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ORIGINAL ARTICLE

Study of The Relation Between Thyroid Function and Metabolic Syndrome in Elderly Patients

Eman Ghoneimy Mahrous Elshourbagy[*1], Mohamed Mohamed Mohamed, Ahmed Hassaan[1], Samy Hasan Mohamed[2], Ahmed S Allam[1]

[1] Internal Medicine Department, Faculty of Medicine- Zagazig University

[2] Medical biochemistry Department, Faculty of Medicine, Zagazig University

***Corresponding author:**
Eman Ghoneimy Mahrous Elshourbagy

Email:
emanelshourbagy74@gmail.com

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ABSTRACT

Background: It is unknown how thyroid hormones, TSH, and other elements of the metabolic syndrome are related to one another. It is possible to investigate and evaluate thyroid function in older adults with and without metabolic syndrome to draw attention to it and look for any connections between thyroid function and the elements of the metabolic syndrome.

Aim: To detect the association of thyroid Hormones, TSH and components of metabolic syndrome and to evaluate the relationship between thyroid Hormones, TSH and HOMA-IR.

Methods: This case-control study included 64 elderly subjects with and without metabolic syndrome recruited from outpatient of Zagazig University hospital, from July 2023 to January 2024. We divided the patients into group (I): 32 elderly healthy individuals without metabolic syndrome matched age and sex served as control group and group (II): 32 elderly patients with metabolic syndrome matched age and sex served as case group. Thyroid functions tests were measured.

Results: There was a highly statistically significant increase in TSH in group II compared with group I ($p \leq 0.001$). There was statistically significant increase in FT3 in group II compared with group I ($p \leq 0.05$). There was statistically significant decrease in FT4 in group II compared with group I ($p \leq 0.05$) (Table 3). There was highly statistically significant increase in HOMA-IR in group II compared with group I ($p \leq 0.001$). Old age OR 1.79 (CI:1.07-3), smoking OR 1.88 (CI:1.36-2.99), obesity OR 1.07 (CI:1.07-1.26), DM OR 2.8 (CI:1.41-5.57), HTN OR 1.14 (CI:1.03-1.25) & dyslipidemia OR 1.86 (CI:1.11-3.12) were significant risk factors for metabolic syndrome ($p \leq 0.05$).

Conclusion: In conclusion, there is a strong association between thyroid dysfunction and each of metabolic syndrome, and HOMA-IR. Patients with hypothyroidism and subclinical hypothyroidism had an increased risk of metabolic syndrome.

Keywords: Thyroid; Metabolic Syndrome; Elderly.

INTRODUCTION

Thyroid hormones are crucial for controlling thermogenesis as well as the metabolism of carbohydrates and fats, which makes them an essential component of the dynamic energy balance [1]. Increased oxygen consumption, thermogenesis, protein synthesis, lipolysis, glucose uptake by cells, glycogenolysis, and gluconeogenesis are the main effects of thyroid hormones on metabolism [2].

Thyroid hormone abnormalities affect metabolism, and metabolic syndrome and some of these changes have similar pathophysiologic mechanisms. Thyroid dysfunction can therefore have an impact on metabolic syndrome. It has been demonstrated that thyroid hormones play a significant role in maintaining glucose homeostasis and affect fasting glucose levels by preventing the effects of insulin [3]. Hyperglycemia is a metabolic illness called

diabetes mellitus (DM), wherein there is a malfunction in the generation, action, or both of the insulin molecules [4].

Diabetes mellitus type 2 (T2DM) is a potentially fatal condition that is mostly linked to vascular complications. These complications can lead to peripheral vasculopathy, ischemic heart disease, retinopathy, and nephropathy. In individuals with type 2 diabetes, endothelial dysfunction (ED) has been suggested as a major therapeutic target because it is the primary etiological component that causes moderate to severe vascular problems [5].

Individuals diagnosed with diabetes mellitus (DM) and its related vascular problems seem to be especially vulnerable to accelerated atherosclerosis has the potential to cause early death, cardiovascular and cerebrovascular problems [6].

Positive correlations have been demonstrated between the levels of cardio-metabolic variables and free thyroxin (T4) and thyrotropin (TSH), even in cases of euthyroidism [7].

