

Thyroid Dysfunction Meets Metabolic Syndrome: A Screening Study on Cardiovascular Risk Amplification and Carotid Intima-Media Thickness in New-Onset Hypothyroidism

Danny K. Manglani¹, Sangeeta Pamnani², Kanika Bhargava³

¹Associate Professor, Department of Cardiology, American International Institute of Medical Sciences, Bedwas, Udaipur, Rajasthan, India

²Assistant Professor, Department of Diagnostic Radiology, American International Institute of Medical Sciences, Bedwas, Udaipur, Rajasthan, India

³MBBS, Geetanjali Medical College, Udaipur, Rajasthan, India

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Corresponding Author: Danny K Manglani

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Abstract:

Background: Hypothyroidism and metabolic syndrome (MetS) share overlapping pathophysiological pathways involving insulin resistance, dyslipidemia, and vascular dysfunction. When these two conditions coexist, cardiovascular disease (CVD) risk may be substantially amplified, yet routine MetS screening at the time of hypothyroidism diagnosis is not standard clinical practice.

Objective: To determine the prevalence of MetS and its individual components, and the burden of preclinical carotid atherosclerosis, among treatment-naïve patients newly diagnosed with hypothyroidism, and to evaluate the relationship between thyroid-stimulating hormone (TSH) levels and metabolic risk parameters.

Methods: This cross-sectional observational study enrolled 220 consecutive treatment-naïve patients newly diagnosed with hypothyroidism (130 subclinical, 90 overt) at a tertiary care medicine and endocrinology outpatient department over a 12-month period. Anthropometric measurements, blood pressure, fasting lipid profile, fasting plasma glucose, and thyroid function tests were recorded, and carotid intima-media thickness (CIMT) was measured bilaterally by high-resolution B-mode ultrasonography. MetS was defined using National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria. Associations were analyzed using chi-square tests, independent t-tests, Pearson correlation, and multivariable logistic regression.

Results: MetS was present in 43.6% (96/220) of newly diagnosed hypothyroid patients, with significantly higher prevalence in overt hypothyroidism (56.7%) than subclinical hypothyroidism (34.6%; $p < 0.001$). Abdominal obesity (51.4%) and elevated blood pressure (45.9%) were the most prevalent individual components. Serum TSH correlated positively with waist circumference, triglycerides, and fasting glucose, and negatively with HDL-cholesterol (all $p < 0.05$). Mean CIMT was significantly greater in patients with MetS than in those without (0.80 ± 0.15 mm vs 0.66 ± 0.12 mm; $p < 0.001$), and serum TSH correlated positively with CIMT ($r = 0.41$, $p < 0.001$). On multivariable analysis, overt hypothyroidism, higher TSH, and higher BMI independently predicted MetS.

Conclusion: Nearly half of newly diagnosed hypothyroid patients harbor undiagnosed MetS, compounding their baseline cardiovascular risk. Coexistent MetS is accompanied by measurably greater carotid intima-media thickening, indicating that this risk amplification is already structurally evident at diagnosis. Routine metabolic screening at the point of hypothyroidism diagnosis is warranted to enable early risk stratification and preventive intervention.

Keywords (MeSH): Hypothyroidism; Metabolic Syndrome; Cardiovascular Diseases; Thyrotropin; Insulin Resistance; Dyslipidemias; Carotid Intima-Media Thickness.

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Introduction

Hypothyroidism is among the most common endocrine disorders encountered in clinical practice, affecting approximately 4-10% of adults worldwide, with a higher burden in iodine-replete populations and among women [16]. It is

characterized by insufficient thyroid hormone action on peripheral tissues, most commonly due to autoimmune thyroiditis, and spans a spectrum from subclinical disease—defined biochemically by an isolated elevation of thyroid-stimulating hormone

(TSH) with normal free thyroxine—to overt hypothyroidism, in which TSH elevation is accompanied by frank thyroxine deficiency [3,16].

