ISSN: 0975-3583,0976-2833 VOL14, ISSUE 06, 2023

Assessment of the Correlation Between Subclinical Thyroid Dysfunction and Heart Failure: A Clinical Study

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Abstract

Background: We investigated whether there was a connection between due to the thyroid hormone's significant influence on the circulatory system, both subclinical thyroid disease and heart failure. **Material and Methods:** The study included 200 people, ranging in age from 45 to 75. Weight, height, and BMI are used to compute body mass index (BMI). measurements were acquired as part of a general physical examination. We measured the patient's heart rate, hip to waist ratio, and hip to waist ratio, and blood pressure. Fasting TSH, Free T3, and Free T4 concentrations were assessed using an ELISA. Echocardiography in The patient was assessed using M-Mode, 2D, and Doppler. **Results:** The subclinical hyperthyroidism group that was not receiving treatment showed no discernible improvements in any of the indicators after three months or six months of follow-up. TSH remains constant at three months but drops at six months while FT3 falls, FT4 rises, E/F rises, LVID rises, Mitral E rises, Mitral A rises. **Conclusion:** In overt hyperthyroidism, HDL levels peaked at 44.16 mg/dl, whereas in overt hypothyroidism, they dropped to 40.37 mg/dl. Subclinical hyperthyroidism (35.33 mg/dl) and overt hypothyroidism (23.18 mg/dl) had the greatest and lowest VLDL levels, respectively.

Keywords: Subclinical thyroid dysfunction, Heart Failure, hyperthyroidism, TSH, cardiovascular impairment.

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Introduction

An abnormality in the heart's structure or function is known as heart failure that stops it from pumping blood quickly enough to satisfy the demands of the body's metabolising tissues, either without raising filling pressures or only when they are already high.[1]

Clinically speaking, a syndrome known as HF is characterised by patients present with common signs like fatigue, ankle edoema, and breathlessness as well as warning signs like increased pulmonary crackles, an improper apex beat, and jugular venous pressure brought on by an anomaly in the heart's structure or function. As a result of myocardial disease, this often leads to systolic ventricular dysfunction. Additionally, anomalies in the heart's there may be a number of abnormalities, but valves, pericardium, endocardium, conduction, and heart rhythm can all contribute to HF. The underlying heart condition must be identified for therapeutic purposes as well. The specific pathology has an impact on the treatments used, such as tailored pharmacological therapy for LV systolic failure or valve surgery for valvular illness.

Relevant thyroid hormone's impact on the cardiovascular system.[2] Numerous symptoms and indicators of overt hyperthyroidism and hypothyroidism, as well as the resulting hemodynamic abnormalities, are brought on by the cardiovascular and vascular systems are

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 06, 2023

affected more or less by thyroid hormone, accordingly. Recently, it has developed into more and more clear that subclinical thyroid dysfunction may have an impact on the circulatory system and raise cardiovascular risk. It is becoming more and more obvious that cardiovascular impairment can result from both thyroid hormone metabolism is impacted by both cardiovascular illness can be acute or chronic.

The majority cellular and molecular mechanisms underlying thyroid hormone's cardiovascular actions have been identified. Thyroid hormone may affect cardiac myocytes in both genomic and nongenomic ways, as seen in Figure 1. The transcriptional activation or repression of particular target genes that encode both structural and functional proteins mediates the genomic effects of thyroid hormone.[3-5]

Despite the fact that the bulk of these disorders seem to affect the cardiovascular system's susceptibility to adrenergic stimulation less, the cardiovascular symptoms connected to hyperthyroidism and hypothyroidism resemble conditions with respectively, an alteration in the level of adrenergic activity.[6,7]

The circulatory system is also significantly impacted by thyroid hormone. Significantly less to reduce peripheral vascular resistance, vascular smooth muscle cells are encouraged to relax.[8-10]

A thyroid hormone deficiency may make HF episodes more likely. Experimental research has demonstrated that hypothyroidism results in cardiac shrinkage as a result of increased and decreased MHC expression. Hypothyroidism also results in ventricular chamber enlargement and impaired cardiac blood flow.[11-13]

The current study set out to determine if heart failure and subclinical thyroid abnormalities are related to identify and treat subclinical thyroid dysfunction in heart failure at an early stage.

Methodology

The desired at Teerthanker Mahaveer Medical College in Moradabad, a study was conducted. Inquiries about patients were made between January 2021 and May 2023. The study's participants were those who presented with heart failure in outpatient and medical crises. 200 patients between the ages of 45 and 75 were ultimately selected through successive sampling, randomly assigned using simple randomization, and then underwent interventional follow-up with Thyroid Profile and Echocardiography at 3 and 6 months. The patients were chosen following written, full consent.

