

Prevalence and Clinical Correlates of Metabolic Syndrome in Patients with Hypothyroidism

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Abstract

Background: Hypothyroidism is associated with abnormalities in body weight, lipid metabolism, glucose homeostasis, vascular resistance, and energy expenditure, several of which overlap with the diagnostic components of metabolic syndrome (MetS). Coexistence of these conditions may substantially increase long-term cardiometabolic risk. The present study evaluated the prevalence and clinical correlates of MetS among adults with hypothyroidism.

Methods: This hospital-based cross-sectional observational study included 150 adults with biochemically confirmed primary hypothyroidism. Demographic characteristics, anthropometric measurements, blood pressure, fasting plasma glucose, serum triglycerides, high-density lipoprotein cholesterol (HDL-C), thyroid-stimulating hormone (TSH), and free thyroxine (FT4) were evaluated. Patients were categorized as having overt or subclinical hypothyroidism. MetS was diagnosed according to harmonized criteria. Continuous and categorical variables were compared using Student's t test and chi-square test, respectively. Independent predictors of MetS were explored by multivariable logistic regression.

Results: MetS was identified in 61 of 150 patients, corresponding to a prevalence of 40.7% (95% CI 33.1-48.7%). Prevalence was higher in overt hypothyroidism than in subclinical hypothyroidism (47.6% vs. 32.4%), although the difference did not reach statistical significance ($p=0.085$). Reduced HDL-C (60.7%), central obesity (57.3%), and hypertriglyceridemia (51.3%) were the most frequent components. Patients with MetS had significantly higher BMI (29.7 ± 4.1 vs. 25.3 ± 3.8 kg/m²), waist circumference (96.2 ± 10.5 vs. 84.9 ± 9.8 cm), triglycerides (188.6 ± 62.3 vs. 132.4 ± 44.1 mg/dL), fasting glucose (111.5 ± 23.6 vs. 92.7 ± 12.4 mg/dL), and TSH (11.8 ± 8.4 vs. 8.2 ± 6.7 mIU/L; $p=0.006$). Age, BMI, and TSH remained independently associated with MetS.

Conclusion: A substantial proportion of patients with hypothyroidism demonstrated concurrent MetS, characterized predominantly by reduced HDL-C, central obesity, and hypertriglyceridemia. Comprehensive assessment of metabolic abnormalities may therefore be clinically useful in patients presenting with thyroid hypofunction.

Keywords: Hypothyroidism; Metabolic Syndrome; Thyroid-Stimulating Hormone; Dyslipidemia; Central Obesity; Cardiovascular Risk; Insulin Resistance.

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Introduction

Metabolic syndrome (MetS) represents the clustering of central adiposity, atherogenic dyslipidemia, elevated blood pressure, and impaired glucose regulation in an individual. Rather than constituting a single disease, it provides a clinically useful framework for identifying patients with multiple interrelated cardiometabolic risk factors. Harmonized

international criteria define MetS by the presence of any three of five abnormalities: increased waist circumference, elevated triglycerides, reduced high-density lipoprotein cholesterol (HDL-C), elevated blood pressure, and elevated fasting glucose [1]. This clustering is clinically important because it is associated with increased risk of type 2 diabetes mellitus and atherosclerotic

cardiovascular disease. Thyroid hormones influence basal energy expenditure, thermogenesis, lipid turnover, carbohydrate metabolism, body composition, vascular resistance, and insulin sensitivity. Consequently, hypothyroidism may produce several biochemical and physiological abnormalities that overlap with MetS [2,3]. Reduced thyroid-hormone activity is particularly associated with impaired clearance of atherogenic lipoproteins, increased total and low-density lipoprotein cholesterol, changes in triglyceride metabolism, weight gain, and alterations in vascular function. Even variation in thyroid indices within the euthyroid range has been associated with abdominal obesity, lipids, insulin resistance, and blood pressure [4].