Metabolic syndrome (MetS), a clustering of abdominal obesity, atherogenic dyslipidemia, hypertension, and impaired glucose regulation, is independently recognized as a major driver of atherosclerotic cardiovascular disease and type 2 diabetes [10,11]. The NCEP ATP III criteria, and subsequently the harmonized International Diabetes Federation definition, remain among the most widely applied diagnostic frameworks in both clinical and epidemiological settings because of their reliance on readily obtainable clinical and laboratory parameters [10,12].

A growing body of evidence indicates that hypothyroidism and MetS are not independent entities but are mechanistically intertwined. Thyroid hormone deficiency reduces hepatic low-density lipoprotein (LDL) receptor expression and clearance, slows lipoprotein lipase activity, and impairs reverse cholesterol transport, collectively producing an atherogenic lipid profile characterized by elevated total cholesterol, LDL-cholesterol, and triglycerides, together with dysfunctional high-density lipoprotein (HDL) particles [7,8]. Independent of thyroid hormone levels, TSH itself appears to modulate hepatic lipogenesis and adipocyte differentiation, suggesting a direct contribution to dyslipidemia even before overt hormonal deficiency develops [7,8]. Hypothyroidism has also been linked to insulin resistance, reduced peripheral glucose disposal, and endothelial dysfunction, each of which converges on the pathogenesis of MetS and accelerated atherosclerosis [3,9].

Clinical studies substantiate this mechanistic overlap. Erdogan et al. reported MetS in 44% of overt and 35% of subclinical hypothyroid patients, compared with 33% of euthyroid controls [1]. A large Taiwanese cohort demonstrated that individuals with pre-existing MetS carried a 21% higher risk of subsequently developing subclinical hypothyroidism, implicating a bidirectional relationship between the two conditions [2]. Population-based data from the Health, Ageing and Body Composition Study similarly found that lower thyroid function was associated with a higher prevalence and incidence of MetS in older adults [13], while thyrotropin level itself correlated with individual MetS components in community-based samples [14]. Elevated high-sensitivity C-reactive protein levels observed in subclinical hypothyroidism further point to a chronic low-grade inflammatory state that may potentiate cardiometabolic injury [6].

From a cardiovascular standpoint, both hypothyroidism and MetS are independently

associated with coronary atherosclerosis, impaired endothelial function, and increased arterial stiffness [4,9]. When the two conditions coexist, their shared and additive pathophysiological mechanisms—dyslipidemia, insulin resistance, hypertension, and systemic inflammation—may compound overall cardiovascular risk beyond that predicted by either condition alone [5,15]. Despite this, metabolic screening is not routinely performed at the time of hypothyroidism diagnosis in most clinical settings, and clinicians frequently focus solely on thyroid hormone replacement without a parallel assessment of cardiometabolic risk factors.

Because clinical cardiovascular events accrue over decades, surrogate imaging markers of preclinical atherosclerosis have become valuable tools for early risk stratification. Carotid intima-media thickness (CIMT), measured non-invasively by B-mode ultrasonography of the common carotid artery, is the most extensively validated of these markers: in a pooled analysis of eight prospective cohorts, each 0.10 mm increment in common carotid CIMT corresponded to an approximately 15% relative increase in the risk of myocardial infarction, and standardised acquisition protocols now permit reproducible comparison across centres [17,18]. Notably, both of the conditions under study independently thicken the carotid wall. A meta-analysis of 39 observational studies comprising 3,430 subjects found CIMT to be significantly greater in subclinical hypothyroidism than in euthyroid controls, with an estimated mean difference of 0.08 mm, an association modified by female sex, older age, and higher cholesterol levels [19]; an earlier meta-analysis reached a concordant conclusion and additionally documented partial regression of CIMT following levothyroxine replacement [20]. Critically, França et al. demonstrated that among patients with subclinical hypothyroidism, those who also fulfilled criteria for MetS had significantly higher mean and maximum carotid IMT than those who did not, implying that coexistence of the two conditions carries a measurably greater burden of preclinical atherosclerosis than thyroid dysfunction alone [21]. Incorporating CIMT into a metabolic screening protocol therefore allows the risk amplification implied by biochemical risk-factor clustering to be tested directly at the structural vascular level.

