Promote Scientific Research is Our Way to Serve the Community

Hormonal Study for patients with congestive heart failure (CHF)

Saba Kh.Al-Tai[1] , Zaid M.M.Almahdawi[2]
[1] *Department of physiology ,Veterinary Medicine College ,Tikrit University ,Tikrit ,Iraq.*
[2] *Department of Biology ,College of Science ,Tikrit University ,Tikrit ,Iraq.*

ARTICLE INFO.
Keywords: Thyroid glands, Aldosterone, Congestive, Heart failure
Name: Saba Kh.Al-Tai
E-mail:
Tel:

ABSTRACT

This study aimed to evaluate the role of some hormones in congestive heart failure patients .the study included the determination of growth hormone, aldosterone hormone, and Thyriod hormones (T_3,T_4,TSH) were involved 125 samples: (75 of them satisfactory sample of patients (Male) with heart failure and 50 (Male) healthy people of the sample was used as a control. The results were as follows: a. highly significant decrease ($P<0.01$) in T3 level in patients with CHF comparison with control groups Also there are a significant decrease ($P<0.05$) in T4 level in the patients with CHF in comparison with control while serum TSH level are highly significant increase ($p<0.01$) in the patients with CHF in comparison with control groups.and a significant increase ($P<0.05$) in serum aldosterone level in CHF than in controls groups.While there were a highly significant decreases($P<0.01$) in serum Growth hormone levels in CHF patients as compared to control group.

Introduction

CVD, Cardiovascular disease is an abnormal functions of the blood vessels or Heart disease (HD). It is a generic term for a assortment of heart situation [1]. The most joint forms of HD is coronary heart disease CHD, or coronary artery disease CAD due it encompass the coronary arteries. Other kind of CVD contain hypertension [2], CHF, stroke, innate cardiovascular disorder, stiffen or stricture (atherosclerosis) of the blood vessels, inclusive the coronary arties, and other diseases of the circulatory system hypertension , high cholesterol, Diabetes, obesity, smoking, lack of exercise, family history, and advanced age are risk factors for cardiovascular disease(or heart disease) [3]

Aldosterone is produced by the adrenal cortex in outer section in (zona glomerulosa) the adrenal gland [4].

In patients with high levels of aldosterone, and to increased express of atrial MR and chronic atrial fibrillation AF, aldosterone role in pathogenesis of atrial fibrillation may collaborate in fibrosis of atrial, [5] hypertrophy of muscle and connecting unrest. Ion reshape can supplemental stimulate of aldosterone mediated rhythm-MR trouble. amendment of calcium channel express, channel of calcium and potassium beside receptor of ryanodine activity can lead to electrophysiological aberration observing in arrhythmias of ventricular [6].

Somatotropin or growth hormone polypeptide hormone contain of 191-amino acids secreted by the pituitary gland from anterior lobe. growth hormone stimulation of cellular uptake of amino acids and protein synthesis. [7]

Growth hormone called a stress hormone that elevate the concentration of free fatty acids and glucose. It also induce production of IGF-1 [8].

Growth hormone in general effects in the tissues of the body as anabolic. such as other protein hormones, [9] thyroid gland contain of two linked lobes. There are found in the front neck, beneath

the laryngeal salience [10] The thyroid gland regulator rate of use of energy sources, regulate the body's sensitivity and protein synthesis, to other hormones. It take part in these processes by during production thyroid hormones, the more effective ones being thyroxine T_4 and
Triiodothyronine T_3, which is more active [11] triiodothyronine T3 and thyroxine T4, are protuction in the thyroid gland in a molar ratio of about 1 to 7 [12] Every enzymatic step in the synthesis and excretion of T4 and T3 is regulated by thyrotropin, also known as thyroid-stimulating hormone TSH [13] The TSH test is the appropriate initial screening for thyroid dysfunction in several cardiovascular clinical entities and risk factors known to be affected by thyroid disease such as hypertension, atrial fibrillation, and dyslipidemia [14] The effects of thyroid hormones on the cardiovascular system are the most clinically useful and sensitive signs of thyroid dysfunction. Regarding pathophysiology thyroid dysfunction has essential cardiovascular consequences in myocardial contractility, peripheral hemodynamics, and heart rate [15] .This study aimed to search the relationship
Between congestive heart failure and thyroid hormones,also the role of Aldosterone in
Patiens with CHF .