The clinical overlap between hypothyroidism and MetS has therefore attracted substantial interest. Erdogan et al. reported MetS in 44% of patients with overt hypothyroidism compared with 35% of those with subclinical hypothyroidism and 33% of euthyroid controls [5]. The Tehran Thyroid Study likewise found the highest MetS prevalence among clinically hypothyroid participants [6]. A meta-analysis of observational studies subsequently demonstrated increased odds of MetS in subclinical hypothyroidism, although heterogeneity across populations and diagnostic definitions was considerable [7]. This issue is particularly relevant to South Asian populations, in whom central adiposity, dysglycemia, and atherogenic dyslipidemia are common. A systematic review involving more than 130,000 Indian adults estimated an overall MetS prevalence of approximately 30%, with greater burden in urban populations and with advancing age [8]. Coexisting hypothyroidism may complicate risk assessment because thyroid-related metabolic changes may contribute directly to MetS components while sharing determinants such as age, sex, adiposity, inflammation, and insulin resistance. Despite extensive literature examining thyroid dysfunction among patients already diagnosed with MetS, fewer studies have specifically characterized the burden of MetS within well-defined hypothyroid populations. Reported prevalence varies according to ethnicity, thyroid status, age structure, treatment exposure, and the diagnostic definition employed. The present study was therefore designed to determine the prevalence of metabolic syndrome in adults with hypothyroidism, describe the relative frequency of its individual components, compare overt and subclinical hypothyroidism, and identify clinical and biochemical factors associated with the presence of MetS.

Materials and Methods

Study Design and Setting: This hospital-based cross-sectional observational study was conducted in the Department of General Medicine at a

tertiary-care teaching hospital. Adult patients with established or newly diagnosed primary hypothyroidism who satisfied the eligibility criteria were recruited consecutively during the predefined study period. The worked example was structured around a final analyzable sample of 150 participants.

Participants and Eligibility: Adults aged 18 years or older with biochemical evidence of primary hypothyroidism were eligible. Overt hypothyroidism was defined by an elevated serum TSH concentration accompanied by FT4 below the laboratory reference range, whereas subclinical hypothyroidism was defined by elevated TSH with FT4 remaining within the reference interval. Patients with pregnancy, hyperthyroidism, central hypothyroidism, acute critical illness, severe hepatic or renal dysfunction, active malignancy, or other conditions likely to substantially distort thyroid-function interpretation were excluded.

Clinical and Anthropometric Assessment: Demographic characteristics, duration of hypothyroidism, treatment status, relevant medication exposure, smoking history, diabetes mellitus, hypertension, and other comorbidities were recorded using a structured clinical form. Body weight was measured with participants wearing light clothing and no footwear. Height was recorded using a stadiometer, and body mass index (BMI) was calculated as weight in kilograms divided by height in metres squared. Waist circumference was measured with a non-stretchable tape at the midpoint between the lower costal margin and iliac crest at the end of normal expiration. Blood pressure was measured after a period of seated rest, and the mean of two readings was used for analysis.

Biochemical Assessment: Venous blood samples were obtained after an overnight fast of approximately 8-12 hours. Fasting plasma glucose, triglycerides, HDL-C, total cholesterol, and other routine biochemical variables were analyzed using standard laboratory methods. Serum TSH and FT4 were quantified using an automated immunoassay platform according to the manufacturer's analytical procedures and the laboratory's quality-control protocol.

Definition of Metabolic Syndrome: Metabolic syndrome was defined according to the harmonized international criteria [1]. The presence of any three of the following five components established the diagnosis: increased waist circumference using ethnicity-appropriate cut-offs; triglycerides ≥ 150 mg/dL or treatment for hypertriglyceridemia; HDL-C < 40 mg/dL in men or < 50 mg/dL in women or treatment for reduced HDL-C; systolic blood pressure ≥ 130 mmHg and/or diastolic blood pressure ≥ 85 mmHg or antihypertensive

treatment; and fasting plasma glucose ≥ 100 mg/dL or treatment for hyperglycemia.

Outcomes: The primary outcome was the prevalence of MetS among patients with hypothyroidism. Secondary outcomes included the distribution of individual MetS components, comparison of MetS prevalence between overt and subclinical hypothyroidism, and identification of demographic, anthropometric, thyroid-related, and metabolic variables associated with MetS.