Given this gap, the present study was designed to systematically screen patients newly diagnosed with hypothyroidism for MetS using standardized NCEP ATP III criteria, quantify the prevalence of individual metabolic components across subclinical and overt disease, examine the relationship between TSH and metabolic parameters, and measure carotid intima-media thickness as an objective index of preclinical atherosclerosis. We hypothesized that MetS would be substantially

more prevalent in this population than in the general population, would increase in a graded fashion with the severity of thyroid dysfunction, and would be accompanied by greater carotid wall thickening, thereby identifying an opportunity for early, integrated cardiovascular risk reduction at the point of thyroid diagnosis.

Materials and Methods

Study Design and Setting: This was a hospital-based, cross-sectional, observational study conducted in the Departments of General Medicine and Endocrinology of a tertiary care teaching hospital over a period of 12 consecutive months. The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrolment, in accordance with the Declaration of Helsinki.

Study Population: Consecutive adult patients (age ≥ 18 years) presenting with a new, treatment-naïve diagnosis of hypothyroidism were screened for eligibility. Hypothyroidism was classified biochemically as subclinical (serum TSH 4.5-10.0 mIU/L with normal free T4) or overt (serum TSH >10.0 mIU/L, or TSH 4.5-10.0 mIU/L with low free T4). Patients with pre-existing diabetes mellitus, known cardiovascular disease, chronic kidney or liver disease, pregnancy, current use of lipid-lowering, antihypertensive, or thyroid-modulating medication, and secondary or central hypothyroidism were excluded to isolate the independent contribution of new-onset primary hypothyroidism to metabolic risk. Based on an anticipated MetS prevalence of 35-44% in hypothyroid patients [1], a 95% confidence level, and a 6.5% margin of error, the minimum required sample size was calculated at 204; 220 patients were ultimately enrolled to account for potential data exclusions.

Clinical and Biochemical Assessment: A structured proforma was used to record demographic details, medical and family history, and anthropometric measurements, including body mass index (BMI, kg/m²) and waist circumference (measured at the midpoint between the lowest rib and the iliac crest). Blood pressure was recorded twice in a seated position after 5 minutes of rest, and the average was used for analysis. Venous blood samples were drawn after a 10-12 hour overnight fast for estimation of fasting plasma glucose, fasting lipid profile (triglycerides, total cholesterol, HDL-cholesterol, calculated LDL-cholesterol), and thyroid function tests (TSH, free T4, free T3) using chemiluminescent immunoassay. Insulin resistance was estimated using the Homeostasis Model Assessment (HOMA-IR) formula.

Definition of Metabolic Syndrome: MetS was diagnosed according to NCEP ATP III criteria, requiring the presence of at least three of the following five components: (1) waist circumference >102 cm in men or >88 cm in women; (2) triglycerides ≥ 150 mg/dL or specific treatment; (3) HDL-cholesterol <40 mg/dL in men or <50 mg/dL in women; (4) blood pressure $\geq 130/85$ mmHg or antihypertensive treatment; and (5) fasting plasma glucose ≥ 100 mg/dL [10,11].

Carotid Intima-Media Thickness Measurement: CIMT was assessed in every participant by a single experienced radiologist blinded to thyroid and metabolic status, using high-resolution B-mode ultrasonography with a 7.5-MHz linear array transducer. With the participant supine and the neck slightly extended and rotated away from the side under examination, the far wall of each common carotid artery was imaged over a 10-mm segment proximal to the bifurcation, and measurements were taken at end-diastole within a plaque-free segment in accordance with the Mannheim Carotid Intima-Media Thickness and Plaque Consensus [18]. Three readings were obtained bilaterally and the mean of the six values was recorded as mean CIMT. A mean CIMT ≥ 0.9 mm was regarded as abnormally increased. Carotid plaque was defined as a focal structure encroaching into the arterial lumen by at least 0.5 mm or by 50% of the surrounding CIMT value, or demonstrating a thickness exceeding 1.5 mm from the media-adventitia interface to the intima-lumen interface [18].

