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Research Article

ANALYSIS OF RELATIONSHIP OF PARATHYROID HORMONE AND HYPERTENSION

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Abstract:

Introduction: Numerous studies in humans and experimental models have shown that alterations in calcium homeostasis are associated with an increased risk of cardiovascular complication. In particular, changes in systemic calcium metabolism are thought to play an important role in the regulation of blood pressure. **Aims and objectives:** The basic aim of the study is to find the relationship between parathyroid hormone and hypertension in local population of Pakistan. **Material and methods:** This study was conducted at DHQ Hospital, Muzaffargarh during Jan 2018 to July 2018. This study was conducted on 100 patients which was suffering from hypertension and visit the hospital regularly. In all patients, 5 mL of fasting blood sample was taken before hemodialysis and was analyzed for serum calcium, phosphorous, albumin, PTH, and hemoglobin. Serum calcium, phosphorous, and albumin were measured. BP was obtained using an automatic BP monitor. **Results:** Calcium and PTH levels significantly decreased in all hypertensive patients with a cure rate of 99.1%. The mean systolic and diastolic BP decreased in the total population of hypertensive patients and hypertensive patients on antihypertensive therapy. Patients with PHPT experienced a significant decrease in both systolic BP ($P < .001$) and diastolic BP. **Conclusion:** It is concluded that the association between PTH and BP observed in this study contributes to the understanding of calcemic hormones and BP regulation.

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INTRODUCTION:

Numerous studies in humans and experimental models have shown that alterations in calcium homeostasis are associated with an increased risk of cardiovascular complication. In particular, changes in systemic calcium metabolism are thought to play an important role in the regulation of blood pressure. One hypothesis for this link implicates parathyroid hormone (PTH). Serum calcium level is tightly regulated by PTH in a classic negative-feedback system [1]. A small decrease in serum calcium stimulates an abrupt increase in PTH secretion, which leads to calcium mobilization from bone, increased renal tubular calcium reabsorption and increased renal hydroxylation of 25-hydroxyvitamin D to the biologically more active 1,25-dihydroxyvitamin D [2].

Thyroid gland along with the parathyroid glands and heart share a close relationship arising in embryology. In ontogeny, the thyroid and heart migrate together. There is a strong physiological relationship between the two organs, which is affirmed by predictable changes in cardiovascular functions across the entire range of thyroid disease states. Many symptoms and signs recognized in patients with overt hyperthyroidism and hypothyroidism are due to increased or reduced action of thyroid hormone on the heart and the vascular system, respectively [3].

Increases in parathyroid hormone (PTH) have been associated with changes in the vascular tone and renin angiotensin system. Hyper functioning parathyroid glandular disorders have been for long associated with an increased risk of hypertension, though a causal relationship is still not established. Most of the molecular and cellular mechanisms responsible for the cardiovascular effects of the thyroid hormone have been clarified [4]. Thyroid hormone exerts both genomic and non-genomic effects on cardiac myocytes. Studies have confirmed T_3 as the active form of thyroid hormone that accounts for the vast majority of thyroid effects including stimulation of tissue thermogenesis, alterations in the expression of various cellular proteins and action on the heart and vascular smooth muscle cells [5]. The process of the genomic effect of thyroid hormone begins with the entry of T_3 into the cardiomyocyte through specific transport proteins located within the cell membrane [6].

Hypothyroidism is considered a disease that may alter blood pressure (BP) and has been recognized as a cause of secondary hypertension. The most common type of hypothyroidism is that caused by

primary thyroid gland failure. In turn, chronic autoimmune lymphocytic thyroiditis is the most common cause of primary thyroid dysfunction. Replacement of deficient thyroid hormones reduces high BP and total cardiovascular risk. Some studies have indicated a high prevalence of systolic and diastolic hypertension in hypothyroidism, while other studies have reported no association of diastolic hypertension with hypothyroidism in geriatric patients in a primary care setting. Another study reported that diastolic BP correlated significantly with T_4 and T_3 levels in slightly hypothyroid females over 50 years of age [7].

Aims and objectives

The basic aim of the study is to find the relationship between parathyroid hormone and hypertension in local population of Pakistan.

MATERIAL AND METHODS:

This study was conducted at DHQ Hospital, Muzaffargarh during Jan 2018 to July 2018. This study was conducted on 100 patients which was suffering from hypertension and visit the hospital regularly. The exclusion criteria were increased dose or number of prescribed antihypertensive drugs during the last 3 months, cigarette smoking, and a body mass index greater than 25 kg /m², and corticosteroid administration. The participants with hypertension were divided based on the stage of hypertension according to the definition of Joint of National Committee for hypertension.