Materials and Methods

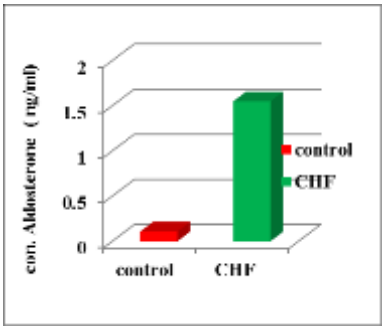
Blood samples were collected from 75 patients with congestive heart faliure, in Rizgary Teaching Hospital in City of Erbil from March 2015 to October 2015.The population of this study for male patients age range from 45 to 80 years who were selected depending on the questionnaire of this study. Patients and controls were age matched،the mean age of patients with CHF was(45.4±3.14) to (80±22.21)year which was slightly higher as compared to controls (44.4±3.14)to (80±22.21).
Determination of serum Aldosterone hormone concentration :Principle of the Assay by using kit from Monobind Inc,(USA) (www.Monobind.com)
Determination of serum Growth hormone concentration was determined by kits of American Monobind company, depending on Enzyme Linked Immunosorbant Assay (ELISA) technique [16].
Serum T3 concentration was determined by kits of American Monobind company, depending on Enzyme Linked Immunosorbant Assay (ELISA) technique [17].
Serum TSH concentration was determined by kits of American Monobind company, depending on Enzyme Linked Immunosorbant Assay (ELISA) technique [18]
Serum T4 concentration was determined by kits of American Monobind company, depending on Enzyme Linked Immunosorbant Assay (ELISA) techinque [19].

The Results: The results in this study showed in Table (1) And figure (1): The mean of serum aldosterone level was higher in CHF than controls (1.55±6.66 versus 0.108±0.167) with statistical

Table -1-

	(mean ± SD)	(mean ± SD)
Parameter	Control group	CHF group
Aldosterone (ng/ml)	0.108±0.167	1.55±6.66 *
Growth Hormone (ng/ml)	1.293 ± 0.212	0.617±0.248 **
T3(ng/ml)	115.0 ±22.3	53.24±7.10 **
T4(ng/ml)	8.58±2.04	7.69±1.95 *
TSH(ng/ml)	1.1232±0.0959	1.504±0.131 **

**high Significant$P$ <, 0.01 comparison with control
*Significant$P<0.05$ comparison with control.



significant difference (P<0.05)

Figure (1):Concentrations of Aldosterone hormone (ng/ml) in patients with CHF and the controls

The results showed in Table (1) figure (2) a significant decreases (P<0.01) in serum Growth hormone levels for CHF patients (0.617±0.248) as compared to control group (1.293 ± 0.212). group (P<0.01).

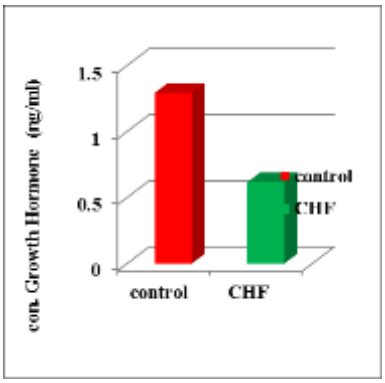
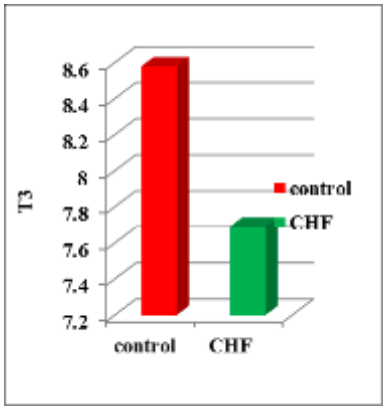


Figure (2):Concentration of growth hormone (ng/ml) in patients with CHF and the controls

Thyroid hormone profiles: in the Table (1) and in figures (3) showed a significant lower (P<0.01) in

T3 concentration in patients with CHF (53.24±7.10) ng/ml in comparison with control groups (115.0 ±22.3) ng/ml. also the same table (1) and figures (4)and (5) showed a significant decrease ($P<0.05$) in T4 concentration in patients with CHF (7.69±1.95) ng/ml in comparison with control groups (8.58±2.04) ng/ml. while TSH concentration showed high significant increase ($p<0.01$) in patients with CHF (1.504±0.131) ng/ml compared with control groups (1.1232±0.0959).