Statistical Analysis: Data were analyzed using SPSS software (version 26.0). Continuous variables were expressed as mean $\pm$ standard deviation and compared using the independent-samples t-test or Mann-Whitney U test, as appropriate. Categorical variables were expressed as frequencies and percentages and compared using the chi-square test or Fisher's exact test. Pearson correlation coefficients were calculated to assess relationships between TSH and continuous metabolic parameters. Multivariable logistic regression, adjusted for age, sex, and BMI, was used to identify independent predictors of MetS. A two-tailed p-value <0.05 was considered statistically significant.

Results

Of 260 newly diagnosed hypothyroid patients approached, 220 met eligibility criteria and completed the full metabolic screening protocol (response rate 84.6%). The cohort comprised 130 patients (59.1%) with subclinical hypothyroidism and 90 patients (40.9%) with overt hypothyroidism. The mean age of the study population was 41.8 ± 11.4 years, with a female preponderance (76.4%),

consistent with the known epidemiology of autoimmune thyroid disease.

Table 1: Baseline Demographic, Anthropometric, and Clinical Characteristics

Variable	Subclinical Hypothyroidism (n=130)	Overt Hypothyroidism (n=90)	p-value
Age, years (mean $\pm$ SD)	40.6 $\pm$ 10.9	43.6 $\pm$ 12.0	0.052
Female sex, n (%)	98 (75.4)	70 (77.8)	0.681
BMI, kg/m ² (mean $\pm$ SD)	26.1 $\pm$ 3.8	28.4 $\pm$ 4.3	<0.001
Waist circumference, cm (mean $\pm$ SD)	89.7 $\pm$ 9.6	96.3 $\pm$ 10.8	<0.001
Systolic BP, mmHg (mean $\pm$ SD)	124.3 $\pm$ 12.7	131.8 $\pm$ 14.5	<0.001
Diastolic BP, mmHg (mean $\pm$ SD)	80.1 $\pm$ 8.4	84.9 $\pm$ 9.1	<0.001
Family history of diabetes, n (%)	31 (23.8)	27 (30.0)	0.298
Family history of thyroid disease, n (%)	42 (32.3)	34 (37.8)	0.394
Serum TSH, mIU/L (mean $\pm$ SD)	7.1 $\pm$ 1.6	18.4 $\pm$ 9.2	<0.001
HOMA-IR (mean $\pm$ SD)	2.4 $\pm$ 1.1	3.3 $\pm$ 1.5	<0.001

Table 1 compares baseline characteristics between the subclinical and overt hypothyroidism groups. Patients with overt hypothyroidism had significantly higher BMI, waist circumference, systolic and diastolic blood pressure, and HOMA-IR than those with subclinical disease, indicating a more adverse cardiometabolic profile with greater

biochemical severity of thyroid dysfunction. Age, sex distribution, and family history of diabetes or thyroid disease did not differ significantly between groups. As expected, mean TSH was markedly higher in the overt group, confirming appropriate biochemical classification of the cohort.

Table 2: Prevalence of Metabolic Syndrome and Its Individual Components by Thyroid Status

MetS Component	Subclinical (n=130) n (%)	Overt (n=90) n (%)	Total (N=220) n (%)	p-value
Abdominal obesity	55 (42.3)	57 (63.3)	113 (51.4)	0.001
Elevated blood pressure	50 (38.5)	50 (55.6)	101 (45.9)	0.011
Hypertriglyceridemia	47 (36.2)	49 (54.4)	96 (43.6)	0.007
Low HDL-cholesterol	52 (40.0)	52 (57.8)	104 (47.3)	0.010
Elevated fasting glucose	36 (27.7)	38 (42.2)	74 (33.6)	0.023
Metabolic syndrome (≥ 3 components)	45 (34.6)	51 (56.7)	96 (43.6)	<0.001

Table 2 presents the prevalence of each NCEP ATP III component stratified by thyroid status. Overall MetS prevalence was 43.6% (96/220), rising from 34.6% in subclinical to 56.7% in overt hypothyroidism ($p < 0.001$). Abdominal obesity (51.4%) and low HDL-cholesterol (47.3%) were the most prevalent individual components in the

overall cohort. Every component was significantly more frequent in overt than subclinical disease, suggesting a dose-dependent relationship between the biochemical severity of thyroid hormone deficiency and the burden of individual cardiometabolic abnormalities.