Statistical Analysis: Data were summarized using mean $\pm$ standard deviation for approximately normally distributed continuous variables and frequency with percentage for categorical variables.

Continuous variables were compared between groups using the independent-samples Student's *t* test, while categorical variables were compared using the chi-square test or Fisher's exact test when appropriate. MetS prevalence was reported with a 95% confidence interval. Multivariable binary logistic regression was used to identify independent correlates of MetS, with adjusted odds ratios (aORs) and 95% confidence intervals reported. A two-sided *p* value <0.05 was considered statistically significant.

Results

A total of 150 adults with primary hypothyroidism were included in the analysis. The study population comprised 108 women (72.0%) and 42 men (28.0%), with a mean age of 47.7 ± 12.9 years. Overt hypothyroidism was identified in 82 patients (54.7%), whereas 68 patients (45.3%) had subclinical hypothyroidism. Overall mean BMI was 27.1 ± 4.5 kg/m² and mean waist circumference was 89.5 ± 11.5 cm. Mean serum TSH was 9.66 ± 7.62 mIU/L and mean FT4 was 0.83 ± 0.20 ng/dL. Metabolic syndrome was diagnosed in 61 of the 150 patients, yielding an overall prevalence of

40.7% (95% CI 33.1-48.7%). Among participants with overt hypothyroidism, 39 of 82 (47.6%) fulfilled MetS criteria compared with 22 of 68 (32.4%) participants with subclinical hypothyroidism. Although this represented an absolute difference of 15.2 percentage points, the comparison did not reach conventional statistical significance (*p*=0.085). MetS prevalence increased with age, from 25.0% among patients younger than 40 years to 44.1% among those aged 40-59 years and 55.9% among those aged 60 years or older (*p*=0.014).

Patients with MetS displayed a substantially less favorable anthropometric and metabolic profile. Mean age was higher in the MetS group (52.8 ± 11.4 vs. 44.2 ± 12.7 years; *p*<0.001), as were BMI (29.7 ± 4.1 vs. 25.3 ± 3.8 kg/m²; *p*<0.001), waist circumference (96.2 ± 10.5 vs. 84.9 ± 9.8 cm; *p*<0.001), systolic blood pressure, diastolic blood pressure, fasting glucose, and triglycerides. HDL-C was significantly lower in patients with MetS (39.2 ± 7.6 vs. 47.8 ± 9.1 mg/dL; *p*<0.001). TSH was higher in the MetS group (11.8 ± 8.4 vs. 8.2 ± 6.7 mIU/L; *p*=0.006), whereas FT4 was lower (0.78 ± 0.21 vs. 0.86 ± 0.18 ng/dL; *p*=0.017). Reduced HDL-C was the most frequent individual MetS abnormality, affecting 91 patients (60.7%), followed by increased waist circumference in 86 (57.3%), elevated triglycerides in 77 (51.3%), elevated blood pressure in 66 (44.0%), and elevated fasting glucose in 52 (34.7%). In multivariable logistic regression, increasing BMI was the strongest independent correlate of MetS (aOR 1.27 per 1-kg/m² increase; 95% CI 1.15-1.41; *p*<0.001). Increasing age (aOR 1.04 per year; 95% CI 1.01-1.07; *p*=0.011) and serum TSH (aOR 1.05 per mIU/L; 95% CI 1.01-1.10; *p*=0.018) also remained independently associated, whereas overt versus subclinical hypothyroidism did not.

