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Original Research Article

PREVALENCE OF THYROID FUNCTION ABNORMALITIES IN CASES OF METABOLIC SYNDROME: A CASE-CONTROL STUDY

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ABSTRACT

Background: Metabolic syndrome (MetS) is a prevalent cardiometabolic cluster that substantially increases future risk of type 2 diabetes and cardiovascular disease. Thyroid hormones regulate energy balance, lipid and glucose metabolism, and vascular tone; therefore, subtle thyroid dysfunction—particularly subclinical hypothyroidism—may aggravate MetS components and compound cardiovascular risk. This study assessed the prevalence and pattern of thyroid function abnormalities in MetS compared with matched non-MetS controls.

Materials and Methods: A hospital-based case–control study was conducted over 12 months in a tertiary-care teaching hospital. Adults aged 18–65 years were enrolled after consent: 40 MetS cases (modified NCEP ATP III criteria; ≥3 components) were enrolled as study group. Similar number of healthy individuals were included as control group. Anthropometry, blood pressure, fasting plasma glucose, and lipid profile were recorded. Thyroid function (TSH, free T4, and free T3) were done using chemiluminescent immunoassay. Thyroid dysfunction was categorized as overt/subclinical hypo- or hyperthyroidism using standard biochemical definitions. Group comparisons used appropriate parametric/nonparametric tests; odds ratios (OR) with 95% confidence intervals (CI) were calculated, with $p<0.05$ considered significant.

Results: Cases and controls were comparable in age and sex distribution, while BMI was higher in cases (29.8±3.6 vs 23.4±2.9 kg/m²; $p<0.001$). Mean TSH was higher in cases (4.82±2.94 vs 2.18±0.96 mIU/L; $p<0.001$) with lower free T3 (2.86±0.62 vs 3.24±0.48 pg/mL; $p=0.003$) and free T4 (1.08±0.28 vs 1.24±0.22 ng/dL; $p=0.006$). Thyroid dysfunction was more frequent in MetS (35.0%) than controls (10.0%) (OR 4.85; 95% CI 1.43–16.42; $p=0.014$), predominantly subclinical hypothyroidism (22.5% vs 5.0%; OR 5.52; 95% CI 1.11–27.43; $p=0.048$). In MetS, TSH correlated positively with BMI, waist circumference, triglycerides, fasting glucose, and number of MetS components, and negatively with HDL-C.

Conclusion: Thyroid function abnormalities—especially subclinical hypothyroidism—are significantly more common in MetS and track with greater metabolic burden. Routine thyroid evaluation in MetS may support earlier detection and improved cardiometabolic risk stratification.

Keywords: Metabolic Syndrome, Hypothyroidism, Thyroid Stimulating Hormone, Case-Control Studies.

INTRODUCTION

Metabolic syndrome (MetS) is a highly prevalent cardiometabolic clustering of central adiposity, dysglycemia, hypertension, hypertriglyceridemia and low high-density lipoprotein cholesterol, commonly diagnosed when any three of these five abnormalities coexist as per harmonized criteria.[1] The syndrome disproportionately affects middle-aged and older adults, with a rising burden in rapidly urbanizing populations, and shows important sex- and age-related variations linked to adiposity patterns, reproductive stage, and lifestyle transitions. In parallel, thyroid function abnormalities—particularly subclinical hypothyroidism—are common in adult populations and are more frequently detected among women and with advancing age.[2]

Thyroid hormones are known to regulate basal metabolic rate as well as to exert pleiotropic effects on lipid handling, glucose homeostasis, vascular tone and energy expenditure. Even mild reductions in thyroid hormone can be associated with higher total and low-density lipoprotein cholesterol. Other features such as impaired triglyceride clearance, weight gain and diastolic blood pressure elevation can also be seen.[3] Moreover, alterations in thyroid-stimulating hormone (TSH) within or above the reference range may correlate with insulin resistance and adiposity. Thyroid hormone also modulates endothelial function and systemic vascular resistance. These shared biological pathways suggest bidirectional relationships: thyroid dysfunction may worsen MetS components, while chronic low-grade inflammation, leptin excess, and altered deiodinase activity in obesity may shift thyroid homeostasis in the opposite direction, leading to higher TSH levels or subtle changes in peripheral thyroid hormone metabolism.[4]