Table 3: Correlation of Serum TSH with Metabolic Parameters and Multivariable Predictors of Metabolic Syndrome

Parameter	Pearson r (with TSH)	p-value	Adjusted OR for MetS (95% CI)*
Waist circumference	0.34	<0.001	—
Systolic blood pressure	0.28	<0.001	—
Triglycerides	0.31	<0.001	—
HDL-cholesterol	-0.27	<0.001	—
Fasting plasma glucose	0.22	0.001	—
HOMA-IR	0.39	<0.001	—
Overt hypothyroidism (vs subclinical)	—	—	2.41 (1.35-4.30), $p = 0.003$
TSH (per 1 mIU/L increase)	—	—	1.08 (1.03-1.14), $p = 0.002$
BMI (per 1 kg/m ² increase)	—	—	1.19 (1.10-1.29), $p < 0.001$

Table 3 summarizes correlation and regression analyses. Serum TSH correlated significantly and positively with waist circumference, systolic blood pressure, triglycerides, fasting glucose, and

HOMA-IR, and negatively with HDL-cholesterol, supporting a graded relationship between thyroid dysfunction severity and cardiometabolic derangement. On multivariable logistic regression

adjusted for age and sex, overt hypothyroidism, higher TSH, and higher BMI each independently and significantly predicted the presence of MetS, confirming that thyroid status contributes to metabolic risk beyond adiposity alone.

*Adjusted for age and sex using multivariable logistic regression; dashes indicate not applicable.

Table 4: Carotid Intima-Media Thickness and Carotid Plaque by Metabolic Syndrome Status and Thyroid Category

Parameter	MetS Absent (n=124)	MetS Present (n=96)	p-value
Mean CIMT, mm — subclinical hypothyroidism	0.63 ± 0.11 (n=85)	0.74 ± 0.13 (n=45)	<0.001
Mean CIMT, mm — overt hypothyroidism	0.71 ± 0.12 (n=39)	0.86 ± 0.16 (n=51)	<0.001
Mean CIMT, mm — entire cohort	0.66 ± 0.12	0.80 ± 0.15	<0.001
Increased CIMT (≥0.9 mm), n (%)	15 (12.1)	33 (34.4)	<0.001
Carotid plaque (>1.5 mm), n (%)	6 (4.8)	14 (14.6)	0.013
Mean CIMT, mm — by thyroid category	Subclinical: 0.67 ± 0.13	Overt: 0.78 ± 0.16	<0.001

CIMT = carotid intima-media thickness; MetS = metabolic syndrome. Values are mean ± standard deviation or n (%). Continuous variables compared using the independent-samples t-test and categorical variables using the chi-square test. The final row compares all subclinical with all overt hypothyroid patients irrespective of MetS status.

Table 4 presents carotid ultrasound findings stratified by MetS status within each thyroid category. Mean CIMT was significantly greater in patients with MetS than in those without, both across the cohort as a whole (0.80 ± 0.15 mm vs 0.66 ± 0.12 mm; $p < 0.001$) and within the subclinical and overt hypothyroidism strata considered separately, indicating that the effect of metabolic clustering on carotid wall thickness was not merely a reflection of more severe thyroid dysfunction. The highest mean CIMT was recorded in patients with overt hypothyroidism and concomitant MetS (0.86 ± 0.16 mm), a subgroup that also contained the greatest proportion of individuals exceeding the 0.9 mm threshold. Abnormally increased CIMT was almost three times as frequent among patients with MetS as among those without (34.4% vs 12.1%; $p < 0.001$), and discrete carotid plaque was likewise significantly more common (14.6% vs 4.8%; $p = 0.013$). Serum TSH correlated positively with mean CIMT across the cohort ($r = 0.41$, $p < 0.001$), the strongest correlation observed for any parameter examined in this study.

