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Low T3 Syndrome in Chronic Heart Failure a Correlational Study

Dr. Shweta Sureshbabu Saritha[1*], Dr. E. Dhandapani[2], Dr. B. Kalaiselvi[3]

[1]PG Resident in Medicine, Sree Balaji Medical College, Chennai India
[2]BSc, MBBS, MD, Professor & Head of the Department Of Medicine, Sree Balaji Medical College, Chennai India
[3]MD,DGO, Professor & Head of The Department Of Pharmacology, ACS Medical College, Velappan Chavadi, Chennai India

***Corresponding author:** Dr. Shweta Sureshbabu Saritha
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Abstract | **Original Research Article**

Thyroid abnormalities are common in chronic heart failure. Severity of heart failure rises by several fold in patient with thyroid dysfunction. ***Aims and objectives:*** The purpose of this prospective study is to determine the patients of chronic heart failure by clinical and investigational methods. To screen the chronic heart failure patients by subjecting them to thyroid profile. To find an association between Low T3 syndrome and chronic heart failure. To determine the severity of cardiac failure in Low T3 syndrome patients. ***Method:*** it is hospital based study of 50 patients, which was conducted in Sree Balaji Medical College. The material for study is formed by adult patient with chronic heart failure between October 2017 to august 2018 fulfilling the inclusion and exclusion criteria. ***Conclusion:*** There is a significant percentage of chronic heart failure patients having low T3 as biochemical parameter.
Keywords: chronic heart failure, ,systolic blood pressure, diastolic blood pressure, segmental hypokinesia, global hypokinesia, PR interval, low t3 syndrome.

INTRODUCTION

Cardiovascular system is one of the most important targets on which thyroid hormone act [1, 2]. More than 80% of biologically active hormone T3 derived from peripheral conversion of T4 secreted by thyroid gland [3]. Clinical and experimental evidence shows T3 plays major role in modulating cardiac contractility and heart rate as well as arterial peripheral resistance [1, 2]. T3 actions were carried out by binding to specific nuclear receptor which regulates gene encoding the functional and structural cardiac proteins, direct, extranuclear, nontranscriptional effects.

The pathophysiological mechanism for low T3 is reduced enzyme activity of 5' monodeiodinase responsible for converting T4 to T3 in peripheral tissues. Low T3 syndrome is euthyroid sick syndrome.

MATERIALS AND METHODS

Study design: prospective and cross sectional study
Sample size: 50
Year of study: 2017-2018
Place: Sree Balaji medical college and hospital, Chennai

Inclusion criteria
Patients with chronic heart failure

Exclusion criteria
- Included clinical evidence of sepsis or cachexia
- Concomitant presence of any predominant severe systemic disease including severe anaemia
- Hb% <5g%
- Other major surgical procedures performed before or within 6 months after the time of thyroid sampling

RESULTS

Table showing prevalence of hypothyroidism, low T3 and CHF only in present study

Group	Number of patients	Percentage (%)
Hypothyroid	29	58
Low T3	10	20
CHF Only	11	22
Total	50	100

Table showing mean age and duration of symptoms in present study

Parameter	Hypothyroid (n=29)	Low T3 (n=10)	CHF Only (n=11)	P-Value
	Mean	Mean	Mean	
Mean Age (years)	54.90±5.49(SD)	60.50±6.15(SD)	59.91±5.99(SD)	< 0.01
Duration of symptoms (months)	2.80± 2.24(SD)	3.85±1.63(SD)	5.64± 6.63(SD)	

Table showing age and sex distribution according to groups in present study

Age groups(yrs)	No. Patients (%)	Hypothyroid (n=29)		Low T3 (n=10)		CHF only (n=11)		Total (n=50)	
		Male	Female	Male	Female	Male	Female	Male	Female
45-50	7(14%)	6	0	0	0	1	0	7	0
50-55	9(18%)	7	0	1	0	0	1	8	1
55-60	16(32%)	5	5	2	1	2	1	9	7
60-65	11(22%)	1	3	1	2	2	2	4	7
65-70	7(14%)	1	1	2	1	1	1	4	3
Total	50	20	9	6	4	6	5	32	18

Table showing mean pulse rate in different groups in present study

Groups	Mean pulse rate (per minute)
Hypothyroid (n=29)	94.9± 4.3 (SD)
Low T3 (n=10)	104± 6.9 (SD)
CHF Only (n=11)	90.9± 4.2 (SD)
Total (n=50)	95.8± 6.5 (SD)

Table showing mean blood pressure in different groups in present study

Parameter	Group	Mean BP (in mm of Hg)	P value
Systolic blood pressure	Hypothyroid (n=29)	122.6± 9.0 (SD)	< 0.215
	Low T3 (n=10)	131.2± 20.8 (SD)	
	CHF Only (n=11)	125.8± 14.0 (SD)	
	Total (n=50)	125.0± 13.3 (SD)	
Diastolic blood pressure	Hypothyroid (n=29)	78.5± 8.0 (SD)	< 0.093
	Low T3 (n=10)	84.6± 12.4 (SD)	
	CHF Only (n=11)	84.1± 8.0 (SD)	
	Total (n=50)	81.0± 9.3 (SD)	