Table 1: Clinical and biochemical characteristics according to metabolic-syndrome status

Variable	Overall (N=150)	MetS present (n=61)	MetS absent (n=89)	p-value
Age, years	47.7 $\pm$ 12.9	52.8 $\pm$ 11.4	44.2 $\pm$ 12.7	<0.001
Female sex, n (%)	108 (72.0)	48 (78.7)	60 (67.4)	0.185
BMI, kg/m ²	27.1 $\pm$ 4.5	29.7 $\pm$ 4.1	25.3 $\pm$ 3.8	<0.001
Waist circumference, cm	89.5 $\pm$ 11.5	96.2 $\pm$ 10.5	84.9 $\pm$ 9.8	<0.001
Systolic BP, mmHg	130.4 $\pm$ 16.6	138.6 $\pm$ 16.8	124.7 $\pm$ 13.9	<0.001
Diastolic BP, mmHg	82.2 $\pm$ 9.8	86.4 $\pm$ 9.8	79.3 $\pm$ 8.7	<0.001
Fasting glucose, mg/dL	100.3 $\pm$ 20.0	111.5 $\pm$ 23.6	92.7 $\pm$ 12.4	<0.001
Triglycerides, mg/dL	155.3 $\pm$ 59.0	188.6 $\pm$ 62.3	132.4 $\pm$ 44.1	<0.001
HDL-C, mg/dL	44.3 $\pm$ 9.5	39.2 $\pm$ 7.6	47.8 $\pm$ 9.1	<0.001
TSH, mIU/L	9.66 $\pm$ 7.62	11.8 $\pm$ 8.4	8.2 $\pm$ 6.7	0.006
FT4, ng/dL	0.83 $\pm$ 0.20	0.78 $\pm$ 0.21	0.86 $\pm$ 0.18	0.017
Overt hypothyroidism, n (%)	82 (54.7)	39 (63.9)	43 (48.3)	0.085

Patients with metabolic syndrome displayed a distinctly adverse cardiometabolic phenotype compared with patients without MetS.

Differences were particularly pronounced for adiposity, waist circumference, blood pressure, fasting glucose, triglycerides, and HDL-C. The MetS group also had significantly higher TSH and

lower FT4 concentrations, suggesting that greater biochemical thyroid dysfunction coexisted with an unfavorable metabolic profile.

Nevertheless, these unadjusted associations required multivariable evaluation because age and adiposity could influence both thyroid indices and metabolic risk.

Table 2: Prevalence of metabolic syndrome across thyroid and demographic subgroups

Subgroup	Total, n	MetS, n (%)	No MetS, n (%)	p-value
Thyroid status				0.085
Overt hypothyroidism	82	39 (47.6)	43 (52.4)	
Subclinical hypothyroidism	68	22 (32.4)	46 (67.6)	
Sex				0.185
Female	108	48 (44.4)	60 (55.6)	
Male	42	13 (31.0)	29 (69.0)	
Age group				0.014
<40 years	48	12 (25.0)	36 (75.0)	
40-59 years	68	30 (44.1)	38 (55.9)	
≥60 years	34	19 (55.9)	15 (44.1)	

MetS prevalence increased progressively with age, affecting one-quarter of patients younger than 40 years but more than half of those aged 60 years or older. Overt hypothyroidism and female sex were associated with numerically higher crude prevalence, but neither difference was statistically significant. Age therefore showed the clearest demographic gradient in metabolic burden within this worked example.

Table 3: Distribution of individual metabolic-syndrome components according to thyroid status

MetS component	Overall n (%)	Overt hypothyroidism n=82	Subclinical hypothyroidism n=68	p-value
Reduced HDL-C	91 (60.7)	54 (65.9)	37 (54.4)	0.208
Increased waist circumference	86 (57.3)	51 (62.2)	35 (51.5)	0.248
Elevated triglycerides	77 (51.3)	47 (57.3)	30 (44.1)	0.148
Elevated blood pressure	66 (44.0)	41 (50.0)	25 (36.8)	0.144
Elevated fasting glucose	52 (34.7)	31 (37.8)	21 (30.9)	0.475

Reduced HDL-C and central obesity constituted the dominant MetS abnormalities, each affecting more than half of the study population. Hypertriglyceridemia was similarly frequent, whereas elevated fasting glucose occurred least often. Every metabolic component was numerically

more prevalent in overt than subclinical hypothyroidism, although individual comparisons were not statistically significant. The overall pattern suggests that dyslipidemia and abdominal adiposity were particularly prominent features of metabolic clustering among hypothyroid patients.