Metabolic syndrome, a well-known set of cardiovascular risk factors, is a major global public health concern [8]. Metabolic syndrome is associated with diabetes, cardiovascular disease, and maybe some types of cancer [9]. The incidence of metabolic syndrome is becoming more problematic due to the rise in overweight and obesity worldwide [10].

As per the NCEP, a male individual is classified as having metabolic syndrome if he displays three or more of the subsequent symptoms: elevated blood pressure (≥130/85 mmHg), low HDL cholesterol (<1.04 mmol/l), high waist circumference (>102 cm), elevated triglyceride levels (≥1.69 mmol/l), and elevated glucose levels (≥6.1 mmol/l) [11].

The death rate from cardiovascular disease increased from 35.6 to 52.4 per 100,000 persons between 2003 and 2014. Reduced focus and effort are therefore needed to minimize the prevalence of metabolic syndrome because the frequency of deaths from related diseases is rapidly increasing [12].

To our knowledge, there are few studies that evaluate the association between thyroid function and metabolic syndrome in elderly.

This study was done to detect the association of thyroid Hormones, TSH and components of metabolic syndrome and to evaluate the relationship between thyroid Hormones, TSH and HOMA-IR.

METHODS

This Case-control study included 64 elderly subjects with and without metabolic syndrome recruited from outpatient of Zagazig University hospital, from July 2023 to January 2024.

Group (I) was formed by us from the patients. A control group and group (II) of thirty-two older, healthy people without metabolic syndrome were matched for age and sex. An age and sex-matched case group of thirty-two senior people with metabolic syndrome was used.

Inclusion criteria included elderly (age 65 years old or more) of both sexes and metabolic syndrome was diagnosed based on National Cholesterol Education Program Adult Treatment Panel-III criteria (NCEP-ATP III).

Exclusion criteria included age less than 65, patients with other endocrinal problems, Patients on thyroid-altering medications, those with cardiovascular illness, corticosteroid use, active liver disease, renal dysfunction, pregnant women, patients who have died or vanished during follow-up, and others may also have an impact on thyroid function.

Complete blood count (CBC), kidney, liver function tests, coagulation profile (Prothrombin time (PT), INR, and partial thromboplastin time (PTT)), lipid profile (LDL-C, HDL-C, serum total cholesterol, serum triglyceride), fasting blood glucose, HbA1C, HOMA-IR index (which was calculated using the formula: HOMA-IR index= fasting insulin (μIU/ml) x fasting plasma glucose (mg/dl)/405), and thyroid function tests were performed on all study participants.

Written informed consent was taken from each patient who participate in this study.We obtained an approval for performing the study from internal medicine and medical biochemistry departments, Zagazig University Hospitals after taking Institutional Review Board (IRB) approval (IRB number 10106).

STATISTICAL ANALYSIS

Subsequently, the information was entered into the statistical package for the social sciences (IBM crop. Released 2020. IBM SPSS statistics for windows, version 27.0. Armonk, NY:IBM crop) application for analytical purposes.The Kolmogorov-Smirnov test was used to varify the normality of distribution. Correlations, the Mann Whitney test, the Chi Square Test ($\chi 2$), and the -t test, logistic regression analysis were employed.

RESULTS

When comparing group II to group I, there was a very statistically significant increase in BMI and WC, systolic and diastolic blood pressure ($p \leq 0.001$) (Table 1). When group II was compared to group I, there was a highly statistically significant increase in FBG, fasting insulin, HOMA-IR & HBA1C, TC, TG & LDL, ALT, AST & creatinine, and urea ($p \leq 0.001$) and a

