Original paper

Evaluation of Brain Natriuretic Peptide levels in Sera of Iraqi Patients with Hyperthyroidism and Hypothyroidism

Zinah Abbass Ali^*

^ College of Pharmacy, University of Babylon, Al-Hilla, Iraq.

Abstract

Background: The thyroid gland is an endocrine gland that is normally positioned in the lower neck. Cardiovascular alteration that coexist with thyroid gland diseases could trigger off the secretion of Brain natriuretic peptide from heart. Stress environment conditions and various factors have an important thumb in pathogenesis of thyroid gland diseases and may possibly eventuality lead to this reply. Volume expansion and pressure overload stimulated Brain Natriuretic Peptide secretion from the ventricular myocardium.

Aim: To evaluate the role of thyroid disorders in stimulating ventricular myocardium's to screate Brain natriuretic peptide.

Materials and Methods: Serum Brain Natriuretic Peptide determined by enzyme-linked immunosorbent assay (ELISA). About 36 Iraqi patients with primary hyperthyroidism and primary hypothyroidism and Twenty two subjects who are apparently healthy were enrolled in this study.

Results: Serum levels of BNP hormone showed a significant elevation in hyperthyroidism as compared with control ($p<0.01$) and significant decrease in hypothyroidism group compared with control group. The results revealed a significant positive correlation between T3, T4 and BNP level in patients with hyperthyroidism while there was no weighty relation between the corresponding thyroid hormones and BNP level in patients with hypothyroidism.

Discussion: A raise in cardiac output, total blood volume, left ventricular end-diastolic volume (LVEDV) and heart rate in hyperthyroid status exerts the "stress" of the cardiac muscles and could be potential stimuli for the emission of BNP.

Conclusion: the measurement of BNP is mine important in patients with hyperthyroidism compared with the cases of hypothyroidis.

Key words: Brain natriuretic peptide (BNP) and left ventricular end-diastolic volume (LVEDV).

Introduction

The a butterfly-shaped gland that situated in the lower anterior part of the neck is called the thyroid gland .It is function is to synthesize thyroid hormones that are secreted into the blood and passed to all of the tissue. Thyroid hormones help the body utilize energy, remain warm, and regulate the brain, muscles, heart and organs to work as they should [(1)].

Hyperthyroidism means overproduction of thyroid hormones while hypothyroidism refers to inability of the thyroid gland to secrete adequate level of thyroid hormone to cover the metabolic load of the body. Uncontrolled hypothyroidism lead to elevate blood pressure, impaired lipid metabolism, impairment of cognition, infertility, and neuromuscular abnormality [(2)].

*for correspondence E-mail: Zenaabbassalubaidy@yahoo.com

Thirty two amino acids is the composition of the Brain natriuretic peptide which (BNP) is a cardiac neurohormone, secreted from ventricles. Pro brain natriuretic peptide is (proBNP) a Prohormone sliced into the biologically dynamic form BNP and amino terminal portion of NT-proBNP. BNP-T and, NT-proBNP peptides secreted into the plasma (3). The incentive for the BNP secretion is a rise ventricular tension according to sodium and H2O, extension in addition to increase ending diastolic volume. It is well known that increase level of natriuretic peptides are the organize of neurohumoral, immune system ,decrease of blood pressure and plasma quantity during coordinat edaction of the brain, kidneys and blood vessel (4). Rise of heart beats, enlarge of left ventricular end-diastolic volume (LVEDV), total blood volume and cardiac output in hyperthyroid apply the "stress" of the cardiac wall and could be probable increase level of the secretion of BNP, and later enlarged serum NTpro BNP concentration (5).

Thyroid disturbance is common. Current estimation proposes that it affects 9% to 15% the woman and a lesser proportion of men [(6)].

This article studies discover the relationship between brain natriuretic peptide level and thyroid hormones in Iraqi patients with hyperthyroidism and hypothyroidism. Previous studies found increased serum levels of BNP in both hyperthyroidism and hypothyroidism**.** No previous study elucidate the relationship between BNP, T3 and T4. In this study, we tried to determine the relation between BNP and thyroid hormones level in primary various cases of thyroid dysfunction.

