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Correlation between hyperthyroidism and heart disease

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Abstract

This study investigates the impact of hyperthyroidism on cardiac function, specifically examining the relationship between elevated thyroid hormone levels (T3, T4, TSH) and B-type natriuretic peptide (BNP), a key biomarker for cardiac stress. Hyperthyroidism can lead to increased heart rate, cardiac output, and in some cases, heart failure. By measuring thyroid hormones and BNP levels in hyperthyroid patients, the study aims to explore how thyroid overactivity contributes to cardiac strain. Blood samples were taken from 180 volunteers: including 130 hyperthyroid patients divided into hyperthyroid patient (50), hyperthyroid patient with heart disease (50) and hyperthyroid patient take drugs (30) and 50 healthy controls group. Patients had significantly higher levels of T3, T4, FT3, and FT4 with reduced TSH compared to controls ($P < 0.00001$). BNP levels were significantly higher in patients with hyperthyroidism and heart illness, and substantially higher in those receiving therapy. Regression research found that FT3 and T3 were substantial positive predictors of BNP levels, but TSH had a significant negative connection. The statistical model explained 56.6% of the variance in BNP ($R^2 = 0.566$), suggesting a strong relationship between thyroid over activity and cardiac stress.

Keywords: Hyperthyroidism, Thyroid hormones, B-type Natriuretic Peptide (BNP), Heart disease

Introduction

Thyroid hormones have a central role in cardiovascular homeostasis. Thyroid hormones modulate cardiac and vascular function, basal metabolic rate, glucose tolerance, serum lipids and kidney function (1–3). Hyperthyroidism is a medical condition in which the thyroid gland produces and secretes an excessive amount of thyroid hormone [2]. an important role in regulating cardiac activities and affecting cardiovascular hemodynamics, thyroid conditions can cause metabolic and hemodynamic changes that may result in heart failure (HF) (3). THs play a crucial role in the metabolism, growth and development of most of the tissues in the body (4,22). As a result, hyperthyroidism has broad effects on the physiological and biochemical functions of multiple systems. It is now widely accepted that patients with hyperthyroidism are at increased risk of several cardiovascular diseases (5, 6).

The natriuretic peptide system involves polypeptide hormones produced by myocardial cells, primarily aimed at regulating volemia and natremia, thus maintaining cardiovascular system homeostasis. The recognition of cardiac endocrine function dates to 1964, while a definitive characterization of natriuretic peptides (NPs) were achieved approximately two decades later [7]

B-type natriuretic peptide (BNP) is a cardiac neurohormone generated by ventricles in response to volume expansion or pressure overload. BNP and N-terminal prohormone of brain natriuretic peptide (NT-proBNP) are two types of natriuretic peptides cleaved from Pro brain natriuretic peptide (proBNP). Compared to BNP, NT-proBNP is more stable and has a longer biological half-life. Thus, NT-proBNP is a better indicator for diagnosing or ruling out HF (8). NT-proBNP is also a good marker for assessing the severity and prognosis of this condition (9).

Understanding the relationship between thyroid hormone profiles—specifically TSH, T3, T4, FT3, and FT4—and BNP levels in patients with hyperthyroidism is crucial. This knowledge can provide valuable insights into the utility of BNP as an early marker of cardiac involvement in hyperthyroid patients, even in the absence of clinically apparent heart failure. Such findings could pave the way for improved risk stratification and early intervention strategies in thyroid-related cardiac dysfunction.

Methods

This case-control study recruited 180 volunteers

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from Medicine City Teaching Hospital and its consulting clinic in Baghdad, Iraq. Participants were divided into four groups: 50 hyperthyroidism patients (11 males, 39 females), 50 hyperthyroidism patients on medication (11 males, 39 females), 30 hyperthyroidism patients with heart disease (9 males, 21 females), and 50 healthy controls (11 males, 39 females). All participants provided informed consent and completed a questionnaire detailing their medical history. Exclusion criteria for healthy controls and patients included individuals with any other chronic conditions, smokers, pregnant women, and those with cancer.

