ORIGINAL ARTICLE

Thyroid-Stimulating Hormone and Its Impact on Coronary Artery Disease and Left Ventricular Function

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Abstract

Background: ***Globally, morbidity and mortality are primarily caused by coronary artery disease (CAD). Thyroid dysfunction is one of the cardiovascular risk factors that are frequently linked to CAD. The impact of thyroid-stimulating hormone (TSH) on left ventricular (LV) function and coronary artery disease (CAD) remains underexplored.***

Objectives: ***To examine the association between serum levels of thyroid stimulating hormone (TSH), the existence and degree of coronary artery disease (CAD) in patients referred for coronary angiography, and the effect of this relationship on left ventricular function.***

Methods: ***This cross-sectional analytic study was conducted on 50 CAD patients performing coronary angiography. Patients were categorized according to TSH values and the existence of severe coronary artery stenosis. Echocardiographic parameters, SYNTAX score, and TSH levels were assessed.***

Results: ***In comparison to the non-significant CAD group, the significant CAD group had a higher SYNTAX score (22.07 ± 13.6, $p < 0.001$) and a lower LV ejection fraction (EF) (56.0 ± 6.67, $p=0.020$). In the substantial CAD group, TSH levels were significantly higher (3.98 ± 2.6, $p=0.009$). EF was substantially lower (54.73 ± 6.90, $p=0.002$) and the SYNTAX score was higher (19.8 ± 15.1, $p=0.002$) in the group with raised TSH. EF and the Tei index ($r=-0.316$, $p=0.025$), as well as the SYNTAX score ($r=-0.302$, $p=0.033$), showed a strong negative association.***

Conclusions: ***Impaired LV function and more severe CAD are linked to elevated TSH levels. TSH may be a helpful marker for determining which patients are more likely to have severe CAD and reduced left ventricular function.***

Keywords: Thyroid-Stimulating Hormone; Coronary Artery Disease; Left Ventricular Function

1. Introduction

Globally, atherosclerosis is the principal factor contributing to cardiovascular disease (CVD), which has emerged as a leading cause of death. Coronary artery disease (CAD) is primarily responsible for the burden of CVD .[1] CAD is a common medical condition that carries a high risk of morbidity and death .[2]

Thyroid hormones have a greater impact on the cardiovascular system; the heart and vascular system are negatively impacted by both hypothyroidism and hyperthyroidism .[3] Impaired left ventricular (LV) systolic function during exercise and impaired LV diastolic function at rest are two cardiac function abnormalities associated with hypothyroidism .[4] Also, CAD and accelerated atherosclerosis are linked to hypothyroidism, which is caused by arterial stiffness, diastolic hypertension, and compromised endothelial function .[5]

In thyrotoxicosis, a high-output cardiac state that can ultimately lead to high-output heart failure, reductions in peripheral systemic vascular resistance are linked to increases in heart rate, cardiac contractility, and cardiac output .[6]

The purpose of this research is to examine the association between serum levels of thyroid stimulating hormone (TSH), the existence and degree of coronary artery disease (CAD) in patients referred for coronary angiography, and the effect of this relationship on left ventricular function.

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2. Patients and methods

Design and population:

Fifty CAD patients undergoing coronary angiography participated in this cross-sectional analytic study at the Department of Cardiovascular Medicine, Faculty of Medicine, Mansoura University; Cath lab unit and Echocardiography unit. The study was done during the period from April 2019 to August 2020.

Patient Selection

Inclusion criteria were adult CAD patients who performed diagnostic coronary angiography either for chronic coronary syndrome or acute coronary syndrome and unknown TSH levels.

Exclusion criteria were patients already diagnosed with hypothyroidism or hyperthyroidism, patients with poor echo windows, EF< 40%, age >70 years old, congenital heart disease, and severe valvular heart disease.

Patient grouping

Two factors were used to categorise the study groups: TSH levels and the existence of severe coronary artery stenosis. Regarding coronary stenosis, 28 patients in Group A had considerable stenosis, while 22 patients in Group B had non-significant stenosis. Group I (normal TSH, n = 9), Group II (elevated TSH, n = 26), and Group III (decreased TSH, n = 15) were the groups based on TSH levels.