Figure(3) : T3 concentration (ng/ml) in the patients with CHF and the controls

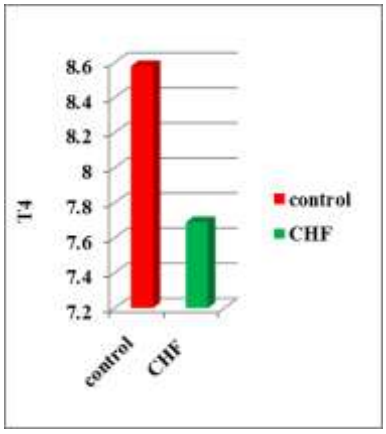


Figure (4): T4 concentration (ng/ml) in the patients with CHF and the controls.

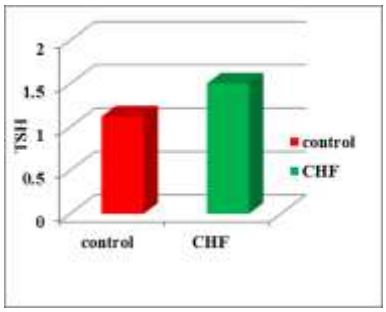


Figure (5): TSH concentration (ng/ml) in the patients with CHF and the controls.

Discussion

These results were in agreement with previous findings. Another studies found the increase of aldosterone in men with CHF, excretion rate is 5- to 6-fold higher than when compared with healthy subjects[20]

Aldosterone affects the body's capacity to control blood pressure[21]. It delivery off the signal to organs, such as the colon and kidney, it can be increase the quantity of sodium which the body send off into the Blood stream or the quantity of potassium releas in urine[22] The hormone as well reason the blood stream to reabsorbation sodium with the water that increase blood volume. All of these acts are an integral part of the increase and decrease blood vessels. [23] not directly, the hormone also helps retention the blood's electrolyte and pH levels.

[24] The RAAS also play a vital role in many non high pressure cases, and in especially in CHF and the other edematous disturbance (ascites with cirrhosis and the nephrotic syndrome) It may be reason for the increased aldosterone hormone decreased cardiac output, or the inadequate arterial circulating volume Which demand the activation of the sympathetic nervous system and the renin–angiotensin–aldosterone system leading to lateral vasoconstriction to preserving blood pressure homeostasis in spirited areas and adjust sodium and water retention to maintain blood volume [25].

These results agree with [26] who reported the changes in growth hormone in congestive heart failure patients (CHF). and do not conform with [27] who found that no effect for HGH in patients with congestive heart failure of any cardiac parameter, e.g diastolic and systolic functions or left ventricular distance.

The name "growth hormone" is misleading; stress produces somatic growth, in a process called "hormesis."

[28] The "growth hormone" involved in many processes other than growth. What is given to. It is being given to people with the purpose of making them lean and muscular, and with the hope of building stronger bones[29] HGH levels naturally decrease with age[30]. It may be reason for the decrease in Growth hormone, associated with Hypothyroidism and plasma GH , IGF (insulin-like growth factor 1) concentrations are significantly lowered[31]The effect of GH significance of the such on cardiac wall stress is prove by the changes that are observed in after correctly the defect growth hormone excretion[32].These results were in agreement with previous, findings [33] and Do not conform with results of [34]Who explained

the relationship between Hyperthyroidism Associated Congestive Heart Failure.

Thyroid hormone has an essential role in the cardiovascular homeostasis [35]. The relation between the cardiovascular system and thyroid hormone it is proven amply demon- strated in many clinical and empiricist studies[36]

Our findings were in agreement with previous findings by, [37]Who explained The mechanism for this case ,1- reduced hepatic transfer of T_4 to T_3, and private with advanced heart failure, as a result of a reduce in action of the monodeiodinase, 2-a reduce in link to serum proteins,*3*- an widened volume of delivery, and *4*- a squabby half-life. Many record document convert thyroid hormone metabolism with rduce serum T_3 levels in patients with CHF [38].Increased intensity of cardiac disease it has been shown to correlate inverse with serum total T_3 levels The cause for the decrease in serum T_3 The converted of T4 to T3 is comparison and a low serum T3 syndrome may improve in cardiac dysfunction This situation is a strong predictor of death-rate in patients with acute heart disease [39].