Thyroid hormones and BNP serum concentration measurement

A 5 ml venous blood sample was collected from each patient into a gel separator tube and centrifuged at 3000 rpm to obtain serum. The serum level of B-type natriuretic peptide (BNP) was measured using the Cobas e411 analyzer (Roche Diagnostics, Switzerland), following the manufacturer's instructions. In addition, serum levels of thyroid hormones including triiodothyronine (T3), thyroxine (T4), free T3 (FT3), free T4 (FT4), and thyroid-stimulating hormone (TSH) were also determined using the same analyzer according to standard laboratory protocols.

Statistical analysis

Data analysis was done on Statistical Package for Social Sciences (SPSS) windows, version 26 (IBM SPSS Statistics, Armonk, NY). That included normality test Shapiro–Wilk was used to determine whether the studied parameters followed a Gaussian distribution. Data appeared as mean ± standard deviation (SD). Mann-Whitney was used to compare the studied groups mean. t-tests, Pearson correlations, and multiple linear regression to assess relationships between thyroid hormones and BNP levels.

Table 1. Sex group distribution between patients and controls

Groups		Patients		Controls	
		NO.	Percent%	NO.	Percent%
Sex	Female	99	76.15%	39	78.00%
	Male	31	23.85%	11	22.00%
Total		130		50	

*P-values < 0.05 were considered statistically significant

Results

Demographic data

The total number of participants was 180, divided into 130 patients and 50 controls. The distribution by sex was 99 females (76.15%) and 31 males (23.85%) in the patient group, and 39 females (78%) and 11 males (22%) in the control group are summarized in (Table1).

The mean age of female patients was 47.49 ± 7.18 years, vs the control group's mean age was 40.72 ± 7.99 years. While mean age of male patients was 46.81 ± 6.96 years, vs the control group's mean age was 46.64 ± 5.71 years as in (Table 2).

Table 2. Age group distribution between patients and controls

Groups		Patients (Mean±S.D)	Controls (Mean±S.D)
Age (year)	Female	47.49 ± 7.18	40.72 ± 7.99
	Male	46.81 ± 6.96	46.64 ± 5.71
Total		130	50

Interpretation of hormonal and bnp levels across the four groups (by gender):

TSH (Thyroid-Stimulating Hormone): Marked decrease in TSH levels in patients with hyperthyroidism, especially those with heart disease or on medication, compared to controls. This difference was highly significant ($P < 0.00001$) in both males and females, as expected due to negative feedback. T3 and T4 (Thyroid Hormones): Significant elevation in T3 and T4 in all hyperthyroid patient groups, especially those with heart complications or receiving treatment. The control group showed normal, lower levels. Males showed slightly higher mean T3 than females in patient groups. Differences were statistically significant, indicating clear hormonal over activity in these patients. FT3 and FT4 (Free Thyroid Hormones): Like total T3/T4, very high levels in patient groups, particularly those on treatment. Control group maintained low, normal values. Differences between males and females are less pronounced but still present with significant P-values. BNP (Brain Natriuretic Peptide - Cardiac Marker): Elevated BNP levels in patients with both hyperthyroidism and heart disease, and even higher than treatment. Suggests cardiac stress associated

with thyroid over activity. Both sexes showed elevated BNP when heart involvement was present. Strong statistical significance implies a real association between thyroid dysfunction and cardiac strain.

Hormonal and BNP levels analysis

TSH levels were significantly lower in hyperthyroid patients, particularly in those with heart disease or on medication ($P < 0.00001$). Also, T3 and T4 levels were elevated especially in those receiving treatment ($P < 0.00001$). FT3 and FT4 showed similar patterns of elevation in patient groups, with significant differences in comparison to controls ($P < 0.00001$). While BNP levels were markedly elevated in patients with hyperthyroidism and cardiac conditions ($P < 0.00001$), indicating increased cardiac stress associated with thyroid dysfunction.

