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**Original Research Article**

# Screening for metabolic syndrome in patients newly diagnosed with hypothyroidism to identify the compounding effect on cardiovascular disease risk

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## ABSTRACT

**Background:** Lowered thyroid hormones and elevated thyroid stimulating hormone (TSH) levels of hypothyroid state are found to be associated with metabolic changes encompassing components of metabolic syndrome (MetS). Both hypothyroidism and MetS are individually associated with risk of cardiovascular disease (CVD). The presence of MetS can have a compounding effect on the CVD risk in hypothyroidism.

**Methods:** A case-control cross sectional study was done in 30 newly diagnosed hypothyroid cases and 30 healthy controls. Waist circumference (WC), height, weight and blood pressure (BP) were recorded; fasting venous blood sample was collected for thyroid function tests, glucose, lipids and insulin. Insulin resistance as homeostasis model assessment for insulin resistance (HOMA-IR) and body mass index (BMI) was calculated. Comparison of means and correlation analysis was done using SPSS version 22.

**Results:** Higher BMI (p=0.001), WC (p<0.001), BP (systolic p=0.039; diastolic p<0.001), total cholesterol (TC) (p=0.001), triglycerides (TGL) (p<0.001), very low-density lipoproteins (p<0.001) and fasting plasma glucose (FPG) levels (p<0.001) were observed in hypothyroid subjects. MetS was found in 11 of the 22 females and three of the eight male subjects with hypothyroidism. Presence of individual components of MetS in hypothyroidism was WC (90%), TGLs (83.3%) and high density lipoprotein (HDL) (73.3%). All female subjects in both groups had higher WC. Among female controls, two had MetS. Hypothyroidism was found to associated with components of MetS.

**Conclusions:** Hypothyroidism associated metabolic changes can lead to the development of MetS. Targeted therapeutic interventions and life style modifications can attenuate compounding effect on of MetS on CVD risk in hypothyroidism.

**Keywords:** Newly diagnosed hypothyroidism, Components of metabolic syndrome, Insulin resistance, Anthropometric indices, Screening, Cardiovascular disease risk

## INTRODUCTION

Thyroid hormones play important roles in regulating body metabolism, development and differentiation of cells. Thyroid disorders are one of the most common endocrine disorders with overall prevalence of hypothyroidism in India reported at 10.95% in general population.[1] Hypothyroidism is known to be associated with several traditional risk factors for atherosclerosis.[2] MetS by itself is a group of multiple disorders encompassing central obesity, impaired fasting glucose, elevated TGL, reduced HDL levels and hypertension.[3] In Indian population, MetS has an overall prevalence of 31.6% in the general population, with a prevalence of 22.9% in males and 39.9% in females.[4] Presence of type 2 diabetes mellitus (T2DM) is commonly observed in MetS, and if not present, subjects with MetS are five times more likely to develop T2DM.[4] Increasing TSH levels is reported to be associated with increase in the number of components of MetS.[5] Indian studies have reported prevalence of MetS in hypothyroidism to be at 53.24%, with a prevalence of 54.9% in males and 95.6% in females.[6] Vice versa, it has also been suggested that in newly diagnosed MetS the presence of hypothyroidism should be checked.[7,8] Metabolic changes encountered in hypothyroidism possibly contribute to development of MetS. As both hypothyroidism and MetS individually are associated with risk of development of CVDs, the presence of MetS in subjects with hypothyroidism compounds the CVD risk.

## METHODS

A case-control observational study was undertaken in subjects attending the outpatient department of Endocrinology and metabolism of Sri Venkateswara Institute of Medical Sciences, Tirupati from March 2021 to August 2022. Sample size was calculated using the formula n=2/d$^2$×Cp power, where power value at 80% power and 0.05 alpha error was considered. The sample size obtained was 60. A total of 133 subjects were screened and subjects on treatment for DM (n=45), thyroid disorder (n=26) and hypertension (n=32) were excluded. Thirty subjects who were naïve to treatment newly diagnosed with hypothyroidism with TSH levels greater than or equal to 15 mIU/l were included into the study as cases (Group 2).[9] Thirty healthy subjects without hypothyroidism, from among hospital staff and subjects accompanying patients, were included into the study as controls (Group 1). Subjects with CVD, liver and kidney disease, pregnant women and individuals not willing to participate in the study were excluded.