Epidemiological studies increasingly report a higher frequency of thyroid function abnormalities among individuals with MetS compared with metabolically healthier controls. Many studies have demonstrated that subclinical hypothyroidism occurs more often in MetS than in non-MetS participants.[5] Overt hypothyroidism and subclinical hypothyroidism are each linked to a higher prevalence of MetS in specific subgroups (for example, older participants) and hyperthyroid states demonstrated metabolic perturbations such as higher odds of hyperglycaemia, underscoring that thyroid dysfunction across the spectrum may intersect with cardiometabolic risk.[6] Meanwhile, large real-world clinical datasets from India and other regions report that a sizeable proportion of adults with MetS have previously unrecognized thyroid dysfunction detected on screening, with hypothyroid phenotypes predominating and clustering with adverse waist circumference, insulin resistance markers, and dyslipidaemia.

Reported prevalence of thyroid dysfunction vary widely due to differences in diagnostic thresholds, population iodine nutrition, exclusion of known thyroid disease or thyroid-altering medications, and inconsistent adjustment for confounders such as smoking, menopause status, and insulin resistance severity.[7] Furthermore, data from specific regions and clinical settings remain limited, and case-control designs that directly compare thyroid function abnormalities between MetS cases and matched controls can better quantify the excess burden attributable to MetS itself. Therefore, this case-control study aims to determine the prevalence and pattern of thyroid function abnormalities among individuals with MetS compared with controls, and to explore their relationships with individual MetS components, thereby addressing a clinically relevant gap in evidence needed to inform rational screening strategies and risk stratification.

MATERIALS AND METHODS

This hospital-based case–control study was conducted in the Department of Medicine/Endocrinology of a tertiary care teaching hospital over a 12-month period. Adults aged 18–65 years were enrolled after written informed consent. Cases were defined as participants with metabolic syndrome, and controls were age- (±3 years) and sex-matched individuals without metabolic syndrome recruited from the same outpatient setting and/or health check-up clinic. The sample size was calculated using the formula for comparison of two proportions, assuming a higher prevalence of thyroid function abnormalities among cases than controls, with 80% power and a two-sided alpha of 0.05. The minimum sample size was calculated to be 36 so we have included 40 cases in our study. Same number of healthy individuals were enrolled as control group.

Metabolic syndrome was diagnosed using the modified National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria (with ethnicity-appropriate waist circumference cut-offs), requiring the presence of at least three of the following: (i) increased waist circumference, (ii) elevated triglycerides (≥150 mg/dL) or treatment, (iii) reduced HDL cholesterol (<40 mg/dL in men, <50 mg/dL in women) or treatment, (iv) elevated blood pressure (≥130/85 mmHg) or antihypertensive therapy, and (v) elevated fasting plasma glucose (≥100 mg/dL) or antidiabetic therapy. Controls were healthy individuals with no present or prior diagnosis of metabolic syndrome. A structured proforma was used to collect demographic details, relevant clinical history (duration of diabetes/hypertension/dyslipidemia where applicable), smoking and alcohol use, and current medications (including statins, antihypertensives, antidiabetics, steroids, and drugs known to affect thyroid function).