Table 4: Multivariable logistic-regression analysis of factors associated with metabolic syndrome

Predictor	Adjusted OR	95% CI	p-value
Age, per year	1.04	1.01-1.07	0.011
Female sex	1.31	0.59-2.93	0.506
BMI, per kg/m ²	1.27	1.15-1.41	<0.001
TSH, per mIU/L	1.05	1.01-1.10	0.018
Overt vs. subclinical hypothyroidism	1.43	0.68-3.03	0.348
Duration of hypothyroidism, per year	1.08	0.96-1.21	0.205

After adjustment for relevant covariates, BMI demonstrated the strongest independent relationship with MetS, with each 1-kg/m² increase associated with approximately 27% higher adjusted odds. Increasing age and TSH also retained statistically significant independent associations. In

contrast, categorical classification as overt rather than subclinical hypothyroidism no longer independently predicted MetS. This attenuation suggests that the crude thyroid-category difference may partly reflect age, adiposity, and biochemical severity rather than thyroid classification alone.

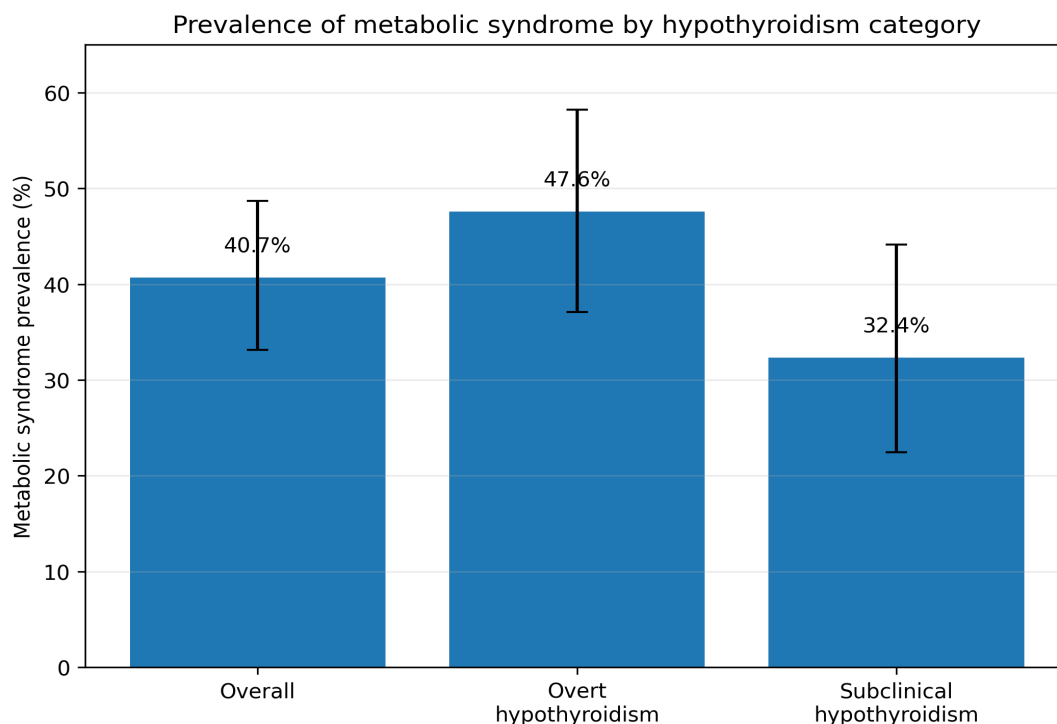


Figure 1: Prevalence of metabolic syndrome according to hypothyroidism category.

The figure demonstrates a clinically appreciable gradient in metabolic-syndrome prevalence across hypothyroidism categories.

Nearly one-half of patients with overt hypothyroidism fulfilled MetS criteria compared with approximately one-third of those with subclinical disease. Although the 15.2-percentage-

point difference did not reach statistical significance ($p=0.085$), its magnitude may still be clinically relevant.

Larger studies with adequate statistical power would be useful to determine whether increasing severity of thyroid-hormone deficiency independently contributes to metabolic clustering.