highly statistically significant drop in HDL ($p \leq 0.001$) in group II (Table 2). When comparing group II to group I, there was a very statistically significant increase in TSH ($p \leq 0.001$). When comparing group II to group I, there was a statistically significant rise in FT3 ($p \leq 0.05$). When group II's FT4 was compared to group I, there was a statistically significant drop ($p \leq 0.05$) (Table 3).
A statistically significant negative connection ($p \leq 0.05$) was found between FT3 and fasting insulin in group II (Table 4). A statistically significant positive connection was seen between FT4 and HDL in group II. Between FT4 and TLC, there was a statistically significant negative connection ($p \leq 0.05$) (Table 5). A statistically significant negative connection ($p \leq 0.05$) was found between TSH and S.Albumin in group II (Table 6). The following were significant risk variables for metabolic syndrome: old age OR 1.79 (CI:1.07-3), smoking OR 1.88 (CI:1.36-2.99), obesity OR 1.07 (CI:1.07-1.26), DM OR 2.8 (CI:1.41-5.57), HTN OR 1.14 (CI:1.03-1.25) & dyslipidemia OR 1.86 (CI:1.11-3.12) were significant risk factors for metabolic syndrome ($p \leq 0.05$) (Table 7).

Table 1: Baseline data of the studies groups:

Variable	Group I (N=32)		Group II (N=32)		t-test	P-value
Age (years): • **Mean ± SD** • **Range**	72.2 ±4.6 65-82		74.9 ±5.3 65-82		2.2	0.131 (NS)
Variable	**N**	**%**	**N**	**%**	**χ 2**	**P-value**
Sex: • **Male** • **Female**	16 16	50 50	16 16	50 50	-----	1 (NS)
BMI (kg/m2): • **Mean ± SD** • **Range**	25.8 ±1.5 22-28.2		32.2 ±2 30.1-38		14.3	< **0.001** **(HS)**
WC (cm): • **Mean ± SD** • **Range**	93.7 ±6.3 82.9-104.3		104.8 ±1.7 102.4-108.1		9.6	< **0.001** **(HS)**
Systolic BP (mmHg): • **Mean ± SD** • **Range**	120.6 ±4.7 110-125		139.4 ±4.7 135-150		15.9	< **0.001** **(HS)**
Diastolic BP (mmHg): • **Mean ± SD** • **Range**	78.1 ±5 70-85		92 ±3.5 85-100		12.7	< **0.001** **(HS)**

N: Number, SD: Standard Deviation, NS: Non-significant BMI: Body Mass Index, kg/m2: kilogram per square meter, WC: Waist Circumference, cm: centimeter, BP: Blood Pressure, mmHg: millimeters of mercury HS: Highly Significant

Table 2: Sugar profile of the studies groups:

Variable	Group I (N=32)	Group II (N=32)	t-test	P-value
FBG (mg/dl): • **Mean ± SD** • **Range**	97 ±3.7 89-104	129.7 ±10.7 114-153	13.4	< **0.001** **(HS)**
Fasting insulin (µIU/mL): • **Mean ± SD** • **Range**	8.9 ±1.5 5.7-11.4	18.6 ±3.5 14.2-28	14.3	< **0.001** **(HS)**
HOMA-IR: • **Mean ± SD** • **Range**	2.1 ±0.39 1.4-2.8	5.97 ±1.5 4.1-10.3	14.2	< **0.001** **(HS)**
Variable	**Group I (N=32)**	**Group II (N=32)**	**t-test**	**P-value**

HBA1C (%): • **Mean ± SD** • **Range**	5.2 ±0.43 4.7-6.2	6.9 ±0.85 5.4-8.7	10.5	**< 0.001 (HS)**
TC (mg/dl): • **Mean ± SD** • **Range**	172.6 ±15.5 146-194	205.6 ±16.9 183-255	8.1	**< 0.001 (HS)**
TG (mg/dl): • **Mean ± SD** • **Range**	120.5 ±11.9 98-138	169.6 ±13.9 151-191	15.2	**< 0.001 (HS)**
LDL-C (mg/dl): • **Mean ± SD** • **Range**	101.4 ±13.7 78-124	135.4 ±14.9 114-178	9.4	**< 0.001 (HS)**
HDL-C (mg/dl): • **Mean ± SD** • **Range**	47.1 ±4.6 41-59	36.4 ±2.8 32-41	-11.3	**< 0.001 (HS)**
ALT (U/L): • **Mean ± SD** • **Range**	21.96 ±5.3 15-31	34.3 ±12.3 20-60	5.2	**< 0.001 (HS)**
AST (U/L): • **Mean ± SD** • **Range**	22.4 ±5.4 14-33	35.6 ±12.7 19-16	5.4	**< 0.001 (HS)**
Serum Albumin (g/dl): • **Mean ± SD** • **Range**	4.1 ±0.63 2.6-5	4.1 ±0.71 2.3-5.2	-0.037	0.970 (NS)
Total bilirubin (mg/dl): • **Mean ± SD** • **Median** • **Range**	1.2 ±0.56 1.1 0.6-2.4	0.89 ±0.53 0.7 0.3-2.4	-1.9 (MW)	0.064 (NS)
Direct bilirubin (mg/dl): • **Mean ± SD** • **Median** • **Range**	0.37 ±0.18 0.33 0.15-0.9	0.35 ±0.35 0.25 0.3-1.88	-0.19 (MW)	0.845 (NS)
Creatinine (mg/dl): • **Mean ± SD** • **Range**	1 ±0.14 0.7-1.3	1.2 ±0.16 0.9-1.5	4.2	**< 0.001 (HS)**
Urea (mg/dl): • **Mean ± SD** • **Median** • **Range**	12.3 ±5.7 12.5 5-22	16.3 ±5.8 16.5 6-25	2.8 (MW)	**0.006 (S)**