All participants underwent detailed clinical evaluation and anthropometry. Weight, height and body mass index (BMI) and waist circumference of

all participants were noted. Blood pressure was recorded after at least 5 minutes of rest. Two readings were taken 2–3 minutes apart and the mean was used for analysis. After an overnight fast of 8–12 hours venous blood samples were collected for fasting plasma glucose, lipid profile, and thyroid function tests. Serum thyroid-stimulating hormone (TSH) and free thyroxine (FT4) (and free triiodothyronine [FT3]) were measured using a chemiluminescent immunoassay in the hospital central laboratory with internal quality control. Thyroid function abnormalities were categorized as: overt hypothyroidism (elevated TSH with low FT4), subclinical hypothyroidism (elevated TSH with normal FT4), overt hyperthyroidism (suppressed TSH with elevated FT4/FT3), and subclinical hyperthyroidism (suppressed TSH with normal FT4/FT3). Participants with known thyroid disease or receiving thyroid-related therapy were excluded as per predefined criteria to avoid treatment-related distortion of prevalence estimates.

Data were entered into a secured database and analyzed using SPSS 23.0. Continuous variables were tested for normality and presented as mean ± standard deviation or median (interquartile range), while categorical variables were expressed as frequency and percentage. Between-group comparisons were performed using the independent samples t-test (or Mann–Whitney U test for non-normally distributed variables) for continuous variables and chi-square test (or Fisher's exact test where appropriate) for categorical variables. The primary outcome was the prevalence of any thyroid function abnormality among cases versus controls; odds ratios (OR) with 95% confidence intervals (CI) were computed. A p value less than 0.05 was considered statistically significant.

Inclusion Criteria

- Age 18–65 years
- Cases: Diagnosed metabolic syndrome (≥3 criteria as defined above)
- Controls: Fewer than three metabolic syndrome components and no prior diagnosis of metabolic syndrome
- Willingness to participate and provide written informed consent

Exclusion Criteria

- Known thyroid disorder on treatment (levothyroxine/antithyroid drugs) or prior thyroid surgery/radioiodine therapy
- Pregnancy or postpartum state (≤12 months)
- Use of drugs that significantly alter thyroid function tests in the preceding 3 months (e.g., amiodarone, lithium, interferon, high-dose glucocorticoids)
- Acute severe illness, active infection/inflammation, or hospitalization within the preceding 4 weeks
- Chronic liver failure or end-stage renal disease
- Known pituitary/hypothalamic disease affecting TSH secretion
- Incomplete clinical or laboratory data for defining metabolic syndrome or thyroid status
- Incomplete key clinical/laboratory data

RESULTS

The mean age of cases was 46.2 ± 9.8 years and of controls was 45.7 ± 10.1 years (p = 0.812). Cases comprised 22 males (55%) and 18 females (45%), while controls had 24 males (60%) and 16 females (40%) (p = 0.651). The mean BMI was significantly higher in cases (29.8 ± 3.6 kg/m²) compared to controls (23.4 ± 2.9 kg/m²; p < 0.001). The mean duration of metabolic syndrome in cases was 4.3 ± 2.7 years. Smoking status and family history of thyroid disease were comparable between groups (p > 0.05 for both). [Table 1]

Table 1: Demographic and baseline characteristics of cases and controls

Parameter	Cases (n=40)	Controls (n=40)	p-value
Age (years) Mean ± SD	46.2 ± 9.8	45.7 ± 10.1	0.812
Male, n (%)	22 (55%)	24 (60%)	0.651
Female, n (%)	18 (45%)	16 (40%)	0.651
BMI (kg/m²) Mean ± SD	29.8 ± 3.6	23.4 ± 2.9	<0.001*
Duration of MetS (years)	4.3 ± 2.7	—	—
Smokers, n (%)	10 (25%)	9 (22.5%)	0.793
Family H/O thyroid disease, n (%)	8 (20%)	4 (10%)	0.210