### *Anthropometric measurements*

Subjects were made to stand bare foot and straight against a wall for measurement of height at maximum inspiration. A headboard and a measuring tape were used to measure the height. An electronic scale was used to measure the weight. WC was measured midway between the upper margin of iliac crest and lower margin of the last ribs at the end of expiration using a measuring tape.

BP was recorded with the subject seated comfortably with the elbow slightly flexed, supporting the forearm at heart level on the table and by applying the sphygmomanometer cuff above the elbow.

### *Sample collection*

Five ml of peripheral venous blood from the median cubital vein was collected using aspectic precautions from subjects who were fasting for 8-10 hours. Three ml of blood was transferred into additive free vacutainers, which were allowed to stand for 30 minutes, centrifuged at 2000 rpm for 15 minutes and serum obtained was stored in labelled vials at -80ºC until biochemical analysis. Two ml of blood was transferred to sodium fluoride and potassium oxalate anticoagulant containing vacutainers, centrifuged immediately and plasma separated.

### *Biochemical analysis*

Serum sample was analyzed for TSH, thyroxine (T4), insulin by chemiluminescence immunoassay using Beckman system packs on Access, Beckman Coulter (California, USA). TC by cholesterol oxidase peroxidase method, TGLs by enzymatic colorimetric methodand HDL by selective inhibition methodusing commercially available kits (Agappe diagnostics Ltd Ernakulum, Kerala), FPG by glucose oxidase peroxidase method using commercially available kit (Pathozyme diagnostics, Maharashtra, India) on AU 480, Beckman coulter, (California, USA). Calculated parameters were LDL, VLDL, BMI, insulin resistance as HOMA-IR.[10-12]

MetS observed in subjects was as per national cholesterol education program's adult treatment panel III report (NCEP ATP III) guidelines with presence of 3/more of following:[13] (i) Increased WC for men ≥90 cm, for women≥80 cm, (ii) elevated TGLs (≥150 mg/dl), (iii) Low HDL cholesterol (<40 mg/dl in men, <50 mg/dl in women), (iv) Hypertension (≥130/85 mmHg) and (v) FPG (≥100 mg/dl).

### *Statistical analysis*

Data distribution was studied using Kolmogrov Smirnov test. Comparison between means was done using unpaired two-tailed T test for data with normal distribution or Mann-Whitney 'U' test for data not having normal distribution. Correlation between parameters was done using Pearson's correlation or Spearman rank correlation for data having normal and non normal distribution respectively. Statistical analysis was performed using IBM SPSS (Statistical package for the social sciences) version 22. Statistical significance was considered at p<0.05.

The study was approved by institutional ethics committee (ROC.No.AS/11/IEC/SVIMS/IEC No.1118). Written informed consent was obtained from all the study participants for the use of the participant's data for research and educational purposes. The procedures followed were in accordance with the ethical standards of Helsinki declaration revised guidelines (2013).

## RESULTS

Data expressed as Mean ± Standard deviation for data with normal distribution and median (Inter quartile range) for data with non-normal distribution.

Subjects newly diagnosed with hypothyroidism were found to have significantly higher BMI, WC, systolic and diastolic BP (SBP and DBP), with elevated TC, TGL, VLDL and FPG levels compared to controls (Table 1).

Among the 30 hypothyroid subjects 14 subjects were found to have MetS (Table 2).

Association of hypothyroidism with components of MetS found TSH to be associated positively with BMI, WC and DBP, with T4 found to be have negative associations with WC, DBP (Table 3).

Association of hypothyroidism with components of MetS found TSH to be associated positively with FPG, TC, TGL and VLDL, with T4 found to be have negative associations with FPG, TC, TGL, VLDL (Table 4).