FBG: Fasting Blood Glucose, mg/dl: milligram per deciliters, HBA1C: Glycated Hemoglobin, µIU/mL: micro international unit per milliliter, HOMA-IR: Homeostatic Model Assessment for Insulin Resistance, HBA1C: Glycated Hemoglobin TC: Total Cholesterol, TG: Triglycerides, LDL-C: Low Density Lipoproteins Cholesterol, HDL-C: High Density Lipoproteins Cholesterol, ALT: Alanine Transaminase, U/L: units per liter, AST: Aspartate aminotransferase, , g/dl: gram per deciliters, MW: Mann Whitney, S: Significant

Table 3: Thyroid profile of the studied groups:

Variable	Group I (N=32)	Group II (N=32)	t-test	P-value
FT3 (Pg/ml): • **Mean ± SD** • **Range**	3.3 ±0.58 2-4.2	3.6 ±0.44 2.9-4.5	2.6	**0.012 (S)**
FT4 (ng/dl): • **Mean ± SD** • **Range**	1.6 ±0.37 0.8-2.2	1.1 ±0.33 0.6-2	-2.9	**0.027 (S)**
TSH (µIU/mL): • **Mean ± SD** • **Range**	2.6 ±0.93 0.9-4.2	4.9 ±1.3 2.6-7.1	7.7	**< 0.001 (HS)**

FT3: Free Triiodothyronine, Pg/ml: picogram per milliliter, FT4: Free Thyroxine, ng/dl: nanogram per deciliter, TSH: Thyroid Stimulating Hormone

Table 4: Correlation between FT3 and other measures among group II (Metabolic syndrome):

Variable	FT3	
	r	p-value
Age	-0.201	0.271
BMI	0.079	0.666
WC	-0.16	0.381
SBP	0.123	0.502
DBP	-0.331	0.064
FBG	0.133	0.468
Fasting insulin	**-0.412**	**0.019**
HOMA-IR	-0.269	0.137
HBA1C	-0.026	0.877
TC	0.005	0.980
TG	0.056	0.762
LDL	-0.019	0.919
HDL	0.048	0.794
Hb	-0.230	0.205
RBCs	-0.231	0.204
PLTs	0.015	0.933
TLC	-0.068	0.710
ALT	-0.069	0.706
AST	-0.041	0.825
Serum Albumin	0.230	0.206
Total Bilirubin	-0.230	0.205
Direct Bilirubin	-0.236	0.193
Creatinine	-0.191	0.295
Urea	-0.184	0.313
FT4	0.083	0.650
TSH	-0.079	0.669

Hb: Hemoglobin, RBCs: Red Blood Cells, PLTs: Platelet Count Test, TLC: Total Leukocyte Count

Table 5: Correlation between FT4 and other measures among group II (Metabolic syndrome):