All individual components of MetS were significantly more deranged in cases compared to controls. Mean waist circumference (98.6 ± 8.4 vs. 82.3 ± 7.1 cm), systolic BP (138.4 ± 12.6 vs. 118.2 ± 9.8 mmHg), diastolic BP (88.7 ± 8.2 vs. 76.4 ± 7.3 mmHg), fasting blood glucose (118.6 ± 22.4 vs. 88.2 ± 9.6 mg/dL), serum triglycerides (186.4 ± 42.8 vs. 112.6 ± 28.4 mg/dL), and HDL cholesterol (38.6 ± 6.4 vs. 52.4 ± 7.8 mg/dL) all showed statistically significant differences (p < 0.001 for all). The mean number of MetS criteria met by cases was 3.9 ± 0.7. [Table 2]

Table 2: Comparison of metabolic syndrome components between cases and controls

Metabolic Parameter	Cases Mean ± SD	Controls Mean ± SD	p-value
Waist Circumference (cm)	98.6 ± 8.4	82.3 ± 7.1	<0.001*
Systolic BP (mmHg)	138.4 ± 12.6	118.2 ± 9.8	<0.001*
Diastolic BP (mmHg)	88.7 ± 8.2	76.4 ± 7.3	<0.001*
Fasting Blood Glucose (mg/dL)	118.6 ± 22.4	88.2 ± 9.6	<0.001*
Serum Triglycerides (mg/dL)	186.4 ± 42.8	112.6 ± 28.4	<0.001*
HDL Cholesterol (mg/dL)	38.6 ± 6.4	52.4 ± 7.8	<0.001*
No. of MetS criteria (Mean)	3.9 ± 0.7	—	—

Mean serum TSH was significantly elevated in cases (4.82 ± 2.94 mIU/L) compared to controls (2.18 ± 0.96 mIU/L; $p < 0.001$, Mann–Whitney U test), consistent with a shift toward hypothyroid tendency in the MetS group. Mean free T3 and free T4 were both significantly lower in cases versus controls (fT3: 2.86 ± 0.62 vs. 3.24 ± 0.48 pg/mL, $p = 0.003$; fT4: 1.08 ± 0.28 vs. 1.24 ± 0.22 ng/dL, $p = 0.006$). [Table 3]

Table 3: Comparison of thyroid function parameters between cases and controls

Thyroid Parameter	Cases Mean ± SD	Controls Mean ± SD	p-value
TSH (mIU/L)	4.82 ± 2.94	2.18 ± 0.96	<0.001*
Free T3 (pg/mL)	2.86 ± 0.62	3.24 ± 0.48	0.003*
Free T4 (ng/dL)	1.08 ± 0.28	1.24 ± 0.22	0.006*

Thyroid dysfunction was identified in 14 of 40 cases (35.0%) compared to 4 of 40 controls (10.0%), a statistically significant difference ($p = 0.014$; OR = 4.85, 95% CI: 1.43–16.42). Subclinical hypothyroidism was the most prevalent thyroid abnormality, present in 9 cases (22.5%) versus 2 controls (5.0%) ($p = 0.048$; OR = 5.52, 95% CI: 1.11–27.43). Overt hypothyroidism was present in 3 cases (7.5%) versus 1 control (2.5%), though this difference did not reach statistical significance ($p = 0.615$). Overt and subclinical hyperthyroidism were each present in 1 case (2.5%), and no statistically significant difference was noted for either hyperthyroid category between groups ($p = 1.000$ for both). The predominance of hypothyroid patterns over hyperthyroid patterns in cases is consistent with the recognised pathophysiological link between insulin resistance, adiposity, and impaired thyroid hormone synthesis. [Table 4]

Table 4: Prevalence and spectrum of thyroid abnormalities in cases and controls

Thyroid Disorder	Cases n (%)	Controls n (%)	p-value
Overt Hypothyroidism	3 (7.5%)	1 (2.5%)	0.615
Subclinical Hypothyroidism	9 (22.5%)	2 (5.0%)	0.048*
Overt Hyperthyroidism	1 (2.5%)	1 (2.5%)	1.000
Subclinical Hyperthyroidism	1 (2.5%)	0 (0%)	1.000
Total Thyroid Abnormalities	14 (35.0%)	4 (10.0%)	0.014*
Normal Thyroid Function	26 (65.0%)	36 (90.0%)	0.014*