Table 1. Hormonal and BNP levels analysis

Test	Sex	Patients			Controls	P-Value
		Hyperthyroid only	Hyperthyroid + heart disease	Hyperthyroid on Drugs		
TSH	Male	2.13 ± 1.51	0.11 ± 0.08	0.08 ± 0.07	2.68 ± 1.24	< 0.00001
	Female	2.11 ± 1.18	0.10 ± 0.09	0.08 ± 0.04	2.42 ± 1.11	< 0.00001
T3	Male	2.29 ± 0.53	6.63 ± 2.04	8.06 ± 2.29	2.23 ± 0.46	< 0.00001
	Female	2.21 ± 0.51	6.94 ± 2.35	7.90 ± 1.85	2.29 ± 0.57	< 0.00001
T4	Male	107.47 ± 33.88	227.47 ± 39.24	222.22 ± 35.64	102.43 ± 20.27	< 0.00001
	Female	105.14 ± 23.94	242.83 ± 49.65	218.43 ± 29.15	101.63 ± 22.91	< 0.00001
FT3	Male	4.60 ± 0.85	8.10 ± 1.21	8.19 ± 0.83	4.25 ± 0.67	< 0.00001
	Female	4.71 ± 0.92	8.10 ± 0.92	8.32 ± 0.93	4.40 ± 0.55	< 0.00001
FT4	Male	16.18 ± 2.84	30.98 ± 7.64	32.46 ± 8.14	15.23 ± 3.06	< 0.00001
	Female	16.64 ± 2.79	32.57 ± 9.04	31.14 ± 7.33	14.57 ± 2.90	< 0.00001
BNP	Male	116.17 ± 22.27	200.90 ± 23.64	232.12 ± 39.93	96.08 ± 15.90	< 0.00001
	Female	120.85 ± 20.87	210.74 ± 27.06	227.93 ± 37.59	99.74 ± 17.43	< 0.00001

Regression analysis

The regression model demonstrated that FT3 and T3 had the most substantial positive impact on BNP levels, indicating a direct influence of active thyroid hormones on cardiac function. TSH exhibited a strong inverse relationship, indicating potential recovery or compensatory effects in patients on medication.

Table 2. Regression analysis

Group	NO	BNP (Mean ± SD)	P-Value vs Control
Hyperthyroid only	30	283.89 ± 126.27	P = 0.00001
Hyperthyroid + Heart	50	91.09 ± 23.48	P = 0.00001
Hyperthyroid on Drugs	50	200.25 ± 59.63	P = 0.00001
Control	50	55.45 ± 26.08	—

Discussion

It was noticed that thyroid function status could affect serum BNP levels, which was mostly related to a direct excitatory effect of thyroid hormones on the release of BNP [4]. Several published studies observed the levels of BNP in hyperthyroid patients [5-11].

This study demonstrates a significant association between hyperthyroidism and elevated B-type Natriuretic Peptide (BNP) levels, highlighting the potential cardiovascular stress present even in the absence of overt heart failure symptoms. All hyperthyroid subgroups, including untreated patients, those on medication, and those with

documented heart disease showed significantly elevated levels of FT3, FT4, and T3, along with suppressed TSH levels compared to the control group. These patterns are consistent with the known pathophysiology of thyroid hormone excess and its suppressive effect on TSH through negative feedback. These findings are consistent with recent literature suggesting that hyperthyroidism leads to increased cardiac output and ventricular stretch, which stimulates BNP secretion. The observed correlations underline the potential utility of BNP in early cardiac risk assessment in hyperthyroid patients, even in the absence of overt cardiac symptoms.

Focusing on the relation between BNP and thyroid hormones, we noticed that there was significant positive relation between BNP and thyroid hormones (F.T3 and F.T4), this comes in agreement with Arikan et al. [7] who documented a positive correlation between serum BNP and thyroid hormones. \When studying BNP in relation to dysthyroid clinical state, we observed that patients with major clinical symptoms had showed higher BNP levels than those with minor complaints. This comes in accordance with the studies of Ertugrul et al. [8,21] and Schultz et al. [11]. who observed significant decrease in BNP levels, after euthyroidism was achieved. This finding supports the importance of BNP to guide therapy in subclinical hyperthyroid patients.

Conclusion

Elevated active thyroid hormones, particularly FT3 and T3, are associated to elevated BNP levels, emphasizing hyperthyroidism's cardiovascular complications. These findings emphasize the necessity of detecting thyroid dysfunction early and managing it well to reduce cardiac risk.

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