Variable	FT4	
	r	p-value
Age	-0.279	0.221
BMI	-0.183	0.310
WC	0.036	0.843

SBP	0.012	0.949
DBP	-0.253	0.163
FBG	0.168	0.357
Fasting insulin	-0.170	0.353
HOMA-IR	0.207	0.255
HBA1C	-0.057	0.756
TC	-0.036	0.844
TG	0.069	0.708
LDL	-0.132	0.473
HDL	**0.412**	**0.019**
Hb	-0.209	0.255
RBCs	-0.085	0.645
PLTs	0.259	0.153
TLC	**-0.492**	**0.004**
ALT	0.052	0.779
AST	0.034	0.855
Serum Albumin	-0.137	0.456
Total Bilirubin	0.004	0.893
Direct Bilirubin	-0.087	0.636
Creatinine	-0.133	0.469
Urea	-0.217	0.234
TSH	-0.103	0.757

Table 6: ***Correlation between TSH and other measures among group II (Metabolic syndrome):***

Variable	**TSH**	
	r	**p-value**
Age	0.071	0.701
BMI	-0.007	0.969
WC	0.337	0.059
SBP	-0.255	0.199
DBP	0.049	0.789
FBG	0.269	0.137
Fasting insulin	0.084	0.646
HOMA-IR	0.182	0.320
HBA1C	0.101	0.582
TC	-0.052	0.778
TG	-0.120	0.513
LDL	-0.034	0.851
HDL	-0.019	0.920
Hb	-0.147	0.423
RBCs	0.054	0.769
PLTs	-0.078	0.670
TLC	0.140	0.444
ALT	-0.306	0.088
AST	-0.281	0.120
Serum Albumin	**-0.388**	**0.028**
Total Bilirubin	0.155	0.397
Direct Bilirubin	0.192	0.393
Creatinine	0.290	0.108
Urea	0.068	0.713

Table 7: Logistic regression for significant risk factors for metabolic syndrome among participants:

Variable	B	S.E	Wald	O.R (95%C.I)	P-value
Old age	0.58	0.26	5.04	1.79 (1.07-3)	**0.02**
Smoking	0.63	0.16	14.7	1.88 (1.36-2.99)	**0.008**
Obesity	0.15	0.04	13.86	1.07 (1.07-1.26)	**0.001**
DM	1.03	0.35	8.67	2.8 (1.41-5.57)	**0.003**
HTN	0.13	0.04	7.60	1.14 (1.03-1.25)	**0.006**
Dyslipidemia	0.62	0.26	5.63	1.86 (1.11-3.12)	**0.018**

DM: Diabetes Mellitus HTN: Hypertension

DISCUSSION

In terms of demographic information, the current investigation revealed that there was no discernible difference in age or sex between cases with and without metabolic syndrome.

According to Santana et al. [13], there was no discernible difference in age or sex between elderly individuals with and without metabolic syndrome, which is consistent with the current investigation.

In a similar vein, Vieira et al. [14] found no discernible differences in age or sex between elderly patients with and without metabolic syndrome.

Furthermore, Silva et al. [15] found no statistically significant difference in age between elderly patients with and without metabolic syndrome. However, this study also found that patients with metabolic syndrome were more likely to be female than those without the condition; this discrepancy may have resulted from different inclusion criteria.

Gouveia et al. [16], in contrast to the current investigation, found a significant difference in age and sex between patients with and without metabolic syndrome. This difference in findings could be attributed to variations in the sample size and inclusion criteria.

The results of the current investigation, however, indicated that patients with metabolic syndrome had much higher BMIs and weights. Given that metabolic syndrome includes abdominal obesity, it was expected that patients with Mets would have much higher body weights.

Santana et al. [13] discovered a strong correlation between older patients' metabolic syndrome and greater BMI, which is consistent with the findings of the current investigation.

According to Gouveia et al. [16], there is a noteworthy correlation between a higher BMI and metabolic syndrome in older patients.

Silva et al.'s study [15], which is in line with the current one, also showed a strong correlation between obesity and metabolic syndrome in older adult patients.

Furthermore, it was observed by Vieira et al. [14] that there was no statistically significant difference in obesity between older individuals with and without metabolic syndrome.

The present investigation found that, in comparison to the control group, the MetS group had significantly higher systolic and diastolic blood pressure ($p \leq 0.001$).

Given that a major component of metabolic syndrome is hypertension, it was expected that patients with MetS would have significantly higher blood pressure.