Inclusion criteria

Each of the patients, whose ages vary from 45 to 75, suffers from heart failure.

Exclusion criteria

1. Individuals with pre-existing diagnoses of hyper- and hypothyroidism.
2. Patients taking thyroid-impairing drugs such as lithium, amiodrone, interferon, radioiodine, Interleukin-2 and inhibitors of tyrosin kinase.
3. people on levothyroxine or anti-thyroid drugs.

The results of the comprehensive history, which covered the family history, personal history, dietary history, drug history, general physical examination, and systemic examination, were recorded on a proforma. Information about height and weight is used to calculate body mass index. (BMI). Obtained as a result of a general physical examination. The weight was measured in kilogrammes and was accurate to the nearest 0.5 kilogrammes using a common weighing scale. The subject's shoes and socks were taken off before being measured, and their height was recorded while they were standing.

While seated, the appropriate size cuff was used to measure the right upper limb blood pressure. The radial pulse was measured with the subject lying flat. We looked at personality, rhythm, and rate.

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Breathing rate, BMI, and waist to hip ratio were noted. Each of the selected subjects performed an overnight fast of 10 to 12 hours. Blood samples taken while fasting were taken in the morning.

Fasting TSH, Free T3, and Free T4 concentrations were assessed using an ELISA. Echocardiography in The patient was assessed using M-Mode, 2D, and Doppler.

Statistical analysis: The study's data were traditional analytical statistics 0.05 or less was considered to be a significant p value. when using to evaluate the data.

RESULTS

200 heart failure patients took part in the trial at Teerthanker Medical College & Research Centre, Moradabad.

Table 1: Age Distribution

Age group	No. of patients	Percentage
45-54	44	22.0%
55-64	81	40.5%
65-75	75	37.5%

The majority of patients in the study group, 81 (40%) were in the 55-64 age range, followed by 75 (37.5%) patients in the 65-75 age range, and 44 (22%) patients in the 55-64 age range.

Table 2: Sex Distribution

	No. of patients	Percentage
Male	114	57%
Female	86	43%

200 patients were treated, 114 (57%) were male, and 86 (43%) were female.

Table 3: Prevalence of Thyroid Dysfunction In Heart Failure Patients

Type of Thyroid disorder	Number of patients	Percentage (%)
Sub - clinical hypothyroidism	24	12%
Subclinical Hyperthyroidism	6	3%
Overt hypothyroidism	16	8%
Overt hyperthyroidism	6	3%
Total no. of patients of heart failure with thyroid dysfunction	52	26%

Out of 200 patients who presented with heart failure at Moradabad, Teerthanker Mahaveer Medical College, 24 (12%) had subclinical hypothyroidism, while 3% had preclinical hyperthyroidism. Six (3%) cases of overt hyperthyroidism and 16 (8%) cases of overt hypothyroidism were also present.

Table 4: prevalence of hypertension and diabetes in study sample

	Diabetes mellitus	Hypertension	IHD
No. of cases	64	113	120
Percentage	32%	56.5%	60%

64 patients (32%) had type 2 diabetes mellitus in the study group. while 113 patients (56.5%) had hypertension. Ischemic heart disease affected 120 patients, or 60% of the total.

Table 5: lipid profile in study sample

	Total cholesterol (Mean±SD)	Serum Triglyceride (Mean±SD)	HDL (Mean± SD)	VLDL (Mean±SD)	LDL (Mean±SD)
Subclincial hypothyroidism	184.79± 49.92	168.88± 47.42	42.66± 8.98	25.77±9.54	162.90±48.55
Subclinical hyperthyroidism	172.50± 47.10	146.67± 44.50	42.33± 5.24	35.33±8.90	157.50±44.06

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Overt hypothyroidism	210.75± 53.68	176.94± 40.02	40.37± 7.35	23.18±8.00	172.56±55.11
Overt hyperthyroidism	161.00± 69.58	136.00± 68.29	44.16± 10.47	31.20±13.65	148.53±59.92

Each patient's initial lipid profile was completed. In the subclinical hypothyroidism group, the mean total cholesterol value was 184.79 mg/dl, STG were 168.88 mg/dl, HDL were 42.66 mg/dl, VLDL were 25.77 mg/dl, and LDL were 162.90 mg/dl. In the subclinical hyperthyroidism group, the mean total cholesterol value was 172.50 mg/dl, STG were 146.67 mg/dl, HDL were 42.33 mg/dl, VLDL were 35.33 mg/dl, and LDL were 157.50 mg/dl. In the group of people with overt hypothyroidism, the average level of total cholesterol was 210.75 mg/dl, STG were 176.94 mg/dl, HDL were 40.37 mg/dl, VLDL were 23.18 mg/dl, and LDL were 172.56 mg/dl. In the group of people with overt hyperthyroidism, the average level of total cholesterol was 161 mg/dl, STG were 136 mg/dl, HDL were 44.16 mg/dl, VLDL were 31.20 mg/dl, and LDL were 148.53 mg/dl.

