METABOLIC SYNDROME AND LIPID ABNORMALITIES IN NON-OBESE INDIVIDUALS: PREVALENCE AND LIMITATIONS OF BODY MASS INDEX -BASED SCREENING IN A RURAL AFRICAN COMMUNITY

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ABSTRACT:

Background:
Metabolic syndrome (MetS) is a cluster of abnormalities that heighten cardiovascular and type 2 diabetes risk. While obesity is a key driver, metabolic dysfunction can also occur in individuals with normal or low body mass index (BMI), escaping detection by BMI-based screening. This study assessed BMI distribution in MetS patients, focusing on metabolically obese normal-weight (MONW) and metabolically unhealthy lean (MUL) phenotypes, using triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C) as dyslipidemia markers.

Methodology:
This cross-sectional study included 75 MetS patients at Igbinedion University Teaching Hospital, diagnosed using the World Health Organisation (WHO) criteria. BMI stratified patients: underweight (<18.5 kg/m²), normal weight (18.5–24.9 kg/m²), overweight (25–29.9 kg/m²), and obese (≥30 kg/m²). Standard enzymatic assay assessed metabolic parameters, including TG and HDL, which were analysed across BMI categories. Statistical analysis was done using the IBM SPSS version 26, and confidence levels were set at 95% with a p-value of < 0.05 considered statistically significant.

Results:
Overweight (32.0%) and obese (9.3%) patients were fewer than normal-weight (40.0%) and underweight (18.7%) patients meeting MetS criteria. Among normal-weight patients, 33.3% had low HDL-C and 29.3% had high TG. In underweight individuals, 17.3% had high TG and 13.3% low HDL-C. TG levels varied significantly across BMI categories ($p = 0.008$).

Conclusion:
Dyslipidemia is prevalent among MetS patients with normal or low BMI, revealing BMI's inadequacy as a sole risk predictor. Expanded screening incorporating lipid and anthropometric measures is essential, particularly in rural African populations.

Keywords: Metabolic Syndrome, Body Mass Index, Metabolically Obese Normal Weight, Metabolically Unhealthy Lean, High-Density Lipoprotein, Triglycerides

INTRODUCTION

Metabolic syndrome (MetS) is a cluster of interrelated metabolic abnormalities, including central obesity, insulin resistance, dyslipidemia, and hypertension, that significantly increase the risk of cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM) [1]. The prevalence of MetS is increasing globally, driven by lifestyle changes, urbanisation, and dietary shifts, making it a significant public health concern. Traditionally, obesity, particularly central adiposity, has been considered a primary driver of MetS. However, emerging evidence suggests that metabolic dysfunction can occur in individuals with normal or even low body mass index (BMI), challenging the conventional notion that excess weight is the primary determinant of metabolic health [2].

The metabolically obese normal-weight (MONW) and metabolically unhealthy lean (MUL) phenotypes describe individuals who, despite having a normal or low BMI, exhibit metabolic disturbances such as insulin resistance, elevated triglycerides (TG), low high-density lipoprotein (HDL), and systemic inflammation [3]. These phenotypes are thought to arise from factors such as visceral fat accumulation, genetic predisposition, and an unhealthy lifestyle, even in the absence of overt obesity. The presence of these phenotypes underscores the limitations of BMI as a sole marker of metabolic risk. It highlights the need for broader anthropometric and biochemical assessments to accurately identify at-risk individuals [4]. Dyslipidemia, particularly elevated TG and reduced HDL levels, is a hallmark of MetS and a well-established risk factor for CVD. Studies have shown that even in non-obese individuals, high TG and low HDL levels are strongly associated with increased cardiovascular risk, insulin resistance, and endothelial dysfunction [5]. The present study investigates the BMI distribution among MetS patients, with a particular focus on the prevalence of MONW and MUL phenotypes.

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Previous studies have exposed the failure of BMI to serve as a predictor of metabolic dysfunction, such as the study by Bradshaw *et al*. (2013), where about 24% of normal-weight adults (BMI < 25) were metabolically abnormal, placing them at elevated risk for chronic diseases despite appearing "healthy" by BMI standards. [6] Also, A landmark report in *The Lancet Diabetes & Endocrinology* declared BMI unreliable for individual health assessment. Experts instead recommend alternative metrics, such as waist-to-hip ratio or direct adiposity measurements, given the BMI's failure to accurately capture underlying health status. [7] By analysing TG and HDL levels across different BMI categories, this study aimed to highlight the presence of metabolic risk in non-obese individuals and emphasise the inadequacy of BMI as a standalone anthropometric screening tool for risk stratification for cardio-metabolic dysfunction. Furthermore, it explores the potential need for alternative anthropometric indices, such as waist circumference, waist-to-height ratio, body fat percentage, and wrist measurements,

to improve metabolic risk stratification in clinical settings.