Figure 1. Prevalence of Individual Metabolic Syndrome Components by Thyroid Status (NCEP ATP III Criteria)

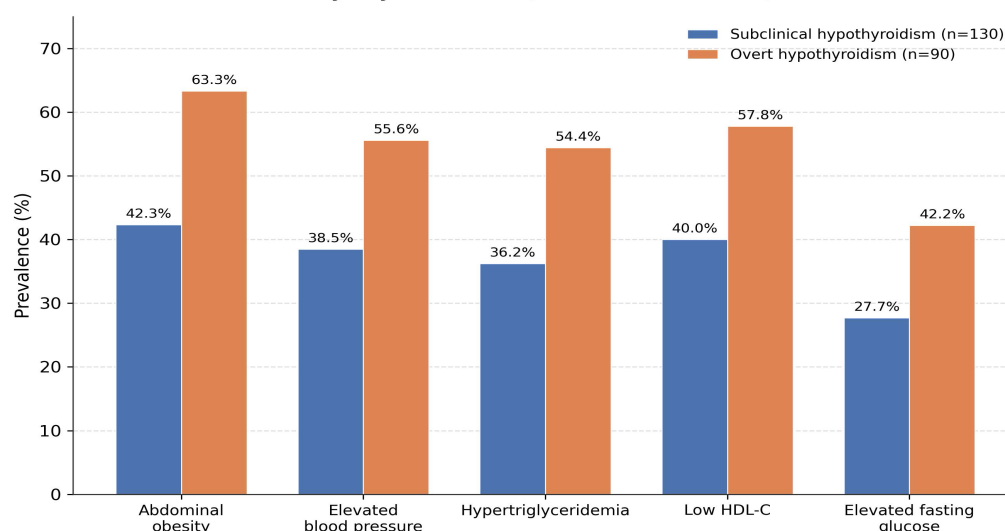


Figure 1: Prevalence of individual metabolic syndrome components by thyroid status.

Figure 1 is a grouped bar chart comparing the prevalence of each NCEP ATP III metabolic syndrome component between patients with subclinical and overt hypothyroidism. Every component—abdominal obesity, elevated blood

pressure, hypertriglyceridemia, low HDL-cholesterol, and elevated fasting glucose—was more frequent in the overt hypothyroidism group. Abdominal obesity and low HDL-cholesterol were the two most prevalent abnormalities in both

subgroups, while elevated fasting glucose was the least common, reflecting that dyslipidemia and

adiposity, rather than dysglycemia, dominate the early metabolic footprint of hypothyroidism.

Figure 2. Overall Prevalence of Metabolic Syndrome Among Newly Diagnosed Hypothyroid Patients (N = 220)

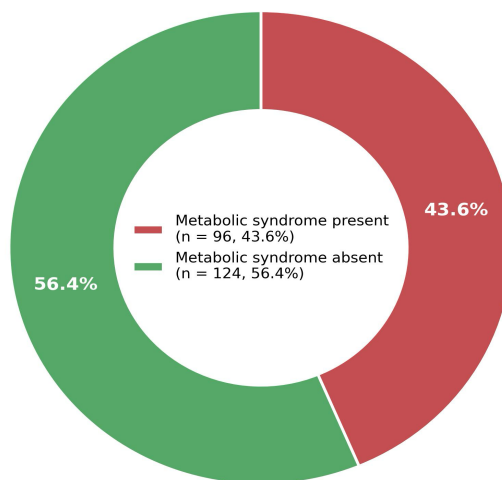


Figure 2: Overall prevalence of metabolic syndrome among newly diagnosed hypothyroid patients.