The hormones T3 and T4 are infact participated in the organization of oxygen uptake and function of muscular as transcription that many of the genes exact in skeletal muscle [40]

hypothyroidism also causes increases in total cholesterol and low-density lipoprotein cholesterol analogous to the rise in TSH level[41]

So the Low concentration of T4 and T3 leads to increase of TSH by negetive feedback mechanism to organized the hormone concentration in blood, pituitary gland and hypothalamus [42].

Conclusions

Congestive heart failure disease is one of the most popular diseases in Iraq .It is a common disease in men.and there is a relationship between thyriod gland, heart and renal functions. Congestive heart failure disease causing an increase in Growth hormone level.

References

1-Sutcliffe, P; Connock, M; Gurung, T; Freeman, K; Johnson, S; Ngianga-Bakwin, K; et al . (2013). "Aspirin in primary prevention of cardiovascular disease and cancer: a systematic review of the balance of evidence from reviews of randomized trials.". PLOS ONE **8** (12): e81970

2-Moran, A.E; Forouzanfar, M.H; Roth, G.A; Mensah, G.A; Ezzati, M; Murray, C.J.and Naghavi, M (2014). "Temporal trends in ischemic heart disease mortality in 21 world regions, 1980 to 2010: the Global Burden of Disease 2010 study.". Circulation **129** (14): 1483–92.

3- Mendis, S.; Puska,, P. and Norrving, B. (2011). Global atlas on cardiovascular disease prevention and control (1ed.). Geneva: World Health Organization in collaboration with the World Heart Federation and the World Stroke Organization. p. 48.

4-Feltner, C; Jones, C.D.; Cené, C.W.; Zheng, Z.J.; Sueta, C.A; Coker-Schwimmer, E.J.*et al*. (2014). "Transitional care interventions to prevent readmissions for persons with heart failure: a systematic review and meta-analysis.". Annals of Internal Medicine **160** (11): 774–84.

5-Qin ,W.; Rudolph, A.E.; Bond, B.R.; Rocha, R.; Blomme, E.A.; Goellner, J.J.et al.(2003). Transgenic model of aldosterone-driven cardiac hypertrophy and heart failure. Circ Res. ; 93: 69–76.

6- Rafiq , K.; Hitomi, H.; Nakano, D.; et al (2011) Pathophysiological roles of aldosterone and mineralocorticoid receptor in the kidney. J Pharmacol Sci ;115:1-7

7- Ranabir, S. and Reetu, K. (2011). "Stress and hormones". Indian J Endocrinol Metab 15 (1): 18–22.

8- Bartholomew ,E.F.; Martini,F. and Nath ,J.L. (2009). Fundamentals of anatomy & physiology. Upper Saddle River, NJ: Pearson Education Inc. pp. 616–617.

9- Grugni, G.; Giardino, D.; Crino, A.; Malvestiti, F.; Ballarati ,L; Di Giorgio, G .et al.(2011) Growth hormone secretion among adult patients with Prader-Willi syndrome due to different genetic subtypes. J Endocrinol Invest.; 34(7):493– 497.

10- Longo, D.; Fauci, A.; Kasper, D.; Hauser, S.; Jameson, J.and Loscalzo, J. (2012). Harrison's Principles of Internal Medicine (18th ed.). New York: McGraw-Hill. pp. 2913, 2918..

11-Szinnai, G. (2014). "Genetics of normal and abnormal thyroid development in humans". Baillière's Best Practice Research Clinical Endocrinology Metabolism **28**: 133–150.

12-Tamura, K.; Takayama, S.; Ishii, T; Mawaribuchi S.; Takamatsu, N. and Ito, M.(2015). "Apoptosis and differentiation of Xenopus tail-derived myoblasts by thyroid hormone.". J Mol Endocrinol. **54** (3): 185–92.

