CODEN [USA]: IAJPBB ISSN: 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES

SJIF Impact Factor: 7.187
http://doi.org/10.5281/zenodo.3960745

Available online at: http://www.iajps.com *Research Article*

AN ANALYSIS OF SYSTEMIC HYPERTENSION WITH SUBCLINICAL HYPOTHYROIDISM

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Article Received: May 2020 **Accepted:** June 2020 **Published:** July 2020

Abstract:

Aim: *Thyroid dysfunction, both hypothyroidism and hyperthyroidism, may increase the risk of hypertension. However, it is still controversial whether mild thyroid dysfunction, such as subclinical hypothyroidism, affects blood pressure. Aim of this study was to explore relationship of hypertension with different levels of thyroid stimulating hormone in subjects with subclinical hypothyroidism and euthyroidism.*

Methods: *This cross-sectional study was conducted at Department of General Medicine, District Headquarter Hospital, Gujranwala for one-year duration from March 2019 to March 2020. A total of 500 newly diagnosed hypertensive subjects were evaluated for demographic characteristics, body mass index, smoking habits, serum TSH, and free T4. Subjects were further divided into various groups depending on TSH levels. Statistical software, SPSS version 17.0 was used for analysis.*

Results and Conclusion: *In the present study, Systolic BP and diastolic BP was higher in subclinical hypothyroidism subjects than that of the euthyroid group (P less than 0.05 for both). From this study, we can conclude that subclinical hypothyroid subjects have more tendencies to develop hypertension than euthyroid subjects. Therefore, subclinical hypothyroid subjects should be regularly screened for hypertension.*

Keywords: *Hypertension, Hypothyroidism, Euthyroidism*

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Please cite this article in press Fajar Ikram et al, ***An Analysis Of Systemic Hypertension With Subclinical Hypothyroidism.,*** *Indo Am. J. P. Sci, 2020; 07(07).*

INTRODUCTION:

Thyroid dysfunction, both hypothyroidism and hyperthyroidism, may increase the risk of hypertension. However, it is controversial whether mild thyroid dysfunction, such as subclinical hypothyroidism, affects blood pressure[1-2]. Thyroid stimulating hormone (thyrotropin, TSH) and normal free thyroxine (T4) levels are high in patients with subclinical hypothyroidism[3-4]. Previous research has suggested that subclinical abnormalities at TSH levels are associated with harmful effects on the cardiovascular system[5-6]. The research also suggests a link between subclinical hypothyroidism and hypertension, which was confirmed by some later, but not all of them, large cross-sections and case control studies[7-8]. The purpose of this study is to investigate the link between hypertension and various levels of thyroid stimulant hormone in patients with subclinical hypothyroidism and euthyroidism. Thyroid hormone relaxes vascular smooth muscle cells, thereby reducing peripheral vascular resistance. In addition, sympathetic nervous system activation, increased arterial stiffness, a decrease in the rate of glomerular filtration, abnormalities of sodium metabolism and endothelial dysfunction may adversely affect the regulation of blood pressure (BP) in association with hypothyroidism.6-8 Additionally, subclinical hypothyroidism has been reported to impair both left ventricular relaxation and systemic vascular resistance (SVR).9 Healthy, normotensive individuals exhibit a 10-20% nocturnal decrease in BP during the night. This decrease is subject to the influence of psychosocial, behavioral, and neurohumoral factors.10 Non-dipping BP, defined as an average nighttime BP reduction less than 10% of the average daytime BP,11 has been linked to target-organ damage, cerebrovascular accidents, and cardiovascular morbidity and mortality, both in hypertensive and normotensive individuals. A relationship between hypothyroidism and non-dipping has been reported in normotensive patients but there is insufficient information regarding the relationship between subclinical hypothyroidism and non-dipping.

MATERIALS AND METHODS:

This cross-sectional study was conducted at Department of General Medicine, District Headquarter Hospital, Gujranwala for one-year duration from March 2019 to March 2020. A total of 500 newly diagnosed cases of hypertension were assessed in terms of demographic characteristics, body mass index, smoking habits, serum TSH and free T4. Participants were divided into different groups by TSH levels. All elating hypothyroidism and hyperthyroidism problems have been excluded from the study.