Methods

The following was administered to the patients:

Thorough history-taking with an emphasis on demographic information (age, occupation, residence), baseline clinical data (symptoms of angina, atypical chest pain, heart failure, arrhythmias, chronic illnesses, and medical regimens), and risk factor screening (obesity, dyslipidemia, smoking, diabetes, hypertension, and premature CAD family history).

Comprehensive examination: focusing on vital signs (including pulse and blood pressure(. The cardiac examination emphasized detecting cardiomegaly, murmurs, and heart failure signs. The chest and abdominal exams focused on pulmonary congestion, pleural effusion, heart failure, liver disease, and CKD. Other systems were reviewed to exclude non-cardiac causes of chest pain and systemic diseases .[7]

Baseline laboratory investigations were conducted after a 12-hour fast, including serum TSH levels, diabetes screening based on ADA criteria, lipid profile assessment for dyslipidemia, and serum creatinine for renal function evaluation. TSH was measured using Roche HITACHI cobas e 411, with a normal range of 0.27-4.2 mIU/mL. HBA1C, fasting plasma glucose, or 2-hour plasma glucose values were used to diagnose diabetes. Total cholesterol, LDL-C, HDL-C, and triglyceride values were used to diagnose dyslipidemia. A blood creatinine level of less than 1.5 mg/dL was considered renal impairment .[8]

Electrocardiogram:

Standard 12 lead ECG with assessment ST-T wave changes, arrhythmia, or any conduction abnormalities.

Echocardiography:

The American Society of Echocardiography's guidelines for echocardiographic procedures and measurements were followed. A General Electric Vivid E9 XDclear Dimensions ultrasound system with the matrix M5Sc transducer was used .[9,10] During the echocardiographic examination, the patient was positioned in the left lateral position, and 2D, M-mode echocardiography, Pulsed-wave Doppler (PWD), and Tissue Doppler Imaging (TDI) were used to assess the morphology and function of the left ventricle (TDI). The LV systolic function was determined through the evaluation of LV volumes, ejection fraction, and fractional shortening [9]. PW Doppler measured diastolic function using trans-mitral flow velocities, while TDI measured velocities at the mitral valve annulus using the apical 4-chamber view were recorded. Three categories were established for diastolic dysfunction based on TDI measurements: Grade I (delayed relaxation); E/e' ratio < 8, Peak E/A ratio < 0.8, Deceleration time (DT) > 200 ms, Grade II (pseudo-normal); E/e' ratio between 8–15, Peak E/A ratio between 0.8–1.5, DT between 160–200 ms, and Grade III (restrictive); E/e' ratio > 15, Peak E/A ratio > 2.0, DT < 160 ms .[11]

The non-invasive Doppler-derived Myocardial Performance Index, also referred to as the Tei index, assesses the left ventricle's systolic and diastolic function. A normal MPI value of less than 0.40 is calculated as the ratio of ejection time (ET) to the sum of isovolumetric contraction time (IVCT) and isovolumetric relaxation time (IVRT). MPI = (TST-ET)/ET or Isovolumetric Contraction Time (IVCT) is added with Isovolumetric Relaxation Time (IVRT), which is divided by ejection time. MPI= (IVCT+IVRT)/ET (Normal value of MPI is ≤ 0.40) [12].

Coronary Angiography:

Coronary angiography and SYNTAX score were assessed for all patients to identify lesion sites (ostial, midshaft, distal) and bifurcation lesions in the LAD, LCX, and RCA. The Seldinger technique was used, along with 6F or 7F catheters, to accomplish the procedure via the right femoral or radial artery.

The SYNTAX Score, with low SYNTAX (0–22), intermediate SYNTAX (23–32), and high SYNTAX

(>33), is an angiographic grading method used to assess CAD severity [13]. Computer software that consists of sequential and interactive self-guided question types calculates the SYNTAX score [14]. The degree of significant coronary artery stenosis used in our study is defined as a luminal narrowing of ≥70% in one or more major coronary arteries or ≥50% in the left main coronary artery, as determined by coronary angiography.

Statistical methods

The statistical analysis was conducted using SPSS version 20 and Excel (Microsoft Office 2013). The Kolmogorov-Smirnov test was used to assess the normality of the data. The data groups were presented as frequencies and percentages, and the Chi-square test was performed to compare them. The quantitative data was expressed as mean ± SD or median and range for two-group comparisons using the Student's t-test or Mann-Whitney test; for more than two groups, Kruskal-Wallis or One-Way ANOVA was utilized. Variable relationships were analyzed using Pearson correlation, diagnostic sensitivity, and specificity were assessed using ROC curves, and independent predictors were found using regression analysis. Set at $p < 0.05$ for significance.