13-Fehrenbach, H. (2012). *Illustrated Anatomy of the Head and Neck. Elsevier. p. 158.*

14-Danzi ,S.and Klein, I.(2012). Thyroid hormone and the cardiovascular system. *Med Clin North Am*.;96(2):257-268.

15- Henry, J.D.(1996). Clinical Diagnosis and Management of laboratory Methods, WB Saunders Company, 324 .

16-Braverman, L.E.;Utigen, R.D.; Werner, E. and Ingbar's(1996). 'The Thyroid- *A Fundamental and Clinical Text'* 7th Ed. Philadelphia. Lippinscott-Raven.

17- Fisher, D.A, (1996). "Physiological variations in thyroid hormones. Physiological and pathophysiological considerations", *Clin Chem*, 42, 135-139 .

18-Barker, S.B.(1984). "Determination of Protein Bound Iodine." *Journal Biologiacl Chemistry* 173, 175. (1948).

19-Patel, B.M.; Mehta, A.A. (2012). Aldosterone and angiotensin: role in diabetes and cardiovascular diseases. Eur J Pharmacol 2012; 697: 1-12.

20-Sowers, J.R. (2009). Whaley-Connell A, Epstein M*: Narrative review:* The emerging clinical implications of the role of aldosterone in the metabolic syndrome and resistant hypertension. Ann Intern Med 150: 776–783,

21-Gross, E,; Rothstein, M,; Dombek, S. and Juknis, H.I.(2005) Efon blood pressure and the renin-angiotensin-aldosterone system infect of *spironolactone* n oligo-anuric hemodialysis patients. Am J Kidney Dis 46: 94–101,

22-Leroy, V.; De Seigneux ,S.; Agassiz ,V.;Hasler ,U.; Rafestin-Oblin, M.E., Vinciguerra et al. (2009) Aldosterone activates NF-kappaB in the collecting duct. *J Am Soc Nephrol* 20 131–144**.**

23-Wilkinson-Berka, J.L.; Tan, G.;Jaworski, K.and Miller, A.G.; (2009).Identification of a retinal aldosterone system and the protective effects of mineralocorticoid receptor antagonism on retinal vascular pathology. *Circ Res* 104*:* 124–133.

24-Ori, Y.; Chagnac, A.; Korzets, A.; Zingerman, B.; Herman-Edelstein, M; Bergman, M;et al. (2013) Regression of left ventricular hypertrophy in patients with primary aldosteronism/low-renin hypertension on low-dose spironolactone. *Nephrology, Dialysis, Transplantation* 28: 1787–1793

25-Maison, P. (2003). Chanson P. Cardiac effects of growth hormone in adults with growth hormone deficiency. *Circulation.*; 108: 2648–2652.

26-Faridi, J.; Fawcett, J., Wang, L. and Roth, R.A. (2003). Akt promotes increased mammalian cell size by stimulating protein synthesis and .inhibiting protein degradation. *Am J Physiol Endocrinol Metab*.; *285*: *E964*–E972

27-Dreifuss, P.(2002) Dilatative kardiomyopathie und wachstumshormon (Dilated cardiomyopathy and growth hormone). *Z Kardiol.* ; 91: 973–977.

28-Swerdlow, ,A.J.; Higgins, C.D.; Adlard, P. and Preece ,M.A.(2002). Risk of cancer in patients treated with human pituitary growth hormone in the UK, 1959-85: a cohort study. *Lancet*.;360:273-7.

29-Bell, J.; Parker; K.L.;Swinford, R.D.; Hoffman, A.R.; Maneatis, T and Lippe B (2010). Long-term safety of recombinant human growth hormone in children. *J Clin Endocrinol Metab*.;95:167-77.

30-Akin, F.; Yaylali, G.F.;, Turgut, S.; et al. (2009). Growth hormone/insulin like growth factor axis in patients with subclinical thyroid dysfunction. Growth Horm IGF Res,;19, 252 255

31-Mills, J,L.(2004). Schonberger LB, Wysowski DK, Brown P, Durako SJ, Cox C, et al. Long-term mortality in the United States cohort of pituitary-derived growth hormone recipients. *J Pediatr*. ;144:430-6.

32-Mayer, O.; Simon, J.; Filipovský, J.; Plásková ,M.and Pik R(2006).Hypothyroidism in coronary heart disease and I ts relation to selected risk factors. Vasc Health Risk Manag,;2(4):499–506. Available at: http://www.Pubmedcentral .nih.gov/articlerender.cgi?artid=1102697. Accessed April 16, 2014.