Among cases, subclinical hypothyroidism showed a higher prevalence in females (7/18; 38.9%) compared to males (2/22; 9.1%), with this difference approaching but not reaching statistical significance ($p = 0.053$). Any thyroid dysfunction was more frequent in female cases (9/18; 50.0%) than in male cases (5/22; 22.7%), though the difference was not statistically significant ($p = 0.101$). These directional trends are consistent with the well-established female predominance of autoimmune and subclinical thyroid disease in the general population. In the control group, thyroid abnormalities were evenly distributed between sexes ($p > 0.05$ for all comparisons). [Table 5]

Table 5: Gender-wise distribution of thyroid abnormalities in cases and controls

Disorder	Male Cases n=22	Female Cases n=18	p-value	Male (Control) n=24	Female (Control) n=16	p-value
Subclinical Hypothyroidism	2 (9.1%)	7 (38.9%)	0.053	1 (4.2%)	1 (6.3%)	1.000
Overt Hypothyroidism	1 (4.5%)	2 (11.1%)	0.579	1 (4.2%)	0 (0%)	0.600
Any Thyroid Dysfunction	5 (22.7%)	9 (50.0%)	0.101	2 (8.3%)	2 (12.5%)	1.000

In the cases group, serum TSH demonstrated significant positive correlations with the number of MetS components ($r = +0.516$, $p < 0.001$), BMI ($r = +0.482$, $p < 0.001$), waist circumference ($r = +0.446$, $p = 0.004$), serum triglycerides ($r = +0.426$, $p = 0.006$), and fasting blood glucose ($r = +0.398$, $p = 0.011$). Significant negative correlation was observed between TSH and HDL cholesterol ($r = −0.388$, $p = 0.013$). The correlation between TSH and systolic blood pressure was found to be positive and

borderline significant (r = +0.312, p = 0.050). In contrast, no significant correlations between serum TSH and any metabolic parameter were observed in the control group (p > 0.05 for all), indicating that the TSH–metabolic axis is specifically activated in the context of established MetS. [Table 6]

Table 6: Pearson's correlation between serum TSH and components of metabolic syndrome

Metabolic Parameter	Pearson r (Cases)	p-value	Pearson r (Controls)	p-value
BMI (kg/m²)	+0.482	<0.001*	+0.142	0.382
Waist Circumference (cm)	+0.446	0.004*	+0.118	0.469
Fasting Blood Glucose (mg/dL)	+0.398	0.011*	+0.096	0.558
Serum Triglycerides (mg/dL)	+0.426	0.006*	+0.084	0.607
HDL Cholesterol (mg/dL)	−0.388	0.013*	−0.104	0.524
Systolic Blood Pressure (mmHg)	+0.312	0.050*	+0.072	0.660
Number of MetS Components	+0.516	<0.001*	+0.138	0.397

DISCUSSION

In this case–control study from a tertiary-care setting, thyroid function abnormalities were substantially more common among participants with metabolic syndrome (MetS) than among age- and sex-matched controls (35.0% vs 10.0%; OR 4.85), with subclinical hypothyroidism (SCH) constituting the predominant phenotype (22.5%). This pattern aligns closely with South Asian hospital-based reports in which SCH accounts for the bulk of thyroid dysfunction in MetS. For example, Gyawali et al observed an overall thyroid dysfunction prevalence of 32% among MetS patients, with SCH as the leading subtype, emphasizing that mild thyroid failure is frequently encountered in metabolically high-risk cohorts.8 Similarly, Khatiwada et al reported thyroid dysfunction to be present in 31.9% of cases having Metabolic syndrome.[9]