Methods and Patients

The study is conducted in Babylon Maternity and Pediatric Teaching Hospital in Biochemistry Department. Serum brain natriuretic peptide determined by enzyme- linked immunosorbent assay (ELISA).

From all patient the history was taken include: age, residence, smoking, drug, and surgical history, No drugs were given to those patients that may hinder with the parameter. About 36 Iraqi patients (18 with primary hyperthyroidism and 18 primary hypothyroidism). This group have no history of diabetes mellitus, inflammatory, disease hypertension and is not smoking. Data were written as Mean ± SD done by using SPSS version 18. Student′s F-test was used to determine the normality of the distribution of all variables and the significant differentiation between the groups was establish by Pearson relationship analysis. P values less than 0.05 P are considered as difference.

Results

Serum levels of hormone showed significant increase in hyperthyroidism compare with control ($p<0.01$) and significant decrease in hypothyroidism group compared with control group. The results showed the level of BNP in Hypothyroidism is (49.27±5.67), T4 (2.05 ± 0.99) and T3 (0.41 ± 0.2).BNP 88.5±7.5 in hyperthyroidism, T4 (0.41 ± 0.2) and T3 (4.8 ±2.32). As shown in table (1).

The results revealed a significant positive correlation between and T3, T4 and BNP level in patients with hyperthyroidism while there was no weighty relation between the corresponding thyroid hormones and BNP level in patients with hypothyroidism as shown in table (2), figure (1) and figure (2).

Discussion

Table- 1demonstrate the results with significant raise in the concentration of BNP in patients with hyperthyroidism group in comparison with normal group (P<0.01) and significant reduce in the concentration of BNP in patients with low level of thyroid hormone comparison among those of normal subject (P<0.01).

Table 1. Biochemical changes of hyperthyroidism and hypothyroidism and control.

	Hypothyroidism n=18	**Hyperthyroidism Group n=18**	**Control group n=22**	**Student′s F-test**
BNP **pg/ml** **Mean± SD** **Range**	49.27±5.67 (40.1-59.2)	88.5±7.5 (70 -99.5)	65.2±1.5 (62-66.9)	p < 0.05
Total T_4 µg/dl **Mean± SD** **Range**	2.05 ± 0.99 (0.56-3.399)	14.9 ±8.02 (1.48-24.5)	7.1 ±1.1 (4.47-9.05)	p < 0.05
Total T_3 ng/ml **Mean± SD** **Range**	0.41 ± 0.2 (0.11-0.70)	4.8 ±2.32 (1.40-8.10)	1.46 ±1.09 (0.96-7.67)	p < 0.05

Table 2. Correlation among BNP, T3, T4 in patients with hyperthyroidism

parameres	**Hyperthyroidism**		**Hypothyroidism**	
BNP vs T3	**r**	**p**	**r**	**p**
	0.611	0.01	0.05	0.82
BNP vs T4	0.58	0.01	0.07	0.7

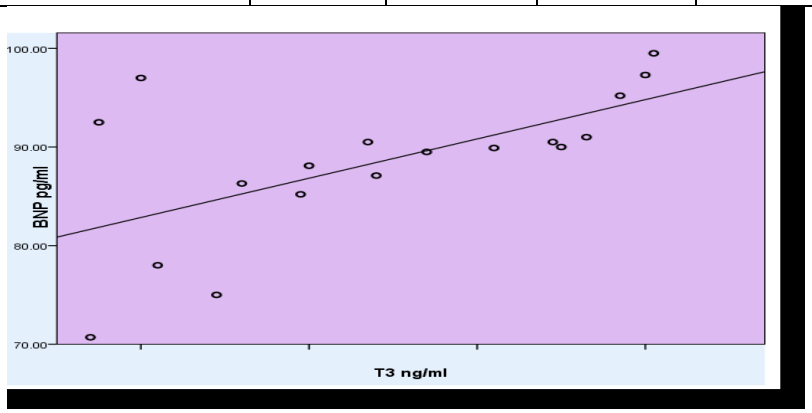

Figure 1. Correlation between T3 and BNP in patients with hyperthyroidism(r=0.611,p<0.01).