3. Results

The present study included 50 patients (48.0% males). Mean age was (61.0 ± 5.28). Among participants, 34% smoked, 46% had diabetes, 70% had hypertension, 40% reported stress, and 16% had a family history of cardiovascular disease. Vessel affection rates were: no vessel (44%), single vessel (8%), two vessels (18%), and three vessels (30%). Syntax scores were mostly <22 (68%), with 14% in the 22-32 range and 18% >32. Clinical presentations included 76% stable coronary conditions and 24% ACS. Based on TSH levels, 18% had normal, 52% elevated, and 30% decreased TSH. In CAD evaluation, 44% had non-significant stenosis, while 56% had significant stenosis. Based on syntax score, cases were classified into 3groups; SS<22 (n=34); SS 22-32 (n=7), and SS >32 (n=9). Regarding thyroid status, cases divided to normal TSH group (n=9), decreased TSH group (n=15) and elevated TSH group (n=26).

No significant difference regarding age, sex, history of DM, HTN, smoking, family history and stress among studied groups (Table 1).

Table 1. Demographic data among studied groups classified according to TSH level.

PARAMETER		GROUP I (NORMAL TSH) (N=9)		GROUP II (ELEVATED TSH) (N=26)		GROUP III (DECREASED TSH) (N=15)		P
AGE*	Mean ± SD	60.0 ± 6.30		62.19 ± 4.06		59.53 ± 6.32		P=0.251 P[1]=0.855 P[2]=1.00 P[3]=0.373
SEX	Male	4	44.4%	13	50.0%	7	46.7%	P=0.952 P[1]=0.774
	Female	5	55.6%	13	50.0%	8	53.3%	P[2]=0.916 P[3]=0.837
DM		3	33.3%	11	42.3%	9	60.0%	P=0.358 P[1]=0.636 P[2]=0.206 P[3]=0.275
HTN		7	77.8%	17	65.4%	11	73.3%	P=0.740 P[1]=0.490 P[2]=0.808 P[3]=0.598
SMOKING		2	22.2%	11	42.3%	4	26.7%	P=0.424 P[1]=0.282 P[2]=0.808 P[3]=0.317
FAMILY HISTORY		1	11.1%	5	19.2%	2	13.3%	P=0.802 P[1]=0.577 P[2]=0.873 P[3]=0.629
STRESS		3	33.3%	12	46.2%	5	33.3%	P=0.652 P[1]=0.503 P[2]=1.00 P[3]=0.422

Chi-Square test, one way ANOVA*. P, between 3 groups; P1, between normal TSH group and elevated TSH group; P2, between normal TSH group and decreased TSH group; P3, elevated TSH group and decreased TSH group. *Significant (P value < 0.05), DM: Diabetes mellitus, HTN: Hypertension, TSH: Thyroid stimulating hormone

When comparing the major CA stenosis group to the non-significant CA stenosis group, a statistically-significant lower EF was seen in significant CAD group with a statistically-significant higher SYNTAX score and a significantly higher TSH level than the non-significant CA stenosis group (Table 2).

Table 2. Comparative analysis of echocardiographic parameters, SYNTAX score, blood pressure, laboratory parameters, among studied groups based on significant CAD.

Parameter		Group A (Non-significant CA stenosis) (N=22)		Group B (Significant CA stenosis) (N=28)		P
EF		61.36 ± 9.07		56.0 ± 6.67		0.020
SYNTAX score		3.00 ± 0.01		22.07 ± 13.6		<0.001
SYNTAX score group*	<22	22	100.0%	12	42.9%	<0.001
	22-32	0	0.0%	7	25.0%	
	>32	0	0.0%	9	32.1%	
Tei-index		0.63 ± 0.18		0.72 ± 0.29		0.208
Systolic BP (mmHg)		140.22 ± 22.4		134.2 ± 24.4		0.382
Diastolic BP (mmHg)		84.09 ± 13.05		81.6 ± 11.4		0.478
Hb (g/dl)		12.16 ± 1.29		12.31 ± 1.48		0.710
TSH		2.05 ± 2.2		3.98 ± 2.6		0.009