Associations between components of MetS found positive associations between (i) BMI with WC, SBP, DBP, TGL and VLDL; (ii) WC with TC, TGL, VLDL, FPG, insulin and HOMA-IR; (iii) SBP with DBP, TGL and VLDL; (iv) DBP with TGL, VLDL and FPG; (v) TC with TGL, VLDL, HDL, LDL and FPG; (vi) TGL with VLDL, FPG, insulin and HOMA-IR; (vii) VLDL with FPG, insulin and HOMA-IR; (viii) HDL with LDL; (ix) FPG with insulin and HOMA-IR; and (x) Insulin with HOMA-IR (Table 5).

**Table 1: Demographic, anthropometric, clinical characteristics and biochemical profile among the study subjects.**

| Variables | Group 1 controls, (n=30) | Group 2 hypothyroid cases, (n=30) | P value |
|---|---|---|---|
| **Age (in years)** | 35.97±9.79 | 38.50±12.44 | 0.385 |
| **Gender (M/F)** | 3/27 | 8/22 | - |
| **BMI (kg/m$^2$)** | 25.53±4.49 | 29.17±3.24 | 0.001* |
| **WC (cm)** | 83.68±6.13 | 93.30±9.93 | <0.001* |
| **SBP (mm Hg)** | 115.57±6.51 | 121.13±12.90 | 0.039* |
| **DBP (mm Hg)** | 72 (5.75) | 81.5 (14.25) | <0.001* |
| **TSH (mIU/l)** | 1.80 (1.78) | 36 (30.73) | <0.001* |
| **T4 (ng/ml)** | 93.5 (21.25) | 53 (23) | 0.001* |
| **TC (mg/dl)** | 159.27±29.72 | 186.40±27.54 | 0.001* |
| **TGL (mg/dl)** | 102.46±56.20 | 194.97±51.37 | <0.001* |
| **VLDL (mg/dl)** | 20.49±11.24 | 38.99±10.27 | <0.001* |
| **LDL (mg/dl)** | 97.78±23.40 | 105.78±22.41 | 0.182 |
| **HDL (mg/dl)** | 41±8.11 | 41.63±5.45 | 0.724 |
| **FPG (mg/dl)** | 82.5 (7.25) | 91 (13.75) | <0.001* |
| **Insulin (µIU/ml)** | 9.90 (7.63) | 10.70 (22.85) | 0.237 |
| **HOMA-IR** | 2.06 (1.28) | 2.41 (6.12) | 0.139 |

*Statistical significance at p<0.05.

**Table 2: Presence of MetS in subjects with hypothyroidism.**

| Clustering of components of MetS | Males, (n=8) | Females, (n=22) | Total, (n=30) |
|---|---|---|---|
| **WC + TGL + HDL** | 01 (12.5%) | 05 (22.72%) | 06 (20%) |
| **WC+TGL+FPG** | 01 (12.5%) | 01 (4.54%) | 02 (6.66%) |
| **TGL+HDL+DBP** | 01 (12.5%) | 0 (0%) | 01 (3.33%) |
| **TGL+HDL+FPG** | 0 (0%) | 01 (4.54%) | 01 (3.33%) |
| **WC+TGL+HDL+FPG** | 0 (%) | 03 (13.6%) | 03 (3.33%) |
| **WC+TGL+HDL+DBP+FPG** | 0 | 01 (4.54%) | 01 (3.33%) |
| **Total no. with MetS** | 03 (37.5%) | 11 (50%) | 14 (46.66%) |

*WC: males ≥90 cms, females ≥80 cms; TGL ≥150 mg/dl; HDL: males <40 mg/dl, females <50 mg/dl; FPG ≥100 mg/dl; SBP≥130 mmHg; DBP≥85 mmHg.

**Table 3: Correlation analysis of hypothyroidism with anthropometric and BP components of MetS.**

| Variables | T4 | TSH |
|---|---|---|
| | ρ | ρ |
| | P | P |
| **BMI (kg/m$^2$)** | -0.236 | 0.342 |
| | 0.069 | 0.007** |
| **WC (cm)** | -0.355 | 0.366 |
| | 0.005* | 0.004** |
| **SBP (mm Hg)** | -0.079 | 0.143 |
| | 0.0550 | 0.277 |
| **DBP (mm Hg)** | -0.331 | 0.378 |
| | 0.010* | 0.003** |

*ρ=Spearman's rho-correlation coefficient, **Statistical significance at p<0.05.