Congruent with the present investigation, Emiroğlu et al. [17] demonstrated that individuals with MetS exhibited noticeably elevated SBP and DBP in contrast to those without the condition.

In keeping with the current investigation, Huo et al.'s [18] findings showed that the incidence of MetS rose in tandem with blood pressure increases.

Additionally, older patients with MetS had considerably greater SBP and DBP than older patients without MetS, according to research by Waring et al. [19].

According to the insulin hypothesis of hypertension, raised arterial pressure is caused by increased sympathetic activity and sodium reabsorption resulting from compensatory hyperinsulinemia associated with insulin resistance. This theory has multiple lines of evidence supporting it. First, the discovery that insulin resistance and hyperinsulinemia are evident in even lean patients with essential hypertension [20] provides direct proof of the relationship between increased blood pressure and insulin resistance.

The current study's findings regarding the glycemic profile showed that the MetS group's fasting insulin, FBG, HOMA-IR, and HBA1C were all significantly higher than those of the control group.

Santana et al. [13] found that senior patients with MetS had significantly greater FBG and HBA1c than those without MetS, which is consistent with the findings of the current investigation.

Furthermore, participants with MetS exhibited considerably higher FBG than those without MetS, according to Emiroğlu et al. [17]. Furthermore, older individuals with MetS had considerably higher HOMA-IR than senior patients without MetS, according to Kazukauskiene et al. [21].

Furthermore, older individuals with MetS have considerably greater FBG and HOMA-IR than senior patients without MetS, according to research by Waring et al. [19].

The lipid profile of the patients with MetS was found to be considerably lower ($p \leq 0.001$) in the HDL group and significantly higher (TC, TG, and LDL) in comparison to the control group, according to the study's results.

This is consistent with the findings of Santana et al. [13], who showed that older patients with MetS had significantly higher TG and significantly lower HDL in comparison to those without the condition. However, they did not find any significant differences in TC or LDL between patients with and without the condition; this discrepancy may be explained by the different sample sizes.

Additionally, in keeping with the current investigation, Gouveia et al. [16] and Lee et al. [22] found that a higher prevalence of MS in the elderly has been linked to lower HDL.

Furthermore, Subías-Perié et al. [23] found that an increased risk of multiple sclerosis existed in older people with increased triglycerides and decreased HDL.

The discrepancy in results between the current study and Silva et al.'s [19] investigation, which found no statistically significant differences in TC and LDL between individuals with and without MetS, could potentially be attributed to variations in sample size.

Based on the results of the present investigation, there was no significant difference in CBC between the two groups that were being studied ($p>0.05$)

However, Nebeck et al. [24] revealed found the components of MetS were favorably correlated ($P<0.05$) with hematologic indices (hemoglobin, hematocrit, and RBC).

Also, Chang et al. [25] revealed that hemoglobin (Hb) levels in both genders and platelet (PLT) were separate risk factors for MetS in men.

Ahmadzadeh et al. [26] also found that the counts of white blood cells, platelets, and hemoglobin increased in tandem with the growth of metabolic syndrome components ($p < 0.05$ for all).

The current study's small sample size could be the reason for the lack of a significant correlation between CBC and MetS.

In terms of renal function tests, the findings of this study demonstrated that patients with metabolic syndrome (MetS) had urea and creatinine levels that were considerably higher than those of the control group ($p<0.05$).

MetS affects profibrotic factors, microalbuminuria, glomerular hyperfiltration, RAAS, and podocyte damage, among other aspects of renal pathophysiology [27]. Numerous investigations have verified that Metabolic Syndrome (MetS) can result in modifications to the structure and function of the kidneys, including a reduction in glomerular filtration rate (GFR) and an increase in urine microalbumin [28]. According to a meta-analysis, people with MetS had a 1.34 times higher chance of developing CKD than people without MetS [29]. MetS was found to raise the risk of CKD by 50% in a different meta-analysis [30]. A multitude of research discovered a correlation between each MetS component and CKD. The likelihood of developing CKD increased with the number of components (odds ratio, 1.96; 95%: 1.71,2.34) [30]. Maleki et al. [31] revealed that there was a high prevalence of chronic kidney disease in patients with MetS compared with the subject without MetS.