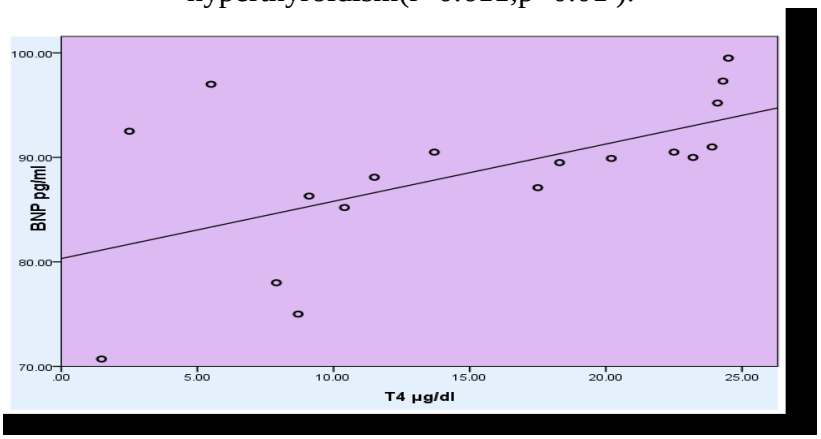

Figure 2. Relationship among T4 and BNP in patients with hyperthyroidism (r=0.58, p<0.01).

In Hyperthyroidism and hypothyroidism, cardiac functions were significantly changed. The special effects of hyperthyroidism contain hemodynamic change in heart such as low systemic vascular resistance also raise in cardiac output, heart rate, blood amount, pressure and impair heart contractility. These deviations lead to ventricular expanse and pressure overload, which might be a reason for concomitant rise in BNP concentrations. T3 and T4 encouraged liberate of BNP from both cultured a trial and ventricular myocytes a dose-dependent mode [(7)].

Araise cardiac output, totality blood volume, left ventricular end-diastolic volume (LVEDV) and heart rate in hyperthyroid status exerts the "stress" of the cardiac and could be potential stimuli for the emission of BNP, and later levels raise of the BNP [(8)].

Table (2) reveals a significant linear correlation between BNP and both T3 and T4 while there is no significant correlation between those parameters in patients with hypothyroidism .This can be attributed to the changes in the peripheral T4 to T3 in patients with hypothyroidism which may lead to changes in cardiac functions, hemodynamic factors which are induced by the concomitant increase of thyroid hormones while those changes were not significant in patients with primary hypothyroidism [(9)]. Therefore the measurement of BNP is important in patients with primary hyperthyroidism compared with the cases of hypothyroidism .Future studies are needed to further elucidate the effects of different treatment regimens of hyperthyroid patients on BNP levels of the corresponding patients.

Conclusion

The measurement of BNP is mine important in patients with hyperthyroidism compared with the cases of hypothyroidism.

References

1. Singer PA. Thyroiditis. Acute, subacute, and chronic. *Med Clin North Am*. 1991;75:61-77.
2. Boucai L, Hollowell JG, Surks MI. An approach for development of age-, gender-, and ethnicity-specific thyrotropin reference limits. *Thyroid*. 2011; 21:5-11.
3. Lapointe MC. Molecular regulation of the brain natriuretic peptide gene. Peptides. 2005; 26:944-956.
4. Hall C. Essential biochemistry and physiology of NTproBNP. Eur J Heart Fail. 2004; 6:257-260.
5. Ertugrul DT, Yavuz B, Ata N, Yalcin AA, Kucukazman M (Decreasing brain natriuretic peptide levels after treatmentfor hyperthyroidism. Endocr J. 2009; 56:1043-1048.
6. Canaris GJ, Manowitz NR, Mayor G, Ridgway EC. The Colorado thyroid disease prevalence study. *Arch Intern Med*. 2000;160: 526 –530.
7. Cini G, Carpi A, Mechanick J, Cini L, Camici M. Thyroid hormones and the cardiovascular system: pathophysiology and interventions. Biomed Pharmacother. 2009; 63:742-753.
8. Arikan S, Tuzcu A, Gokalp D, Bahceci M, Danis R Hyperthyroidism may affect serum N-terminal pro-B-type natriuretic peptide levels independently of cardiac dysfunction. ClinEndocrinol (Oxf). 2007; 67:202-207.
9. Martin Andrew Crook Clinical Biochemistry & Metabolic eighth Medicine) edition hodderarnold a hachetteuk company: 2006; 11: 164-175.