RESULTS:

Table 1 shows the features of all the topics in our study. In patients with subclinical hypothyroidism, systolic blood pressure and diastolic blood pressure were higher than in the group (P.05 for both)

Table 1. Patient's Characteristics

	Subclinical Hypothyroidism	Euthyroidism
Total Number of Subjects	41	439
Age (Years)	50.4±12.4	48.9±11.3
Gender (Female %)	78.1	70.4
Smoking Habits (%)	14.6	13.9
BMI (Kg/M^2)	24.6±3.6	24.1±3.1
TSH	7.1±2.1	2.5±1.2
Systolic BP (mmHg)	130.5±20.4	127.3±19.4
Diastolic BP (mmHg)	82.9±12.8	81.1±12

DISCUSSION:

The results of our study show a link between subclinical hypothyroidism and elevated blood pressure levels. Various mechanisms may explain why subclinical hypothyroidism has a negative effect on blood pressure[10-12]. Clinical hypothyroidism is known to increase blood pressure due to increased systemic vascular resistance. Several studies show that cardiovascular disorders are present in the subclinical stage of hypothyroidism[13-15]. Luboshitzky et al. In the subclinical hypothyroidism group, the incidence of hypertension was significantly higher than in the normal control group, which was confirmed by the result we achieved[16-18]. The Rotterdam study showed that subclinical hypothyroidism is an independent risk factor for atherosclerosis and myocardial infarction. Blood hypercoagulation, viscosity of blood and lipid abnormalities in patients with subclinical hypothyroidism may increase the risk of atherosclerosis[19-20]. These factors can also play a role in pathogenesis, where subclinical hypothyroidism affects blood pressure. A number of studies have suggested that the risk of hypertensive cardiovascular complications correlates more closely with 24-hour, daytime, or

nighttime ABPM than with the BP measured in the office. Thyroid hormone plays a role in blood pressure homeostasis. The increase of systemic vascular resistance may be the main mechanism causing hypertension in clinical hypothyroidism patients. The prevalence of hypertension in the subclinical hypothyroidism group has been suggested to be higher than that in the normal control group.18 Even though TSH was in the normal range, there was a linear increase in SBP and DBP correlating with increasing serum TSH levels, and the prevalence of hypertension also increased. The only study ever published on this topic, Marcia et al., reported a slightly higher BP in patients with normotensive subclinical hypothyroidism than in the controls, but it did not reach clinical significance. In our study, we demonstrated significantly higher mean, daytime and nighttime DBP, and nighttime SBP in subclinical hypothyroidism patients by including more patients. DBP may vary directly with serum TSH levels over the entire spectrum of thyroid disease.6 Overt hypothyroidism is associated with increased SVR, decreased cardiac contractility, and decreased cardiac output. These complications may be the result of diastolic hypertension in hypothyroid patients. Elevated diastolic BP in subclinical hypothyroidism patients has been shown in prior studies, but these were done with patients who already had hypertension or with older patients. Our study is the first study that showed elevated DBP in normotensive subclinical hypothyroidism patients compared to age and sex-matched controls. Increasing trend of DBP may have become statistically significant with increasing number of patients in our study. The relationship between non-dipping and cardiovascular accidents and end-organ damage is well known. Hormones affecting BP such as cortisol, catecholamines and renin are also thought to be affecting non-dipping. Some studies had demonstrated that thyroid disfunction affects these hormones. Another hypothesis on the mechanism of non-dipping is an in equilibrium between the sympathic and parasympathic systems. The upregulation of sensitivity of the sympathic system due to TSH and declines in vascular relaxation can lead to non-dipping.5 TSH induces increased sodium reuptake from the kidneys, and sodium sensitivity of patients with non-dipping can play a role in its pathophysiology.7 A previous study demonstrated that non-dipper hypertensive patients had lower serum FT3 levels than dipper patients and that FT3 was an independent predictor of non-dipper hypertension. In a recent study, the number of non-dippers was found to be significantly higher in the group involving patients with overt hypothyroid and subclinical hypothyroidism compared to healthy euthyroid individuals. An essential limitation of thyroid dysfunction studies is that the time of onset of thyroid pathology in patients with subclinical hypothyroidism is not precisely known. The time required for the development of abnormalities of blood pressure and lipid levels is difficult to detect. Therefore, prospective studies with a large number of patients are needed to detect the effect of subclinical hypothyroidism on hypertension and other cardiovascular risk factors such as dyslipidemia and inflammation. Another limitation of our investigation was that it was based upon data provided by a single center, with a small number of patients. Also, ABPM was only performed once, which was another study limitation

CONCLUSION:

From this study, we can conclude that subclinical hypothyroid subjects have more tendencies to develop hypertension than euthyroid subjects. Therefore, subclinical hypothyroid subjects should be regularly screened for hypertension.

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