Table 6: comparison of thyroid profile and 2D echocardiography in subclinical hypothyroidism in cases at baseline, 3 months & 6 months of treatment

Parameter	At baseline (mean)	At 3 months (mean)	'p' value	At 6 months (mean)	'p' value
TSH (µ I.U.)	9.2	6.4	0.000	4.16	0.000
FT3(pg/ml)	1.94	2.01	0.024	2.12	0.000
FT4(ng/dl)	1.14	1.37	0.000	1.45	0.000
LVEF (%)	43.00	46.08	0.001	48.92	0.006
LVIDd (cm)	5.88	5.83	0.626	5.69	0.195
Mital E (cm/sec)	66.75	71.25	0.035	73.67	0.085
Mitral A (cm/sec)	83.00	79.58	0.084	75.17	0.028
E/A	0.95	0.93	0.917	1.02	0.450

0.05 was considered a significant p value.

The modification of FT3, FT4, LVEF, and Mitral In comparison to the baseline, a velocity was substantial at 3 months and 6 months. Although LVIDd decreased and Mitral E and E/A increased, the results were not statistically significant.

Table 7: comparison of thyroid profile and 2D echocardiography in subclinical hypothyroidism in controls at baseline, 3 months & 6 months of treatment

Parameter	At baseline (mean)	At 3 months (mean)	'p' value	At 6 months (mean)	'p' value
TSH (µ I.U.)	8.86	8.99	0.253	8.89	0.268
FT3(pg/ml)	2.32	2.23	0.335	2.17	0.332
FT4(ng/dl)	1.50	1.43	0.001	1.41	0.001
LVEF (%)	44.17	43.58	0.002	43.25	0.001
LVIDd (cm)	5.97	5.96	0.668	5.97	0.971
Mital E (cm/sec)	72	70.92	0.090	71.17	0.318
Mitral A (cm/sec)	94.58	94.92	0.457	94.17	0.720
E/A	0.76	0.74	0.550	0.75	0.790

0.05 was considered a significant p value.

While the other parameter changes were not significant, at 3 and 6 months, there were significant changes in FT4 and LVEF, indicating disease progression and decreasing cardiac function.

Table 8: comparison of thyroid profile and 2D echocardiography in subclinical hyperthyroidism in cases at baseline, 3 months & 6 months of treatment

Parameter	At baseline (mean)	At 3 months (mean)	'p' value	At 6 months (mean)	'p' value
TSH (µ I.U.)	0.12	1.16	0.019	1.26	0.016

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FT3(pg/ml)	2.13	1.93	0.151	1.86	0.235
FT4(ng/dl)	1.23	1.16	0.412	1.3	0.467
LVEF (%)	45.00	48.0	0.035	50	0.013
LVIDd (cm)	5.90	5.76	0.044	5.63	0.018
Mital E (cm/sec)	81.33	84.67	0.199	88	0.109
Mitral A (cm/sec)	79.33	77.33	0.742	75	0.420
E/A	1.12	1.13	0.959	1.22	0.412

0.05 was considered a significant p value.

The changes in the other parameters after 3 months and 6 months were not significant, however the changes in the parameters like TSH, LVEF, and LVIDd were significant at those times.

Table 9: comparison of thyroid profile and 2D echocardiography in subclinical hyperthyroidism in controls at baseline, 3 months & 6 months of treatment

Parameter	At baseline (mean)	At 3 months (mean)	'p' value	At 6 months (mean)	'p' value
TSH (µ I.U.)	0.26	0.26	1.000	0.233	0.428
FT3(pg/ml)	2.14	1.80	0.416	1.80	0.146
FT4(ng/dl)	1.07	1.05	0.786	1.10	0.755
LVEF (%)	51.33	52.67	0.529	52	0.678
LVIDd (cm)	5.70	5.90	0.442	5.93	0.439
Mital E (cm/sec)	77	79.33	0.118	78	0.225
Mitral A (cm/sec)	78	78.0	0.728	79	0.691
E/A	1.08	1.11	0.446	1.08	0.955

0.05 was considered a significant p value.

After three months and six months of follow-up, the subclinical hyperthyroidism group that was not getting therapy did not experience any appreciable changes in any of the parameters. FT3 decreases, FT4 increases, E/F increases, LVID increases, Mitral E increases, Mitral A increases, and TSH stays the same at three months but declines at six months.