(mIU/L)				
Creatinine (mg/dl)		1.00 ± 0.27	1.09 ± 0.29	0.274
TC (mg/dl)		190.9 ± 42.9	204.6 ± 44.5	0.278
LDL (mg/dl)		129.8 ± 44.2	140.4 ± 47.7	0.425
HDL (mg/dl)		35.0 ± 7.17	35.2 ± 5.1	0.944
TG (mg/dl)		130.0 ± 55.6	145.1 ± 51.3	0.327

Chi-Square test*, independent T test. P between two groups, **significant (P value < 0.05), values represent mean ± SD, BP: blood pressure, EF: ejection fraction, Hb: Hemoglobin, HDL: high-density lipoprotein, LDL: low-density lipoprotein, M: mean, N: number, SD: standard deviation TG: triglycerides, TSH: thyroid stimulating hormone, TC: total cholesterol.

The DBP was statistically significantly lower in the lowered TSH group compared to the raised and normal TSH groups. Furthermore, total cholesterol and LDL were significantly greater in the normal and raised TSH groups compared to the lowered TSH group. Additionally, TG was considerably greater in the group with elevated TSH than in the group with lowered TSH (Table 3).

Table 3. Comparison of blood pressure values, and laboratory parameters among studied groups classified according to TSH level.

Parameter		Group I (Normal TSH) (N=9)	Group II (Elevated TSH) (N=26)	Group III (Decreased TSH) (N=15)	P
Blood pressure*	Systolic (M ±SD)	136.6 ± 23.3	132.1 ± 19.9	145.3 ± 28.3	P =0.227 P [1]=1.00 P [2]=1.00 P [3]=0.260
	Diastolic (M ±SD)	85.5 ± 11.5	87.1 ± 12.5	73.3 ± 4.8	P =0.001 P [1]=1.00 P [2]=0.027 P [3]=0.001
Hb (g/dl)	M ± SD	12.44 ± 1.29	12.33 ± 1.24	12.0 ± 1.72	P =0.698 P [1]=1.00 P [2]=1.00 P [3]=1.00
Creatinine (mg/dl)	M ± SD	1.07 ± 0.28	1.07 ± 0.30	1.01 ± 0.26	P =0.824 P [1]=1.00 P [2]=1.00 P [3]=1.00
TC(mg/dl)	M ± SD	205.5 ± 44.1	216.1 ± 45.1	164.0 ± 11.2	P =<0.001 P [1]=1.00 P [2]=0.039 P [3]=<0.001
LDL(mg/dl)	M ± SD	143.4 ± 45.8	151.5 ± 50.6	103.7 ± 6.5	P =0.003 P [1]=1.00 P [2]=0.045 P [3]=0.003
HDL(mg/dl)	M ± SD	36.0 ± 5.6	33.6 ± 4.3	37.3 ± 8.1	P =0.148 P [1]=0.907 P [2]=1.00 P [3]=0.176
TG(mg/dl)	M ± SD	130.5 ± 67.3	154.9 ± 49.7	114.7 ± 41.9	P =0.045 P [1]=0.669 P [2]=1.00 P [3]=0.047

Chi-Square test, One-Way ANOVA*. P, between 3 groups; P1, between normal TSH group and elevated TSH group; P2, between normal TSH group and decreased TSH group; P3, between elevated TSH group and decreased TSH group. Significant (P value < 0.05), Abbreviations as in table 2.

In comparison to normal and lowered TSH groups, the number of coronary vessels was considerably higher in the elevated TSH group. The elevated TSH group exhibited a significantly higher SYNTAX score when compared to the normal and lowered TSH groups. When comparing the increased TSH group to the normal and lowered TSH groups, there was a noticeable decrease in EF (Table 4).

Table 4. Comparison of angiographic and echocardiographic parameters among studied groups classified according to TSH level.