METHODS

Study Design

This cross-sectional study was conducted at Igbinedion University Teaching Hospital, Nigeria, involving 75 patients diagnosed with Metabolic Syndrome (MetS) based on the World Health Organisation (WHO) criteria. The minimum sample size was determined and calculated using the Cochran formula [8]:

$n = Z^2 \times p(1–p) / d^2$

Where:

- Z = 1.96 (for a 95% confidence level)
- p = expected prevalence of metabolic syndrome in similar rural Nigerian populations (0.30 from prior studies)
- d = margin of error (0.104, chosen for feasibility in a pilot setting)

Substituting these values:

$n = (1.96^2 \times 0.3 (1 - 0.3)) / (0.104)^2$

$n = (3.8416 \times 0.21) / 0.010816$

$n = 0.806736 / 0.010816$

$n = 74.58$

Thus, the calculated sample size of 75 participants was achieved in this study.

The sample size determination was also in keeping with previous similar studies in comparable populations, such as that of Osuji *et al.* (2012), where a similar number of subjects were included in a study that assessed metabolic syndrome in newly diagnosed type 2 diabetes mellitus using National Cholesterol Education Program – Adult Treatment Panel III (NCEP-ATP III) in Nnewi, Nigeria. Also, Aşık *et al*. (2016), who evaluated obesity and metabolic syndrome in Turkey, had a similar sample size. [9,10]

According to the WHO, MetS is identified by the presence of insulin resistance (e.g., type 2 diabetes or impaired fasting glucose) plus at least two of the following:

- Hypertension: Blood pressure ≥140/90 mmHg or current use of antihypertensive medication.
- Dyslipidemia: Triglycerides (TG) ≥150 mg/dL and/or HDL cholesterol <35 mg/dL in men or <39 mg/dL in women.
- Central obesity: Waist-to-hip ratio >0.90 for men, >0.85 for women, or body mass index (BMI) >30 kg/m².
- Microalbuminuria: Urinary albumin excretion rate ≥20 µg/min or albumin-to-creatinine ratio ≥30 mg/g. [11]

Inclusion criteria included Patients aged ≥18 years, those who met the WHO diagnostic criteria for MetS and adult patients without chronic inflammatory conditions, pregnancy, or recent acute illnesses.

Exclusion criteria included patients < 18 years and those who didn't meet the WHO diagnostic criteria for MetS, those below 18 years and adult patients with chronic inflammatory conditions, pregnancy, or recent acute illnesses.

Anthropometric Measurements

Body Mass Index (BMI) Measurement

BMI was calculated using the standard formula: *BMI = Weight (kg) / Height (m)²*

Weight Measurement:

- Weight was measured using a digital weighing scale (Seca 876, Germany).
- Participants were instructed to stand upright in the centre of the scale, with feet slightly apart.
- Measurements were taken with participants wearing light clothing and no shoes to minimise external weight influence.
- The weight was recorded in kilograms (kg) to the nearest 0.1 kg.

Height Measurement:

- Height was measured using a stadiometer, ensuring accuracy and reproducibility.
- Participants were instructed to stand upright, with their heels, buttocks, and upper back against the vertical board of the stadiometer.
- The head was positioned in the Frankfurt horizontal plane (where the lower orbital margin is aligned with the external auditory meatus).
- The measurement was taken at full inspiration, and the height was recorded in meters (m) to the nearest 0.1 cm.

BMI classifications:

- Underweight: <18.5 kg/m²
- Normal weight: 18.5–24.9 kg/m²
- Overweight: 25–29.9 kg/m²
- Obese: ≥30 kg/m² [12]

Biochemical Analysis

- Blood samples were collected after an 8–12 hour overnight fast for lipid profile analysis.
- Venous blood (5 mL) was drawn from the antecubital vein using a sterile vacutainer system.
- Samples were collected into plain (serum) tubes and allowed to clot at room temperature for 30 minutes.
- Clotted blood samples were centrifuged at 3000 rpm for 10 minutes to obtain clear serum.
- If analysis was delayed, the separated serum was immediately analysed or stored at -20°C.

Triglycerides (TG) Assay

Serum TG levels were measured using an enzymatic colorimetric method (GPO-PAP method) with a commercial kit (Roche Diagnostics, Germany). The procedure involved:

1. Hydrolysis of triglycerides by lipoprotein lipase into glycerol and free fatty acids.
2. Phosphorylation of glycerol by glycerol kinase, followed by oxidation via glycerol-3-phosphate oxidase, producing hydrogen peroxide.
3. The hydrogen peroxide reacted with 4-aminophenazone in the presence of peroxidase, forming a quinoneimine dye, which was measured spectrophotometrically at 505 nm. [13]

High-Density Lipoprotein (HDL) Assay

HDL cholesterol was measured using a direct enzymatic clearance method (HDL-C plus method, Roche Diagnostics, Germany):

1. Selective blocking of non-HDL lipoproteins (LDL, VLDL, and chylomicrons).
2. Enzymatic degradation of HDL cholesterol, producing a colored

complex, which was measured at 600 nm. [14]

All assays were performed using a Roche Cobas c311 autoanalyser, following the manufacturer's protocols. Internal quality controls were conducted before each batch of analysis.