DISCUSSION

The study comprised 200 participants with heart failure. The majority of patients, 81 (40%) were between the ages of 55 and 64, followed by 75 (37.5%) between the ages of 65 and 75 and 44 (22%) between the ages of 55 and 64.

Of the 200 participants in our research who had complete heart failure There was subclinical hypothyroidism found in 12% of cases and subclinical hyperthyroidism in 3% of cases. There was overt hypothyroidism in 8% of cases and overt hyperthyroidism in 3%. Subclinical and overt hyperthyroidism followed, with subclinical hypothyroidism being more prevalent than overt hypothyroidism. Similar findings were found in a few study efforts by Hollowell JG et al and Surks MI et al.[14,15]

According to a different study, subclinical hypothyroidism (SH), which typically affects 1–10% of individuals, is a common condition.[16,17] 32% of patients in the study group 56.5% of patients had excessive blood pressure and type 2 diabetes mellitus. 60% of people had ischemic heart disease as their underlying condition.

TSH considerably decreased from 9.2 to 4.16 (p 0.000) in cases with subclinical hypothyroidism, while FT3 dramatically raised from 1.94 to 2.215 (p 0.000). after 3 and 6 months of treatment, according to a comparison of the thyroid profile and 2D echocardiography data. From 1.94 to 2.215, FT4 rose (p 0.00). (p = 0.06) The LVEF increased to 48.92% from 43%. LVIDd decreased from 5.88 to 5.69 cm, Mitral E increased from 66.75 to 73.67, and E/A increased from 0.95 to 1.02 despite the fact that the statistics were not statistically significant. Our results agreed with those from the Mishra TK et al. study.[18] They learned that the Deficits in diastolic function can be reversed a year after

ISSN: 0975-3583,0976-2833 VOL14, ISSUE 06, 2023

beginning thyroxine medication. Recent studies suggest that subclinical hypothyroidism may cause anomalies in the left ventricle's ability to contract and relax. Biondi et al. had similar outcomes. M. Park et al,[19] similarly found that coronary angiographic abnormalities progressed more quickly against those whose TSH levels were strictly kept within the normal range in hypothyroid persons with TSH values in the subclinical hypothyroidism range. The diastolic features are determined by the level of cytosolic calcium that is ATP-dependent and sarcoplasmic reticulum-controlled. Thyroid hormones impact calcium transport,[20] Because of this, diastolic dysfunction may occur in patients with subclinical hypothyroidism. This diastolic dysfunction may indicate systolic dysfunction.[21] Recent studies suggest that the LV's People with heart disease may have impaired systolic and diastolic function. subclinical hypothyroidism. Exercise-related reduction in LV ejection fraction was documented by Bell and co.[22] After using thyroxine, this EF abnormality was fixed. Forfar also observed a little increase exercise-induced increases in ejection fraction and a noticeable improvement after thyroxine medication.[23]

15 (37.5%) of the patients research by Karki P et al,[24] showed diastolic dysfunction. Only one patient displayed a pseudo-normal pattern, whereas 14 of them displayed anomalies related to poor relaxation. 13 of them returned to normal with replacement therapy, whereas the Patient with pseudo-normal Diastolic dysfunction of grade 2 became dysfunction of grade 1. There was still Grade 1 diastolic dysfunction (impaired relaxation) in one patient. The outcomes corresponded with those of our investigation.

After 6 months, the subclinical hypothyroidism non-treated group's thyroid profile and 2D echocardiography results showed an increase in FT4 and a decrease in TSH (p 0.001), respectively. As well as a drop in LVEF from 44.17 to 43.25% (p 0.001), which indicates disease progression and worsening cardiac function in the non-treated group. In the subclinical hyperthyroidism group, once the thyroid profile and 2D echocardiography have been compared, treatment, TSH, LVEF, and LVIDd have improved (p 0.05). Additionally, the mitral E velocity and E/A showed improvements, although the p value was not significant. With the exception of a further reduction in TSH and a slight rise in FT4 with an insignificant p value, the subclinical hyperthyroidism group that was not receiving therapy did not experience any significant changes in any of the parameters after six months of follow-up.

CONCLUSION

In overt hyperthyroidism, HDL levels peaked at 44.16 mg/dl, whereas in overt hypothyroidism, they dropped to 40.37 mg/dl. Subclinical hyperthyroidism (35.33 mg/dl) and overt hypothyroidism (23.18 mg/dl) had the greatest and lowest VLDL levels, respectively. Despite the fact that our investigation produced important findings. Results need to be verified in larger trials with due of the limited size heart failure patients who participated in the experiment.

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