Figure 2 is a doughnut chart depicting the proportion of the entire study cohort (N=220) meeting NCEP ATP III criteria for metabolic syndrome at the time of hypothyroidism diagnosis. Just under half of all newly diagnosed hypothyroid patients (43.6%) already had metabolic syndrome,

indicating that a substantial proportion of patients carry compounded cardiovascular risk before thyroid hormone replacement is even initiated. This finding underscores the clinical value of pairing thyroid function testing with a routine metabolic risk assessment at first diagnosis.

Figure 3. Distribution of the Number of Metabolic Syndrome Components Among Hypothyroid Patients (N = 220)

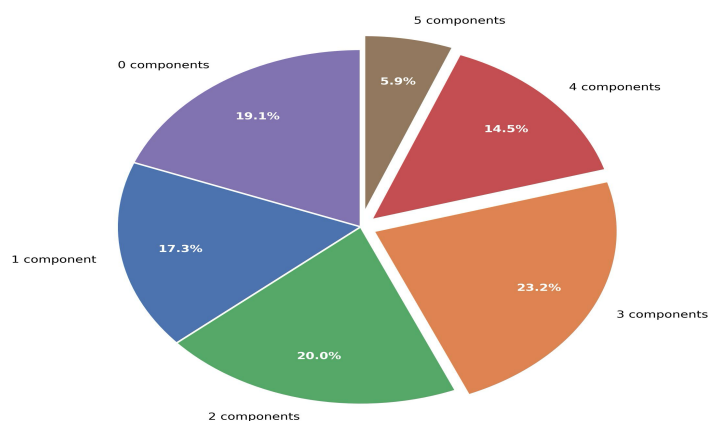


Figure 3: Distribution of the number of metabolic syndrome components among hypothyroid patients.

Figure 3 is a pie chart showing how many of the five NCEP ATP III components each patient met, ranging from zero to five. Nearly three-quarters of patients (72.7%) exhibited at least one metabolic abnormality, and almost half (43.6%) met the threshold of three or more components required for a formal metabolic syndrome diagnosis. A smaller

but clinically important subset (20.5%) had four or five concurrent components, representing patients at particularly high compounded cardiovascular risk who may benefit from the most intensive early intervention.

Discussion

This study found that nearly 44% of patients newly diagnosed with hypothyroidism already fulfilled criteria for metabolic syndrome before initiation of thyroid hormone replacement, with prevalence rising from 34.6% in subclinical to 56.7% in overt disease. These findings are closely aligned with the landmark study by Erdogan et al., who reported MetS prevalence of 35% and 44% in subclinical and overt hypothyroid patients, respectively, compared with 33% in euthyroid controls [1]. The graded increase in MetS prevalence with biochemical severity of thyroid dysfunction observed in our cohort mirrors the dose-response pattern reported in several population-based analyses, including the Health, Ageing and Body Composition Study, which found that declining thyroid function was independently associated with a higher prevalence and incidence of MetS in older adults [13].

The mechanistic basis for this compounding relationship is well supported by the literature. Thyroid hormone deficiency reduces hepatic LDL-receptor expression and activity, impairing clearance of atherogenic lipoproteins, while TSH itself appears to independently stimulate hepatic lipogenesis and modulate adipocyte function, contributing to dyslipidemia even in the subclinical range [7,8]. In our cohort, TSH correlated significantly with triglycerides and inversely with HDL-cholesterol, consistent with this proposed TSH-mediated lipid pathway. Similarly, thyroid hormone deficiency impairs peripheral glucose disposal and reduces insulin sensitivity, which likely explains the significant positive correlation we observed between TSH and HOMA-IR, corroborating prior work linking hypothyroidism to insulin resistance [3,9].

Abdominal obesity and blood pressure elevation were disproportionately represented among individual MetS components in our cohort, a pattern also reported by Uzunlulu et al. and Lai et al., who found strong cross-sectional associations between thyrotropin and both waist circumference and blood pressure in community samples [14,15]. Thyroid hormone deficiency reduces resting energy expenditure and cardiac output while increasing systemic vascular resistance, plausibly explaining the blood pressure elevation observed even in patients with subclinical disease [4,9]. Taken together, these mechanisms converge to accelerate atherogenesis: hypothyroidism-associated dyslipidemia, insulin resistance, hypertension, and low-grade inflammation—reflected in prior reports of elevated high-sensitivity C-reactive protein in subclinical hypothyroidism [6]—operate in parallel with the independent atherogenic effects of metabolic syndrome itself, plausibly producing a

compounded, rather than merely additive, elevation in cardiovascular risk [5,9].