Parameter		Group I (Normal TSH) (N=9)		Group II (Elevated TSH) (N=26)		Group III (Decreased TSH) (N=15)		P
Vessel affected	No vessel affected	7	77.8%	7	26.9%	8	53.3%	P =0.001
	Single-vessel affected	0	0.0%	0	0.0%	4	26.7%	P [1]=0.026
	Two-vessel affected	1	11.1%	6	23.1%	2	13.3%	P [2]=0.377
	Three-vessel affected	1	11.1%	13	50.0%	1	6.7%	P [3]=0.002
SYNTAX score*	M ± SD	3.66 ± 2.0		19.8 ± 15.1		9.06 ± 10.55		P =0.002 P [1]=0.005 P [2]=0.930

								P [3]=0.032
SYNTAX	<22	9	100.0%	13	50.0%	12	80.0%	P =0.045
score	22-32	0	0.0%	5	19.2%	2	13.3%	P [1]=0.028
group	>32	0	0.0%	8	30.8%	1	6.7%	P [2]=0.358
								P [3]=0.128
EF	M ± SD	63.66 ± 11.22		54.73 ± 6.90		61.46 ± 4.99		P =0.002
								P [1]=0.009
								P [2]=1.00
								P [3]=0.021
Tei-index	M ± SD	0.617 ± 0.14		0.729 ± 0.30		0.644 ± 0.20		P =0.438
								P [1]=0.831
								P [2]=1.00
								P [3]=0.984

One-Way ANOVA* and Chi-Square test were used to compare three groups: P, between the groups; P1, between the normal TSH group and elevated TSH group; P2, between the normal TSH group and decreased TSH group; P3, between the elevated TSH group and decreased TSH group. **Significant (P value < 0.05). Abbreviations as in table 2

Strong positive correlations were seen between TSH and diastolic blood pressure, syntactic score, total cholesterol, LDL, and TG, but strong negative correlations were observed between EF, syntactic score, and tei index. Otherwise, no more noteworthy correlation was found (Table 5).

Table 5. Correlation between SYNTAX score, tei index, TSH level and different parameters among studied cases.

		Tei index	SYNTAX score	TSH
Age	r	0.100	0.081	0.226
	P	0.489	0.577	0.115
Systolic BP	r	-0.118	-0.035	-0.244
	P	0.416	0.810	0.088
Diastolic BP	r	-0.047	0.044	0.388**
	P	0.747	0.762	0.005
EF	r	-0.316*	-0.302*	-0.249
	P	0.025	0.033	0.082
Tei-index	r	1	0.064	0.235
	P		0.657	0.101
SYNTAX score	r	0.064	1	0.413*
	P	0.657		0.003
TSH	r	0.235	0.413*	1
	P	0.101	0.003	
Hb	r	0.080	0.091	0.129
	P	0.579	0.531	0.374
Creatinine	r	0.110	0.271	0.124
	P	0.448	0.051	0.392
Total cholesterol	r	0.122	0.086	0.378**
	P	0.401	0.550	0.007
LDL	r	0.077	0.048	0.303**
	P	0.595	0.739	0.032
HDL	r	0.031	-0.006	-0.265
	P	0.830	0.969	0.063
Triglycerides	r	0.151	0.152	0.397**
	P	0.294	0.293	0.004

Pearson correlation, Abbreviations as in table 2

The best SYNTAX score, Tei index, and TSH levels for multivessel disease prediction were determined using ROC analysis. The optimum cut-off values for SYNTAX scores were 7.0. At p = 0.020, the area under the curve (AUC) was 0.870. 0.550 was the best cut-off number for the Tei index. 0.615 was the AUC (p=0.470). The optimal TSH cut-off value was 0.16. 0.984 was the AUC (p=0.002) (Figure 1).

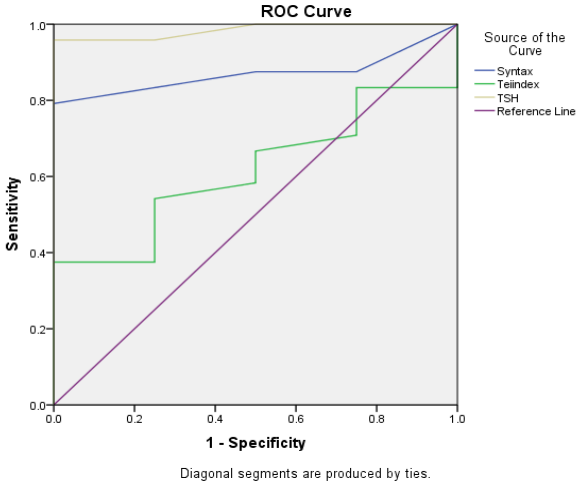

Figure 1. ROC curve for assessment of performance characteristics of SYNTAX score, Tei index and TSH in prediction of multivessel disease from single vessel disease.