Laboratory Tests

In all patients, 5 mL of fasting blood sample was taken before hemodialysis and was analyzed for serum calcium, phosphorous, albumin, PTH, and hemoglobin. Serum calcium, phosphorous, and albumin were measured. BP was obtained using an automatic BP monitor (Omron model HEM-712C, Omron Health Care, Inc., USA). Three measures were taken at rest in a sitting position, with intervals of 5 min between the measurements. The average from the last two measurements was taken for analysis. The participants were stratified into categories: 1) normal BP (NBP): those with systolic/diastolic BP.

Statistical Analyses

Comparisons between the two groups were done using the t test or the chi-square, where appropriate. Data analysis was carried out using the SPSS software (Statistical Package for the Social Sciences, version 15.0, SPSS Inc, Chicago, Ill, USA).

RESULTS:

Calcium and PTH levels significantly decreased in all hypertensive patients with a cure rate of 99.1%. The mean systolic and diastolic BP decreased in the total population of hypertensive patients and hypertensive patients on antihypertensive therapy. Patients with PHPT experienced a significant decrease in both

systolic BP ($P < .001$) and diastolic BP. High BP was present in 34% of the whole sample, and another 16% were taking medication for hypertension. Overweight and obesity (WHO, 1997) was present in 75% of the individuals. Only 23% of whole sample reported practice regular physical exercise and the use of sunscreen was present in 22% of individuals.

Table 01: Analysis of relationship of hyperthyroidism and hypertension

Variable	Whole sample	Normal blood pressure	High blood pressure	Normal blood pressure through medication	P-value
Participants (n)	332	166	112	54	
Age (y)	50 (15)	42 (13)	57 (14)	59 (11)	0.000
BMI (kg/m ²)	29 (6)	27 (5)	30 (6)	31 (6)	0.000
%FFM	68 (10)	69 (10)	67 (9)	65 (9)	0.015
%FM	32 (10)	31 (10)	33 (9)	35 (9)	0.015
Waist circumference (cm)	97 (13)	92 (13)	101 (14)	100 (12)	0.000
Gender (%)					
Male	38	38	45	24	
Female	62	62	55	76	
Systolic BP (mm Hg)	129 (18)	118 (11)	148 (14)	125 (9)	0.000
Diastolic BP (mm Hg)	80 (11)	74 (8)	89 (11)	77 (7)	0.000
Total cholesterol (mg/dL)	190 (41)	184 (41)	198 (42)	193 (39)	0.022
LDL-C (mg/dL)	120 (37)	117 (37)	125 (37)	118 (36)	NS
HDL-C (mg/dL)	43 (12)	43 (11)	43 (12)	44 (11)	NS
Triacylglycerol (mg/dL)	134 (76)	122 (77)	146 (79)	148 (57)	0.009
Glucose (mg/dL)	93 (11)	91 (11)	94 (13)	97 (12)	0.006
Vitamin D insufficiency (%)	86	88	84	87	NS
High PTH (%)	12	10	14	13	

DISCUSSION:

Subclinical hyperthyroidism is characterized by subnormal thyrotropin (TSH) serum levels in the presence of circulating thyroid hormones in the normal range for the general population. It may be due to an intrinsic pathology of the thyroid gland (endogenous subclinical hyperthyroidism) or a consequent suppressive or replacement l-thyroxine therapy (exogenous subclinical hyperthyroidism). Exogenous subclinical hyperthyroidism is the condition more frequently seen in clinical practice [8].

A number of studies have investigated the effects of subclinical hyperthyroidism on the heart, showing that this condition may be associated with various abnormalities of cardiac structure and function. The cardiovascular disorders associated with subclinical hyperthyroidism may be a direct effect of thyroid

hormone disturbance or may reflect an increased arterial pressure level in these patients. There are no consistent studies proving that arterial BP rises in such patients. Recent meta-analyses of five large studies evaluating the incidence of hypertension in these patients did not reveal increased BP levels in individuals with suppressed serum TSH levels and free thyroid hormones within the reference range [9].

The more consistent abnormalities found in patients with subclinical hyperthyroidism are increased heart rate, prevalence of supraventricular arrhythmias, endothelial dysfunction and increased LV mass. This enhancement of LV mass is often associated with rise in systolic function and impaired myocardial relaxation [10]. The rise in LV mass is due to concentric remodeling and is related to the duration of subclinical hyperthyroidism rather than to levels of circulating thyroid hormones. Cardiac involvement in subclinical hyperthyroidism is reversible [11].

Compelling evidence indicated that subclinical hyperthyroidism is associated with increased cardiovascular mortality, and therefore the current opinion is to avoid or correct subclinical hyperthyroidism in all patients affected with benign thyroid disease [12].

CONCLUSION:

It is concluded that the association between PTH and BP observed in this study contributes to the understanding of calcemic hormones and BP regulation. Hypertension associated with common endocrine conditions which are not classically considered to be etiologies involved in the work up of a patient with suspected secondary hypertension.

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