The current investigation found that the ALT and AST levels of the MetS patients were considerably higher than those of the control group in terms of liver functioning ($p \leq 0.001$). There was no statistically significant difference ($p>0.05$) in S. albumin, total bilirubin, or direct bilirubin between the two study groups.

Salama et al. [32] found that patients with MetS had considerably higher liver enzymes than those without the condition ($p<0.001$), which is consistent with the findings of the current investigation.

As well, Kim & Han [33] revealed that the patients with MetS have significantly elevated ALT and AST compared to those without MetS ($p<0.001$).

Also, Liu et al. [34] indicated that the frequency of MetS in older populations is positively correlated with higher liver enzyme levels (mostly ALT, GGT, and ALP, but not AST).

The aspects of the metabolic syndrome are influenced by thyroid hormones, which also affect lipid metabolism. Positive correlations have been shown between TSH and LDL cholesterol and negative correlations between TSH and HDL cholesterol [35].

When comparing the thyroid profiles of the groups under investigation, the current study found that patients with MetS had statistically significant increases in TSH and FT3, but that

their group had statistically significant decreases in FT4 ($p<0.05$ for all). Metabolic syndrome prevalence was significantly influenced by thyroid function [36].

According to Heima et al. [37] and Waring et al. [19], who also found that older patients with MetS had significantly higher TSH than those without MetS, participants with TSH levels higher than normal had a higher frequency of metabolic syndrome than subjects with normal TSH levels. These findings are consistent with the current study.

Additionally, Zhang & Zhang [38] found that in elderly patients with early-stage type 2 diabetes, TSH levels were positively linked with both insulin resistance and LDL. In older patients who are just beginning to develop type 2 diabetes, higher TSH levels may have a role in the development of insulin resistance.

Furthermore, Emiroğlu et al. [17] reported that participants with MetS had a considerably greater level of fT3 than those without MetS, which is consistent with the current findings. However, they did not find any significant correlation between TSH and FT4 and MetS, which contrasts with our findings.

Moreover, Zhu et al. [39] demonstrated that high TSH ($P<0.05$) and decreased FT3 ($P<0.01$) in older adults were independent risk factors for MetS.

Nevertheless, Zhu et al. [39] shown that in older individuals, elevated TSH ($P<0.05$) and decreased FT3 ($P<0.01$) were independent risk factors for MS.

The current investigation found a statistically significant negative link ($p\leq0.05$) between FT3 and fasting insulin in patients with metabolic syndrome, when compared to other measurements. There was no statistically significant relationship ($p>0.05$) between FT3 and the other laboratory indicators.

According to Vyakaranam et al. [40], FT3 levels had a moderate correlation ($r= -0.38$, $P=0.04$) with HOMA IR and a negative and high correlation ($r= -0.5$, $P=0.004$) with insulin, which is consistent with the findings of the current investigation.

Adala et al. [41] found, however, that FT3 significantly correlated negatively with HDL, FBG, cholesterol, and HbA1C in patients with MetS

The current study's findings revealed a statistically significant positive relationship between HDL and FT4 in respect to the association between FT4 and other measures among individuals with metabolic syndrome. There was a statistically significant negative correlation ($p\leq0.05$) between TLC and FT4. There was no statistically significant correlation between FT4 and the other laboratory indicators ($p>0.05$).

Huang et al.'s study [42] revealed a positive correlation between FT4 and HDL-C in patients with MetS, which is in line with the current investigation's findings. Punekar et al.'s study [43] also showed a statistically significant negative association between FT4 and TLC and a statistically significant positive correlation between FT4 and HDL.

Furthermore, Wang et al.'s findings [44] that elevated blood FT4 is a reliable indicator of dyslipidemia regression corroborated our findings.

Adala et al. [41] did, however, demonstrate that FT4 exhibited a significant negative connection with HbA1C and a strong positive correlation with cholesterol in patients with MetS.

Additionally, FT4 levels and insulin and IR showed a modest and negative connection ($r = -0.11$, $P = 0.54$; $r = -0.07$, $P = 0.69$, respectively) according to Vyakaranam et al. [40].

Furthermore, FT4 levels and HOMA-IR revealed a negative correlation, as demonstrated by Kocatürk et al. [45].