**Table 4: Correlation analysis of hypothyroidism with biochemical components of MetS.**

| Variables | T4 | TSH |
|---|---|---|
| | ρ | ρ |
| | P | P |
| **FPG (mg/dl)** | -0.423 | 0.430 |
| | 0.001** | 0.001 |
| **Insulin (µIU/ml)** | -0.060 | 0.088 |
| | 0.649 | 0.503 |
| **HOMA-IR** | -0.113 | 0.131 |
| | 0.389 | 0.317 |
| **T4 (ng/ml)** | 1 | -0.824 |
| | - | <0.001** |
| **TC (mg/dl)** | -0.437 | 0.385 |
| | <0.001** | 0.002** |
| **TGL (mm Hg)** | -0.567 | 0.592 |
| | <0.001** | <0.001** |
| **VLDL (mm Hg)** | -0.567 | 0.592 |
| | <0.001** | <0.001** |
| **HDL (mm Hg)** | -0.240 | 0.207 |
| | 0.064 | 0.113 |
| **LDL (mm Hg)** | -0.196 | 0.099 |
| | 0.134 | 0.452 |

*ρ=Spearman's rho-correlation coefficient, **Statistical significance at p<0.05.

**Table 5: Correlation analysis between components of MetS.**

| Variables | WC | SBP | DBP | TC | TGL | VLDL | HDL | LDL | FPG | Insulin | HOMA-IR |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | ρ | ρ | ρ | ρ | ρ | ρ | ρ | ρ | ρ | ρ | ρ |
| | P | P | P | P | P | P | P | P | P | P | P |
| **BMI (kg/$^2$)** | 0.255 | 0.356 | 0.508 | 0.195 | 0.440 | 0.440 | -0.156 | 0.005 | 0.224 | 0.222 | 0.225 |
| | 0.05# | 0.005# | <0.001# | 0.135 | <0.001# | <0.001# | 0.234 | 0.972 | 0.086 | 0.089 | 0.083 |
| **WC (cm)** | 1 | 0.026 | 0.221 | 0.341 | 0.435 | 0.435 | 0.104 | 0.150 | 0.413 | 0.276 | 0.317 |
| | | 0.842 | 0.089 | 0.008* | <0.001# | <0.001# | 0.430 | 0.252 | 0.001# | 0.033# | 0.014# |
| **SBP (mm Hg)** | | 1 | 0.696 | 0.105 | 0.349 | 0.349 | -0.197 | -0.007 | 0.175 | 0.067 | 0.056 |
| | | | <0.001# | 0.425 | 0.006# | 0.006# | 0.131 | 0.957 | 0.180 | 0.613 | 0.672 |
| **DBP (mm Hg)** | | | 1 | 0.248 | 0.481 | 0.481 | -0.151 | 0.101 | 0.269 | 0.183 | 0.191 |
| | | | | 0.056 | <0.001# | <0.001# | 0.248 | 0.441 | 0.038# | 0.162 | 0.144 |
| **TC (mg/dl)** | | | | 1 | 0.543 | 0.543 | 0.426 | .0882 | 0.307 | 0.095 | 0.127 |
| | | | | | <0.001# | <0.001# | 0.001# | <0.001# | 0.017# | 0.470 | 0.335 |
| **TGL (mg/dl)** | | | | | 1 | 1.000 | -0.042 | 0.173 | 0.496 | 0.278 | 0.308 |
| | | | | | | <0.001# | 0.750 | 0.187 | <0.001# | 0.031# | 0.018# |
| **VLDL (mg/dl)** | | | | | | 1 | -0.042 | 0.173 | 0.496 | 0.278 | 0.305 |
| | | | | | | | 0.750 | 0.187 | <0.001# | 0.031# | 0.018# |
| **HDL (mg/dl)** | | | | | | | 1 | 0.332 | 0.007 | -0.254 | -0.221 |
| | | | | | | | | 0.010* | 0.957 | 0.050 | 0.090 |
| **FPG (mg/dl)** | | | | | | | | | 1 | 0.477 | 0.585 |
| | | | | | | | | | | <0.001# | <0.001# |
| **Insulin (µIU/mL)** | | | | | | | | | | 1 | 0.985 |
| | | | | | | | | | | | <0.001# |

*ρ=Spearman's rho-correlation coefficient, #Statistical significance at p<0.05.

