, Palmieri EA, Lombardi G, Biondi B. Effects of thyroid hormone on the cardiovascular system. *Recent Prog Horm Res.* 2004;59(1):31–50.
- [8] Omidi N, Khorgami M, Tajrishi FZ, Seyedhoseinpour A, Pasbakhsh P. The role of thyroid diseases and their medications in cardiovascular disorders: A review of the

literature. *Curr Cardiol Rev.* 2020;16(2):103–116.

- [9] Walsh JP, Bremner AP, Bulsara MK, O’Leary P, Leedman PJ, Feddema P, et al. Subclinical thyroid dysfunction and blood pressure: a community-based study. *Clin Endocrinol (Oxf).* 2006;65(4):486–491.
- [10] Kaminski G, Makowski K, Michałkiewicz D, Kowal J, Ruchala M, Szczepanek E, et al. The influence of subclinical hyperthyroidism on blood pressure, heart rate variability, and prevalence of arrhythmias. *Thyroid.* 2012;22(5):454–460.
- [11] Goichot B, Caron P, Landron F, Bouée S. Clinical presentation of hyperthyroidism in a large representative sample of outpatients in France: relationships with age, aetiology and hormonal parameters. *Clin Endocrinol.* 2016;84(3):445–451.
- [12] Völzke H, Ittermann T, Schmidt CO, Dörr M, John U, Wallaschofski H, et al. Subclinical hyperthyroidism and blood pressure in a population-based prospective cohort study. *Eur J Endocrinol.* 2009;161(4):615–621.
- [13] Brusseau V, Tauveron I, Bagheri R, Ugbolue UC, Magnon V, Bouillon-Minois JB, et al. Heart Rate Variability in Hyperthyroidism: A Systematic Review and Meta-Analysis. *Int J Environ Res Public Health.* 2022;19(6):3606.
- [14] Vidili G, Delitala A, Manetti R. Subclinical hyperthyroidism: The cardiovascular point of view. *Eur Rev Med Pharmacol Sci.* 2021;25(8):3264–3271.