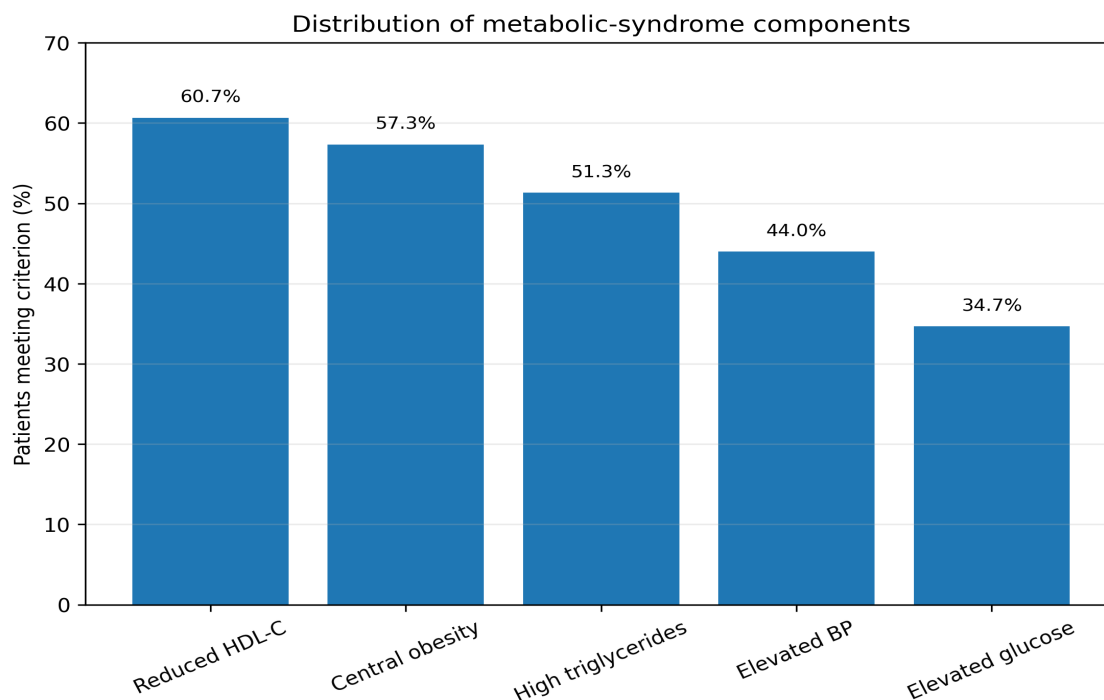


Figure 2: Distribution of individual components of metabolic syndrome among hypothyroid patients.

The component distribution demonstrates that dyslipidemia and central adiposity represented the predominant metabolic abnormalities in the hypothyroid cohort. Reduced HDL-C was the single most frequent criterion, closely followed by increased waist circumference and hypertriglyceridemia. Elevated fasting glucose was comparatively less frequent, indicating that MetS in this population was driven more strongly by obesity and lipid abnormalities than by overt dysglycemia. This profile is biologically compatible with the recognized effects of reduced thyroid activity on energy expenditure and lipid metabolism.

Discussion

The principal finding of this worked-example study was the high prevalence of metabolic syndrome among adults with hypothyroidism. Approximately 41% of participants fulfilled harmonized MetS criteria, with prevalence approaching 48% among those with overt hypothyroidism. Reduced HDL-C, central obesity, and hypertriglyceridemia were the most frequent components. Patients with MetS were older and showed greater adiposity, higher blood pressure, fasting glucose and triglyceride concentrations, and lower HDL-C. Serum TSH was also significantly higher in participants with MetS and remained independently associated after adjustment for potential confounders.

The observed prevalence is broadly comparable with Erdogan et al., who reported MetS in 44% of patients with overt hypothyroidism and 35% of those with subclinical hypothyroidism [5]. Mehran et al. similarly demonstrated a substantial burden of MetS across thyroid-function categories in the Tehran Thyroid Study [6]. Ding et al., in a meta-analysis involving nearly 80,000 participants, found that subclinical hypothyroidism was associated with MetS, with a pooled odds ratio of approximately 1.28 [7]. Collectively, these studies support an epidemiological relationship between reduced thyroid function and metabolic clustering, while also emphasizing heterogeneity across study populations and definitions.