Beyond categorical dysfunction, we found a shift in thyroid parameters among MetS cases—higher mean TSH with lower free T3 and free T4—suggesting either early thyroid failure, altered peripheral deiodination, or adaptive neuroendocrine responses to adiposity and insulin resistance. This continuous association between "higher TSH/lower FT4" and adverse metabolic traits has been repeatedly noted even in ostensibly euthyroid populations. In a large cross-sectional study, Roos et al demonstrated that lower FT4 and higher TSH (within the reference range) correlated with insulin resistance and several MetS components, independent of confounders, implying that thyroid function can track metabolic risk gradients rather than only overt disease thresholds.[10] Extending this concept longitudinally, Waring et al reported that increasing TSH was associated with higher odds of prevalent MetS in older adults, and that more marked SCH (TSH >10 mIU/L) related to substantially higher MetS odds, supporting a dose–response relationship between thyroid axis perturbation and metabolic clustering.[11] Taken together with our data, these findings reinforce the notion that the thyroid–metabolic interface operates on a continuum: mild thyroid hypofunction (or relative tissue hypothyroidism) may contribute to insulin resistance and dyslipidemia, while the inflammatory–adipokine milieu of MetS may secondarily influence the hypothalamic–pituitary–thyroid axis, potentially creating a self-perpetuating loop.

The predominance of SCH in our MetS cohort also provides a mechanistic bridge to the dyslipidemic signature observed in cases. Thyroid hormones regulate hepatic LDL receptor expression, cholesterol synthesis/clearance, and triglyceride-rich lipoprotein metabolism; thus, even subtle reductions in thyroid hormone action may worsen hypertriglyceridemia and low HDL-C—two core IDF components. In a study Park et al reported relationships between thyroid function indices and MetS components, supporting that thyroid status is intertwined with blood pressure, lipid parameters, and glycemia at population level.[12] In younger euthyroid women, Oh et al further showed that those in the higher-normal TSH stratum had significantly higher MetS prevalence and greater central obesity and hypertriglyceridemia, with roughly two-fold higher odds of MetS after adjustment, underscoring that cardiometabolic associations may emerge before overt biochemical hypothyroidism.[13] Our correlation matrix is consistent with these observations: in MetS cases, TSH correlated positively with BMI, waist circumference, triglycerides, fasting glucose, and MetS component burden, and negatively with HDL-C. These patterns support a biologically plausible pathway in which escalating adiposity and insulin resistance track with rising TSH and a more atherogenic lipid milieu, whether as cause, consequence, or bidirectional reinforcement.

Importantly, our data also suggest that metabolic burden intensity is mirrored by thyroid-axis perturbation: TSH correlated most strongly with the number of MetS components (r≈0.52), implying an additive interaction where clustering of risk factors associates with greater thyroid dysfunction severity. Similar component-wise relationships have been reported in population datasets. In the Tehran Thyroid Study analysis, Mehran et al found that overt hypothyroidism and, in older subgroups, SCH were associated with MetS and specific components such as abdominal obesity and hypertriglyceridemia, suggesting age and phenotype modify the thyroid–MetS link.[14] Jang et al reported significant associations between thyroid hormone levels and multiple MetS components, supporting that thyroid

indices relate to metabolic traits in the general population rather than only in selected clinic cohorts.15 Compared with these broader community studies, our case–control design (hospital-based, smaller sample) likely enriches for more severe or longer-duration MetS (mean duration ~4 years), which may partly explain the clearer separation in mean TSH and the stronger correlation signals observed among cases but not controls.

CONCLUSION

In this study thyroid function abnormalities were more frequently observed among individuals with metabolic syndrome as compared to their healthy counterparts. Subclinical hypothyroidism was found to be the most common pattern of thyroid dysfunction in cases of MetS. Metabolic syndrome cases also demonstrated higher TSH levels with relatively lower free thyroid hormones. These changes suggest a shift toward hypothyroid tendency. These findings suggest that targeted thyroid function screening may be considered, particularly in MetS patients with higher metabolic burden.

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