Statistical Analysis

Data were analysed using IBM SPSS version 26.0 (IBM Corp, USA).

- Categorical variables were analysed using the chi-square test, while Kendall Tau-b was used for ordinal variables.
- A p-value <0.05 was considered statistically significant.

RESULTS

BMI Distribution among MetS Patients

Among the 75 MetS patients, 32.0% were classified as overweight, while 9.3% were obese. Notably, a considerable proportion of patients fell within the normal-weight (40%) and underweight (18.7%) categories, showing the prevalence of metabolically obese normal-weight (MONW) and metabolically unhealthy lean (MUL) phenotypes

Table 1: BMI Distribution among MetS Patients

BMI Category	Percentage (%)
Underweight (<18.5 kg/m²)	18.7%
Normal weight (18.5–24.9 kg/m²)	40.0%
Overweight (25–29.9 kg/m²)	32.0%
Obese (≥30 kg/m²)	9.3%

Dyslipidemia across BMI Categories

Relationship between BMI and TG in Patients with MetS

Among normal-weight individuals (40%), 29.3% exhibited elevated triglyceride (TG) levels, while among underweight individuals, 17.3% had elevated TG. In contrast, overweight patients demonstrated a lower prevalence of elevated TG (13.3%), with the lowest occurrence observed in obese patients, where only 2.7% had elevated TG levels. Statistical analysis revealed a significant variation in TG levels across BMI categories ($\chi^2 = 17.412$, df = 6, $p = 0.008$), suggesting that dyslipidemia occurs independently of obesity status. Furthermore, the negative Kendall's tau-b value ($\tau = -0.223$, $p = 0.031$) indicates an inverse correlation between BMI and TG levels, implying that higher BMI does not necessarily correlate with higher triglyceride concentrations.

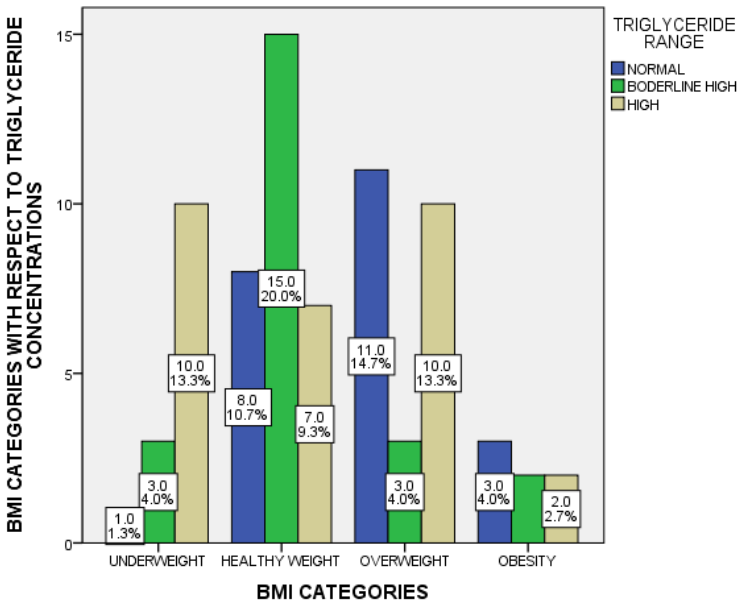

FIGURE 1: Relationship between BMI and TG in Patients with MetS

Underweight (<18.5 kg/m²), Normal weight (18.5–24.9 kg/m²), Overweight (25–29.9 kg/m²), Obese (≥30 kg/m²),

Normal TG: <150 mg/dL, Borderline High TG: 150–199 mg/dL , High TG: ≥ 200mg/dL

Relationship between BMI and HDL in Patients with MetS

In this study population, low concentrations of high-density lipoprotein (HDL) cholesterol, a well-established risk factor for metabolic and cardiovascular diseases, were observed across all BMI categories, with a statistically significant difference in prevalence ($\chi^2 = 24.153$, df = 6, $p < 0.05$). Among underweight individuals, 13.3% exhibited low HDL levels, while 33.3% of normal-weight individuals had reduced HDL concentrations. The prevalence of low HDL was lower in overweight individuals (32%), with 21.3% affected, and was least common in obese individuals (9.3%), where only 2.7% had reduced HDL levels.