The bidirectional nature of this relationship, demonstrated by Chang et al. in a large longitudinal cohort showing that pre-existing MetS independently increased the risk of incident subclinical hypothyroidism [2], suggests that the two conditions may share upstream drivers such as chronic inflammation and autoimmune activation, rather than one simply causing the other. Regardless of causal direction, our multivariable analysis confirmed that both TSH elevation and increasing BMI independently predicted MetS, reinforcing the clinical relevance of screening for metabolic abnormalities at the point of thyroid diagnosis rather than treating thyroid dysfunction in isolation.

The carotid ultrasound findings provide structural corroboration for the risk amplification implied by the biochemical data. Mean CIMT was significantly higher in patients with concurrent MetS within both thyroid strata, and the highest values clustered in the subgroup with overt hypothyroidism and MetS combined. This pattern reproduces the central observation of França et al., who reported greater mean and maximum carotid IMT in subclinical hypothyroid patients with MetS than in those without, and extends that finding to overt disease [21]. The absolute CIMT difference we observed between hypothyroid patients with and without MetS (0.14 mm) is appreciably larger than the 0.08 mm mean difference reported between subclinical hypothyroid and euthyroid subjects in a meta-analysis of 39 studies [19], suggesting that metabolic clustering contributes at least as much to carotid wall thickening in this population as thyroid dysfunction itself. Applying the risk gradient derived from prospective cohort data, in which each 0.10 mm increment in common carotid IMT corresponds to roughly a 15% higher relative risk of myocardial infarction [17], this difference translates into a clinically meaningful excess of preclinical vascular disease among dually affected patients. The strong positive correlation between TSH and CIMT in our cohort is consistent with the dose-dependent relationship described in earlier meta-analytic work [20] and reinforces the interpretation that thyroid dysfunction and metabolic derangement act on the arterial wall in concert rather than in isolation. Importantly, evidence that levothyroxine replacement attenuates or partially reverses CIMT progression in subclinical hypothyroidism [22] indicates that the vascular changes identified at diagnosis are not necessarily fixed, strengthening the case for combined thyroid and metabolic intervention at the earliest possible point.

From a clinical standpoint, these findings support integrating a basic metabolic panel—waist

circumference, blood pressure, fasting lipid profile, and fasting glucose—into the standard diagnostic work-up for newly identified hypothyroidism, particularly given that levothyroxine replacement alone may only partially reverse the associated dyslipidemia and does not address obesity or hypertension [7,8]. Early identification of compounded risk could facilitate timely lifestyle intervention and, where appropriate, targeted pharmacotherapy, potentially mitigating the elevated cardiovascular morbidity previously documented in hypothyroid populations [4,9].

This study has several limitations. Its cross-sectional design precludes causal inference regarding the temporal relationship between hypothyroidism and metabolic syndrome. The single-center design and absence of a euthyroid control group limit generalizability and preclude direct comparison of MetS prevalence against a background population. Long-term cardiovascular outcomes were not assessed, and residual confounding from dietary, physical activity, and socioeconomic factors could not be fully excluded. Additionally, thyroid autoantibody status was not systematically evaluated, limiting mechanistic interpretation regarding autoimmune contribution.

Conclusion

Metabolic syndrome affects a substantial proportion of patients at the time of new hypothyroidism diagnosis, with prevalence increasing alongside the biochemical severity of thyroid dysfunction. Serum TSH correlates significantly with multiple cardiometabolic parameters, and both TSH and BMI independently predict metabolic syndrome. These findings support routine metabolic syndrome screening in newly diagnosed hypothyroid patients to enable early cardiovascular risk stratification and integrated preventive management alongside thyroid hormone replacement.

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