Logistic regression analysis was conducted for prediction of multi vessel disease within cases, using age, gender, laboratory data, smoking, DM, hypertension, EF, Tei index and SYNTAX score as covariates. Univariate analysis revealed that HDL was unique protective factor (Table 6).

Table 6. Regression analysis for prediction of multi vessels disease from single vessel disease.

	UNIVARIATE ANALYSIS			
	p	OR	95% CI	
AGE	0.589	0.946	0.772	1.159
GENDER	0.877	0.846	0.102	7.036
SMOKING	0.756	0.714	0.086	5.959
HYPERTENSION	0.146	6.00	0.535	67.27
DIABETES MELLITUS	0.370	0.333	0.030	3.676
EJECTION FRACTION	0.742	0.973	0.825	1.147
TEI-INDEX	0.359	25.017	0.026	72.46
SYNTAX SCORE	0.089	1.202	0.972	1.487
THYROID STIMULATING HORMONE	0.275	7.872	0.101	14.291
TOTAL CHOLESTEROL	0.078	1.162	0.984	1.37
LOW-DENSITY LIPOPROTEIN	0.131	1.182	0.952	1.469
HIGH-DENSITY LIPOPROTEIN	0.049	0.783	0.608	0.998
TRIGLYCERIDES	0.128	1.036	0.990	1.084

OR., odds ratio; CI., confidence- interval; logistic regression was used.

4. Discussion

The heart and vascular system are significantly impacted by thyroid hormones, and there is a complex relationship between thyroid disorders and cardiovascular illness .[3]

In the current study, compared to patients without considerable coronary artery stenosis, those with significant coronary artery stenosis had a higher SYNTAX score and a considerably lower ejection fraction (EF).

These results align with those of Senthilnathan et al., who found that patients with significant coronary artery stenosis had significantly higher SYNTAX scores than patients without it [1]. In contrast, patients with and without severe coronary artery stenosis did not significantly differ in their ejection fraction, according to Coceani et al.[15].

According to our study, patients with significant coronary artery stenosis had significantly higher TSH levels than those without significant stenosis.

Daswani et al. did not find any significant changes between individuals with and without significant coronary artery stenosis in biochemical parameters such as total cholesterol, LDL, TG, and HDL.[16], however, TSH was found to be considerably higher in patients with severe stenosis by Senthilnathan et al.[1]. In contrast, Senthilnathan et al. also noted that total cholesterol, LDL, and TG were significantly higher in patients with significant stenosis[1], whereas Daswani et al. found no significant difference in TSH levels between the groups .[16]

The study's findings demonstrated a significant inverse relationship between MPI (Tei index) and ejection percentage (EF) and SYNTAX score. Furthermore, there was a strong correlation seen between TSH levels and diastolic blood pressure, SYNTAX score, total cholesterol, LDL, and triglycerides (Table 4).

Elhini et al. discovered strong positive correlation between diastolic blood pressure, BMI, TC, LDL-C, and TG, which are consistent with our findings[17]. MPI (Tei index) and EF showed a significant negative correlation, according to Ammar et al.[18]. On the other hand, Helmy et al. found a weak but non-significant negative correlation between the SYNTAX score and EF [19].

Since Tei Chuwa et al. initially created the Myocardial Performance Index (MPI) in 1995, numerous research investigated MPI's potential as a predictor of CVDs and potential cardiovascular death [12,20]. Echocardiography, a reliable and repeatable diagnostic tool, has evolved to include parameters like MPI for predicting future cardiovascular outcomes. [21,22] Given the common coexistence of cardiovascular and thyroid diseases [23], increasing data in this area is crucial. Small-scale studies suggest that treating hypothyroidism may improve cardiac function and MPI [24], highlighting the need for more prospective data on the cardiovascular outcomes of these patients.

Regarding the thyroid groups' demographic data, there were no significant differences between the groups with respect to age, sex, and CVD risk factors, including smoking, family history, hypertension, diabetes mellitus, or stress.

Patients with normal, raised, or lowered TSH levels did not significantly differ in terms of gender, diabetes, hypertension, or smoking, according to studies by Ling et al. and Elhini et al.[17,25.] However, Elhini et al. found that the elevated TSH group was significantly older .[17]

According to this study, patients with elevated TSH levels had a significantly higher number of stenosed coronaries than those with lower or normal TSH levels.