Table showing mean PR Interval in different groups in present study

PR Interval	Mean PR interval (in seconds)
Hypothyroid (n=29)	0.16± 0.027 (SD)
Low T3(n=10)	0.21± 0.023 (SD)
CHF Only (n=11)	0.15± 0.022 (SD)

(P <0.000)

DISCUSSION

The mean age of low T3 chronic heart failure patients was higher [60.50±6.15(SD) years], Fatigue and generalized weakness, dyspnoea on exertion, swelling of feet, cold intolerance, hair loss, hoarse voice and decrease libido were common symptoms of low T3 chronic heart failure, Alcoholism was higher in patients with low T3 chronic heart failure (80%). Higher number of diabetes mellitus patients were seen in low T3 chronic heart failure (50%). Higher number of hypertensives was seen in low T3 chronic heart failure patients. Cardiomyopathy was most common etiology for chronic heart failure patients (42%) and was common in the age group of 55-60 years (38%) in present study. Idiopathic etiology was common in the age group of 60-65 years (63%). Hypertensive heart disease as etiology was common with age group of 45-50 years (42.8%). IHD etiology was common with age

group of 55-60 years (36.3%). Idiopathic etiology was common etiology with low T3 chronic heart failure patients (60%) and all patients was seen in the age group of 55-65 years. The mean pulse rate was higher in low T3 chronic heart failure group [104±6.9 (SD) beats/min]. The systolic blood pressure was high in low T3 chronic heart failure group [131±20.8 (SD) mm of Hg] and diastolic blood pressure was also higher in low T3 chronic heart failure groups [84.6±12.4 (SD) mm of Hg]. S3 heart sound was present in more number of patients with low T3 chronic heart failure (60%). The mean blood urea level was higher in low T3 chronic heart rate group [74.2±18.9 (SD) mg/dl]. The mean serum creatinine levels were higher in low T3 chronic heart failure group [2.3±0.5 (SD) mg/dl]. The estimated creatinine clearance was lower in low T3 chronic heart failure group [25.8±8.5 (SD) ml/min]. Mean serum LDL was lower in patients with low T3 chronic heart failure group [104.4±3.7(SD) mg/dl]. The mean serum HDL was lower in patients with low T3 chronic heart failure [50.3±8.0(SD)mg/dl]. The mean serum VLDL was higher in patients with low T3 chronic heart failure [29.3±22.8(SD)mg/dl]. The mean serum total cholesterol was lower in patients with low T3 chronic heart failure [181±8(SD)mg/dl]. The mean PR interval is more prolonged in low T3 chronic heart failure group [0.21±0.023(SD)sec]. The systolic dysfunction on 2D Echo was more in hypothyroid chronic heart failure group (31.03%), diastolic dysfunction on 2D Echo was more in low T3 chronic heart failure group (30%) and pericardial effusion was seen in lower number of patients with low T3 chronic heart failure (10%).Global hypokinesia was seen in lesser number of patients with low T3 chronic heart failure (30%).Segmental hypokinesia was seen in more number of patients with low T3 chronic heart failure (3%). The mean ejection fraction was 36.78±5.08 (SD) % in patients with chronic heart failure in present study. The Mean ejection fraction was lower in low T3 chronic heart failure [34.8±3.293 (SD) %].The high pulmonary artery systolic pressure was seen in more number of patients in low T3 chronic heart failure (70%)

Conclusion

There is significant percentage of chronic heart failure patients having low T3 alone as biochemical parameter. It is important to recognize this condition in patients with chronic heart failure as it is associated with increased severity of heart failure, increased in evidence of renal failure which may need additional support of thyroid hormone administration to have a better outcome in patients with chronic heart failure.

References

1. Polikar R, Burger AG, Scherrer U, Nicod P. The thyroid and the heart. Circulation. 1993 May 1;87(5):1435-41.
2. Klein I, Ojamaa K. Thyroid hormone and the cardiovascular system. New England Journal of Medicine. 2001 Feb 15;344(7):501-9.
3. Pilo AL, Iervasi GI, Vitek FR, Ferdeghini MA, Cazzuola FR, Bianchi RO. Thyroidal and peripheral production of 3, 5, 3'-triiodothyronine in humans by multicompartmental analysis. American Journal of Physiology-Endocrinology and Metabolism. 1990 Apr 1;258(4):E715-26.
4. Franklyn, J. A., Gammage, M. D., Ramsden, D. B., & Sheppard, M. C. (1984). Thyroid status in patients after acute myocardial infarction. *Clinical science*, *67*(6), 585-590.
5. Wiersinga WM, Lie KI, Touber JL. Thyroid hormones in acute myocardial infarction. Clinical endocrinology. 1981 Apr;14(4):367-74.
6. Hamilton MA, Stevenson LW, Luu M, Walden JA. Altered thyroid hormone metabolism in advanced heart failure. Journal of the American College of Cardiology. 1990 Jul 1;16(1):91-5.