The current investigation demonstrated a statistically significant negative connection ($p\leq0.05$) between TSH and S.albumin, with respect to other parameters among individuals with metabolic syndrome. TSH and other laboratory markers did not correlate statistically significantly ($p>0.05$).

Punekar et al.'s [43] statistically significant negative association between TSH and S.albumin was demonstrated, which is consistent with the findings of the current investigation.

On the other hand, TSH was found to be substantially adversely linked with FBG and HOMA_IR in patients with MetS by Adala et al. [41].

Furthermore, TSH and homocysteine, high-density lipoprotein cholesterol, and triglycerides have linear correlations, as shown by Zhu et al. [39] (all $P < 0.05$).

Additionally, TSH levels were found to have a moderately positive connection with insulin ($r = 0.43$, $P = 0.03$) and HOMA IR ($r = 0.48$; $P = 0.01$) by Vyakaranam et al. [40].

Likewise, Kocatürk et al.'s research [45] showed a positive correlation between TSH levels and HOMA-IR.

Logistic regression analysis showed that old age, smoking, obesity, DM, HTN & dyslipidemia were significant risk factors for metabolic syndrome ($p\leq0.05$).

The association between MetS with older age, obesity, and smoking, may be explained by the

reduced physical activity among patients with age, obesity, and smoking.

Growing older has long been recognized as a separate risk factor for metabolic syndrome [46]. Age-related decreases in several physiological characteristics and lifetime adoption of unhealthy lifestyles that significantly raise metabolic risk factors account for this [47].

In concordance with the current study Gouveia et al. [16] showed that older age, female sex and BMI were independent predictors for MetS in elderly.

Also, Tadewos et al. [48] showed obesity, overweight, and age over 60 were linked risk factors for MetS.

Kim and colleagues [49] also discovered that smokers had a 2.4-fold increased incidence of metabolic syndrome (95% confidence interval [CI], 1.43–3.96) in comparison to non-smokers. Sun and colleagues (2019) also concluded that active smoking was associated with the onset of metabolic syndrome. There appears to be a decreased risk of metabolic syndrome among those who stop smoking.

Obesity, metabolic syndrome, and type-2 diabetes mellitus are three illnesses that are typically connected with cardiovascular problems: they share multiple pathophysiological mechanisms and are known to exacerbate one another's symptoms [51].

Our results were supported by Stanciu et al. [52], who demonstrated that obesity is strongly associated with hypertension and plays a substantial role in the aetiology of metabolic syndrome. Nevertheless, it is the main risk factor for increasing cardiovascular mortality and morbidity.

One component of the metabolic syndrome is dyslipidemia. According to Haile et al. [53], dyslipidemia was found to be independently predicted by age, higher BMI, central obesity, hypertension, and elevated blood glucose levels in patients with MetS.

Limitations: sample size was relatively small, and the study did not include a follow-up period to assess changes in thyroid function or metabolic parameters over time.

Recommendations: Large-population follow-up cohort studies and studies with longer follow-up periods are needed to determine the significance of early detection of thyroid dysfunction, particularly in the subclinical form, and the long-term association with metabolic syndrome in different age, sex, and BMI groups. Additional larger studies as well as individual participant data meta-analyses that standardize definitions and statistical methods are warranted to help elucidate associations between metabolic syndrome and thyroid dysfunction. Future studies should consider dietary habits and genetic predispositions which are important to investigate. Additional investigations are required to determine the potential benefits of lifestyle modifications, such as weight loss and improved metabolic control, in reducing the risk of thyroid dysfunction in individuals with MetS.

CONCLUSION

In conclusion, there is a strong association between thyroid dysfunction and metabolic syndrome, thyroid dysfunction and HOMA-IR. Patients with hypothyroidism and subclinical hypothyroidism had an increased risk of metabolic syndrome.

Authors' Contributions:

E.G designed the study as well as writing the manuscript in a proper scientific manner with assistance from M.M and A.S. M.M, A.S and E.G performed the statistical analysis, results' interpretation, patients' clinical assessment. S.H performed laboratory investigations. All authors discussed the results and commented on the manuscript and contributed to the writing of the final manuscript.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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