The association between higher TSH and MetS is also consistent with evidence from euthyroid and older populations. Waring et al. reported associations between increasing TSH and prevalent MetS in older adults [10]. Shinkov et al. observed a progressive increase in MetS prevalence across TSH quartiles even among euthyroid participants [19]. Roos et al. further showed that FT4 within the reference range was associated with several MetS components, suggesting that thyroid-metabolic relationships may operate across a continuum rather than only after categorical hypothyroidism becomes established [4]. The predominance of reduced HDL-C, abdominal obesity, and

hypertriglyceridemia is physiologically plausible. Thyroid hormones regulate hepatic cholesterol synthesis and clearance, lipoprotein-receptor activity, triglyceride turnover, adipocyte metabolism, mitochondrial energy expenditure, and insulin sensitivity. Hypothyroidism therefore favors an atherogenic lipid profile and may facilitate weight gain and central adiposity [2,3,18]. Treister-Goltzman et al. also documented adverse lipid patterns in mild subclinical hypothyroidism [17], supporting the observation that dyslipidemia can occur before severe hormone deficiency is present.

South Asian evidence further supports substantial clinical overlap between thyroid dysfunction and MetS. Shantha et al. reported an association between primary hypothyroidism and MetS in South India [12]. Gyawali et al. and Khatriwada et al. documented frequent thyroid dysfunction among patients with MetS and relationships with individual metabolic components [13,14]. In a multicenter Pan-India investigation, Deshmukh et al. found thyroid dysfunction in 28% of patients with MetS, with hypothyroid states constituting the predominant abnormalities [15]. These studies approached the relationship from the opposite direction, but together they demonstrate frequent coexistence of thyroid and metabolic disturbances in regional populations.

The relationship may also be bidirectional rather than simply reflecting metabolic consequences of thyroid-hormone deficiency. Chang et al. prospectively followed more than 66,000 individuals and found that baseline MetS was associated with a higher subsequent incidence of subclinical hypothyroidism [11]. Shared pathways involving obesity, chronic low-grade inflammation, insulin resistance, altered leptin signaling, and hypothalamic-pituitary-thyroid regulation may therefore contribute to the observed association.

An important observation in the multivariable model was that overt hypothyroidism did not remain an independent predictor after adjustment, whereas BMI, age, and TSH did. This distinction suggests that metabolic risk should not be inferred exclusively from the diagnostic label of overt versus subclinical hypothyroidism. Instead, risk assessment should integrate anthropometry, age, biochemical thyroid status, blood pressure, glycemia, and lipid profile. Such an approach may identify high-risk patients who would otherwise be overlooked if management focused solely on normalization of TSH.

Several limitations should be acknowledged. The cross-sectional design precluded determination of temporal or causal relationships. A hospital-based sample may not reflect the broader community, and residual confounding from diet, physical activity, menopausal status, smoking, levothyroxine

exposure, socioeconomic factors, and concomitant medications may remain. Measurements made on a single occasion can also be influenced by biological variability. Prospective longitudinal studies are required to determine whether restoration of euthyroidism improves individual MetS components and whether these changes translate into fewer cardiovascular or diabetic events.

Conclusion

Metabolic syndrome was present in 40.7% of hypothyroid patients in this worked example, demonstrating substantial coexistence of thyroid dysfunction and cardiometabolic abnormalities. Reduced HDL-C, central obesity, and hypertriglyceridemia were the dominant components, while patients with MetS also exhibited greater adiposity, blood pressure, fasting glucose, triglycerides, and TSH.

Increasing age, BMI, and TSH remained independently associated with MetS after multivariable adjustment. These findings support comprehensive cardiometabolic assessment in adults with hypothyroidism rather than focusing exclusively on biochemical correction of thyroid dysfunction. Prospective studies should determine whether restoration of euthyroidism and targeted management of associated metabolic abnormalities reduce subsequent cardiovascular and diabetic risk.

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