When comparing raised TSH to those with lowered and normal TSH, there was a statistically-significant decrease in EF. SYNTAX score was significantly elevated in elevated TSH cases compared to decreased TSH and normal TSH cases. When comparing the reduced TSH group to the raised and normal TSH groups, diastolic blood pressure was significantly lower in the former. Patients with elevated TSH had a significantly lower ejection fraction (EF) and higher SYNTAX score compared to those with decreased or normal TSH levels.

Elhini et al. found that the decreased TSH group had significantly lower diastolic BP and higher EF than the elevated and normal TSH groups .[17] Hatab et al. also reported increased diastolic BP in the elevated TSH group in comparison to the control and decreased TSH groups and a decrease in DBP in the decreased TSH group compared to controls.[26] Senthilnathan et al. noted that elevated TSH was more common in patients with a higher SYNTAX score, while normal TSH was more prevalent in those with low scores .[1] However, Elhini et al. discovered that although the increased TSH group had greater multivessel disease, there was no significant variation in the total number of vessels affected.[17]

In the current study, the groups of elevated and normal TSH levels had significantly greater levels of total and LDL cholesterol than the group with lowered TSH. Triglycerides (TG) were also significantly higher in the group with elevated TSH than in the group with lowered TSH.

These results go in harmony with Elhini et al., noted that both elevated and normal TSH groups had a significantly higher TC and LDL-C, and TG, than decreased TSH group .[17]

Regarding the ROC analysis, the best SYNTAX

score, Tei index, and TSH levels were determined for multivessel disease prediction. The optimal cut-off values for the SYNTAX score, according to the current study, were 7.0. At p = 0.020, the area under the curve (AUC) was 0.870. 0.550 was the best cut-off number for the Tei index. The AUC was 0.615 (p=0.470). TSH's best cut-off values were 0.16. The AUC was 0.984 (p=0.002). To our knowledge, no other study assesses the ROC curve of the Tie index, SYNTAX score, and TSH for the prediction of multivessel disease.

As regard Logistic regression analysis was conducted for prediction of multi vessel disease within cases, using age, gender, laboratory data, smoking, DM, hypertension, EF, Tei index and SYNTAX score as covariates. Because small sample size in our study, univariate analysis showed that HDL was a distinct prognostic factor.

Cantarelli et al. found that age above 40, male gender, arterial hypertension, diabetes, dyslipidemia, history of acute myocardial infarction, and chronic renal failure were the most important characteristics to separate individuals with single-vessel involvement from those with multivessel involvement (p. < 0.01) .[27]

Park et al., documented that age, DM, and LDL were significantly multivessel CAD independent predictors .[28]

The small sample size and single-center design of this study are limitations that might have an impact on how broadly the results can be applied. Furthermore, it is impossible to determine a causal relationship between TSH levels and the severity of CAD due to the study's cross-sectional design.

4. Conclusion

Impaired LV function and more severe CAD are linked to elevated TSH levels. TSH may be a helpful marker for determining which patients are more likely to have severe CAD and reduced left ventricular function.

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There are no conflicts of interest.