33-Papi, G. Chesi, G.; Corsello, S.Ac.; Di Donato, C.a; Milite, M. Ta; Vittoria Ciardullo, et al. (*2009)* "Hyperthyroidism Associated Congestive Heart Failure: A Case-Control Study" *Int J Endocrinol Metab;2: 86-94.*

34-Rodondi, N.; den Elzen ,W.P,; Bauer, D.C.; Cappola, A.R; Razvi, S.; Walsh J.P,et al (2010). Subclinical hypothyroidism and the risk of coronary heart disease and mortality. JAMA; 304: 1365-74.

35-Klein ,I .;and Danzi, S.(2007). Thyroid disease and the heart. Circulation 116\17251735.

36-Nanchen, D.; Gussekloo, J.; Westendorp, R.G.; Stott, D.J.; Jukema, J.W; Trompet ,S.et al. (2012). Subclinical thyroid dysfunction and the risk of heart .failure in older persons at high cardiovascular risk. Journal of Clinical Endocrinology and Metabolism :97 852–861

37-Razvi, S, Ingoe, L.; Keeka G,; Oates, C.; McMillan C.and Weaver JU.(2007). The beneficial effect of L-thyroxine on cardiovascular risk factors, endothelial function, and quality of life in subclinical hypothyroidism: randomized, crossover trial. J Clin Endocrinol Metab ; 92: 1715-23.

38-Collet, T.H; Gussekloo, J; Bauer, D.C; den Elzen, W.P; Cappola, A.R; Balmer, P, et al (2012); Thyroid Studies Collaboration. Arch Intern Med; 172: 799-809.

39-Cooper, D.S.; Biondi, B.(2012). Subclinical thyroid disease. Lancet ; 379: 1142-54

40-Jorde, R..; Waterloo, K.; Storhaug, H.; Nyrnes, A.; Sundsfjord, J. J. and Enssen, T.G.(2006). Neuropsychological function and symptoms in subjects with subclinical hypothyroidism and the

effect of thyroxine treatment. J Clin Endocrinol Metab; 91:145-53.
41- Szinnai, G. (2014). "Genetics of normal and abnormal thyroid development in humans". Baillière's Best Practice Research Clinical Endocrinology Metabolism **28**: 133–150.

دراسة هرمونية لمرضى فشل القلب الاحتقاني

صبا خيرالدين الطائي[1] ، زيد محمد مبارك المهداوي[2]

[1]قسم الفسلجة ، كلية الطب البيطري ، جامعة تكريت، تكريت ، العراق

[2] قسم علوم الحياة ، كلية العلوم ، جامعة تكريت ، تكريت ، العراق

الملخص

هدفت هذه الدراسة الى تقييم دور بعض الهرمونات في مرضى فشل القلب الاحتقاني. تضمنت الدراسة تقدير تركيز هرمون النمو، الالدوستيرون،وهرمونات الغدة الدرقية في مصل دم 125 عينة: 75 عينة منهم لمرضى فشل القلب الاحتقاني (رجال)، 50 عينة منهم للاصحاء (رجال) استخدمت كمجموعة سيطرة .وكانت النتائج كالاتي: وجود ارتفاع معنوي ($p<0.05$) فيى تركيز هرمون الالدوستيرون في مصل دم مرضى فشل القلب الاحتقاني مقارنة مع مجموعة السيطرة (الاصحاء)، وجود انخفاض معنوي عالي ($p <0.01$) في تركيز هرمون النمو في مصل دم مرضى فشل القلب الاحتقاني مقارنة مع مجموعة السيطرة (الاصحاء)، وجودانخفاض معنوي عالي ($p < 0.01$) في مستويات T_3 في مصل دم مرضى فشل القلب الاحتقاني مقارنة مع مجموعة السيطرة (الاصحاء)، كما وجد انخفاض معنوي ($p<0.05$) في مستويات T_4– في حين وجد ارتفاع معنوي عالي ($p<0.01$) في مستوياتTSH في مصل دم مرضى فشل القلب الاحتقاني مقارنة مع مجموعة السيطرة (الاصحاء).