## DISCUSSION

DISCUSSION An increased prevalence of CVD along with odds of prevalent MetS has been reported with TSH more than 10 mIU/l. The mean TSH levels in present study was 36 mIU/l indicating heightened CVD risk. The lower metabolic rate observed in the hypothyroidism due to a decrease in thermogenesis can lead to the development of obesity. Even mild elevation in TSHs levels are reported to be associated with an increase in the occurrence of obesity.[16] In the present study elevated TSH levels was associated with higher BMI and WC with WC found to be associated with lowered T4 levels as well. Though BMI has been used to measure obesity, it is proposed that body fat distribution indicated by WC, is a better measure of abdominal obesity and a better predictor of CVD.[17] Studies attempting to find the best anthropometric indicator as a possible predictor of dyslipidemia reported WC to have additional positive correlation with TGL [14] as found in present study found. As obesity is found to be associated with hypertension, it is interesting to observe that in the present study only BMI had association with both SBP and DBP. The elevated BP has in turn been reported to be associated with higher TSH levels.[18,19] A significant correlation between DBP but not SBP with hypothyroidism has been reported.[20] Similarly we found T4 and TSH to be associated with only DBP. Thyroid hormones control BP by regulating the cardiac output, peripheral vascular resistance and kidney function.[20]

Thyroid hormone regulates gastrointestinal tract motility, increase glucose absorption, increase the hepatic expression of glucose transporters 2, induce the expressions of HMG-CoA reductase in the liver, sterol regulatory element-binding protein-2 essential for LDL receptor activation and regulation of bile acid synthesis which helps in cholesterol excretion. The above functions are disrupted with lowering of thyroid hormone levels and further the elevated TSH in hypothyroidism has been reported to stimulate cholesterol production, cause an increase in lipolysis with decreased clearance of cholesterol and TGL rich lipoproteins. Progressive changes occur with elevation in TSH independent of thyroid hormone levels.[21] Higher TSH levels have been reported to be associated with increased TGL and a higher risk of MetS.[5] A cross-sectional study to assess thyroid function in MetS diagnosed by NCEP/ATP III revealed positive linear correlation of TSH with TGL, TC, LDL and negative correlation with HDL among Indian patients.[22] Present study found association of hypothyroidism with components of MS. Thyroid hormones directly acts on pancreatic β-cells, stimulating insulin secretion and increases glucagon release by pancreatic α-cells.[23] However in hypothyroid state, decreased skeletal muscle and adipose tissue sensitivity to insulin leads to insulin resistance. Also, higher insulin levels may be due to decreased insulin clearance.[24]In the present study, though insulin levels and HOMA-IR were found to be higher in cases compared to controls, it was not significant, indicating progression to insulin resistance state has not occurred. Insulin and HOMA-IR were found to be associated with WC, FPG, TGL and VLDL components of MetS. It is to be noted that though the mean FPG in hypothyroid subjects was not elevated as per guidelines for MetS (≥100 mg/dl), it was significantly elevated when compared to controls and correlated with insulin and HOMA-IR.[13] The subtle changes in FPG, insulin and HOMA-IR can progress to insulin resistance and development of DM in hypothyroid subjects.

## CONCLUSION

Hypothyroidism is found to be associated with the components of MetS, with presence of MetS observed in 46.66% of the subjects with hypothyroidism. As hypothyroidism leads to metabolic dysregulation similar to the metabolic changes also observed in MetS, screening for presence of MetS in hypothyroidism may help in identifying individuals with heightened CVD risk. Targeted therapeutic interventions and life style modifications can attenuate the compounding effect of MetS on CVD risk in subjects with hypothyroidism.

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