References

1. Senthilnathan V, Dhandapani VE, Ramraj B, et al. Relationship between Thyroid Function and Coronary Artery Disease Severity. Annals of Tropical Medicine and Public Health. 2020;23(1):233-239.
2. Yaman B, Cerit L, Günsel HK, et al. Association between Subclinical Hypothyroidism and Coro-Nary Artery Disease. Progress In Nutrition. 2019;21(4):871-875.
3. Khan R, Sikanderkhel S, Gui J, et al. Thyroid and Cardiovascular Disease: A Focused Review on the Impact of Hyperthyroidism in Heart Failure. Cardiol Res. 2020 Apr;11(2):68-75.
4. von Hafe M, Neves JS, Vale C, et al. The impact of thyroid hormone dysfunction on ischemic heart disease. Endocr Connect. 2019 May 1;8(5):R76-r90.
5. Zúñiga D, Balasubramanian S, Mehmood KT, et al. Hypothyroidism and Cardiovascular Disease: A Review. Cureus. 2024 Jan;16(1):e52512.
6. Raguthu CC, Gajjela H, Kela I, et al. Cardiovascular Involvement in Thyrotoxicosis Resulting in Heart Failure: The Risk Factors and Hemodynamic Implications. Cureus. 2022 Jan;14(1):e21213.
7. Mancia G, Fagard R, Narkiewicz K, et al. 2013 ESH/ESC guidelines for the management of arterial hypertension: The Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). Eur Heart J. 2013 01/01;1:37-38.
8. Suh SH, and Kim SW. Dyslipidemia in Patients with Chronic Kidney Disease: An Updated Overview. Diabetes Metab J. 2023 Sep;47(5):612-629.
9. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015 Jan;28(1):1-39.e14.
10. Sutherland GR, Di Salvo G, Claus P, et al. Strain and strain rate imaging: a new clinical approach to quantifying regional myocardial function. J Am Soc Echocardiogr. 2004 Jul;17(7):788-802.
11. Nagueh SF, Appleton CP, Gillebert TC, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography. J Am Soc Echocardiogr. 2009 Feb;22(2):107-33.
12. Tei C, Nishimura RA, Seward JB, et al. Noninvasive Doppler-derived myocardial performance index: correlation with simultaneous measurements of cardiac catheterization measurements. J Am Soc Echocardiogr. 1997 Mar;10(2):169-78.
13. Serruys PW, Onuma Y, Garg S, et al. Assessment of the SYNTAX score in the Syntax study. EuroIntervention. 2009 May;5(1):50-6.
14. Sianos G, Morel MA, Kappetein AP, et al. The SYNTAX Score: an angiographic tool grading the complexity of coronary artery disease. EuroIntervention. 2005 Aug;1(2):219-27.
15. Coceani M, Iervasi G, Pingitore A, et al. Thyroid Hormone and Coronary Artery Disease: From Clinical Correlations to Prognostic Implications. Clinical Cardiology. 2009;32(7):380-385.
16. Daswani R, Jayaprakash B, Shetty R, et al. Association of Thyroid Function with Severity of Coronary Artery Disease in Euthyroid Patients. J Clin Diagn Res. 2015 Jun;9(6):Oc10-3.
17. Elhini SH, Matta RA, Eledawi NN, et al. Impact and predictors of thyroid dysfunction among patients with stenotic coronary artery lesion during late postacute coronary syndrome. Egyptian Journal of Obesity, Diabetes and Endocrinology. 2017;3(1):1-9.
18. Ammar S, ELKeshk E, Tabl M, et al. Relationship between myocardial performance index and severity of coronary artery disease in patients with stable coronary artery disease. Int J Adv Res. 2016;4:494-502.
19. Helmy H, Abdel-Galeel A, Taha Kishk Y, et al. Correlation of corrected QT dispersion with the severity of coronary artery disease detected by SYNTAX score in non-diabetic patients with STEMI. Egypt Heart J. 2017 Jun;69(2):111-117.

20. Kamishirado H, Hayashi T, Hatano H, et al. [Evaluation of restenosis after percutaneous transluminal coronary angioplasty using a Doppler index, the Tei index]. J Cardiol. 1999 Mar;33(3):127-33.
21. Cooper DS, and Biondi B. Subclinical thyroid disease. The Lancet. 2012;379(9821):1142-1154.
22. Veltri F, Rocha FO, Willems D, et al. Prevalence of thyroid dysfunction and autoimmunity in the older population and implications of age-specific reference ranges. Clin Chim Acta. 2017 Feb;465:34-39.
23. Danzi S, and Klein I. Thyroid disease and the cardiovascular system. Endocrinol Metab Clin North Am. 2014 Jun;43(2):517-28.
24. Razvi S, Ingoe L, Keeka G, et al. 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. 2007 May;92(5):1715-23.
25. Ling Y, Jiang J, Gui M, et al. Thyroid Function, Prevalent Coronary Heart Disease, and Severity of Coronary Atherosclerosis in Patients Undergoing Coronary Angiography. Int J Endocrinol. 2015;2015:708272.
26. Hatab AA, Zaki ER, Elmalah AA, et al. Effect of thyroid dysfunction on left ventricular performance using different echo-Doppler modalities. SJAMF. 2020;4(2):276-281.
27. Cantarelli MJdC, Castello Junior HJ, Gonçalves R, et al. Preditores independentes de doença arterial coronária multiarterial: resultados do Registro Angiocardio. Rev Bras Cardiol Invasiva. 2015;70(3):266-270.
28. Park CS, Hong OK, Kim MK, et al. Serum Bone Morphogenic Protein-4 Contributes to Discriminating Coronary Artery Disease Severity. Medicine (Baltimore). 2015 Sep;94(39):e1530.