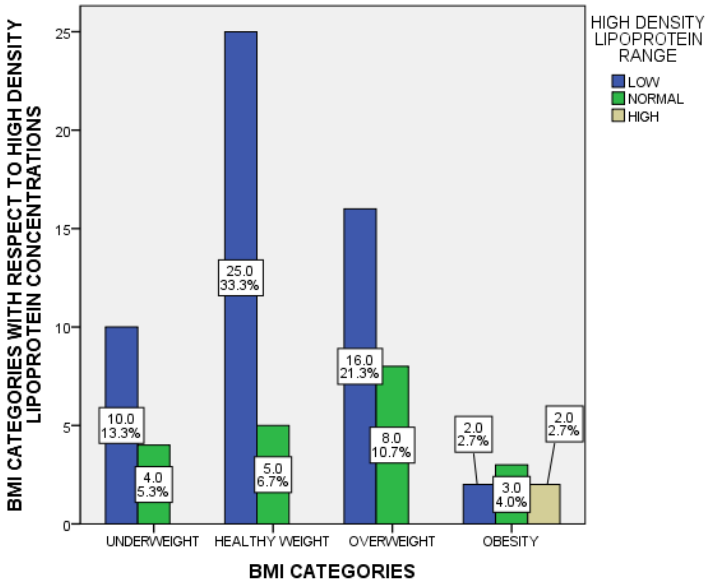

FIGURE 2: Relationship between BMI and HDL in Patients with MetS

Underweight (<18.5 kg/m²), Normal weight (18.5–24.9 kg/m²), Overweight (25–29.9 kg/m²), Obese (≥30 kg/m²),

Low HDL = < 40 mg/dL, Normal HDL: 40–59 mg/dL High HDL: ≥ 60 mg/dL

DISCUSSION

BMI Distribution among MetS Patients

This study highlighted the significant proportion of non-obese individuals with MetS, reinforcing that BMI alone may not be sufficient for metabolic risk assessment. The distribution challenges the conventional reliance on BMI as a singular indicator of metabolic health and highlights the complex and multifaceted nature of metabolic syndrome, where individuals with lower BMI may still exhibit significant metabolic dysfunction. The MONW and MUL phenotypes observed in this study align with a previous finding that normal-weight individuals can exhibit metabolic dysfunction. [15] This paradox is often attributed to underlying factors such as insulin resistance, visceral adiposity, dysregulated adipokine secretion, genetic susceptibility, and sedentary lifestyles, all of which contribute to an increased cardiometabolic risk despite an ostensibly healthy weight. These findings reinforce the necessity of incorporating additional anthropometric indices (e.g., waist circumference, body fat percentage, and wrist circumference), alongside comprehensive metabolic profiling, to improve early detection and risk stratification for MetS, particularly in resource-limited settings. [15]

Dyslipidemia across BMI Categories

Relationship between BMI and TG in Patients with MetS

This study revealed that higher BMI does not necessarily correlate with higher triglyceride concentrations (τ = -0.223, p = 0.031). This unexpected trend challenges the conventional understanding of obesity-driven dyslipidemia and highlights the need for a more detailed metabolic risk assessment that extends beyond BMI, particularly in populations where lean individuals may still exhibit significant lipid abnormalities.

One possible explanation for the pattern is the metabolically obese normal-weight (MONW) phenotype, a condition in which individuals classified as normal-weight based on BMI exhibit metabolic features characteristic of obesity, including insulin resistance and visceral adiposity. Studies have shown that MONW individuals are predisposed to dyslipidemia, including elevated TG levels, despite having a normal BMI. Karelis *et al.* found that metabolically healthy obese (MHO) individuals had significantly lower plasma triglycerides and higher HDL-cholesterol compared to insulin-resistant counterparts despite similar total body fat ($p < 0.05$). [16] Also, Snijder *et al.* reported that greater trunk fat mass was strongly associated with higher fasting glucose (standardized $\beta \approx 0.44$ in men) and post-load glucose ($\beta \approx 0.41$), while greater leg fat mass showed inverse associations ($\beta \approx -0.24$ for fasting glucose, $\beta \approx -0.12$ for post-load glucose), exposing how fat distribution, not BMI per se, predicts metabolic outcomes. This shows the limitations of BMI as a sole indicator of metabolic health, as it does not distinguish between lean mass and fat mass, nor account for fat distribution. [17] Individuals with normal BMI but high visceral fat may exhibit metabolic disturbances similar to those observed in obesity.

Genetic and lifestyle factors could also contribute to the observed trend. Genetic predisposition plays a crucial role in lipid metabolism, with certain polymorphisms influencing TG concentrations independent of BMI. Kathiresan *et al.* demonstrated that individuals in the highest genetic risk score quintile exhibited triglyceride levels approximately 26 mg/dL higher than those in the lowest quintile, a clear genetic influence independent of BMI. [18] Additionally, dietary habits, particularly high intake of simple carbohydrates and saturated fats, as well as physical inactivity, have been strongly linked to elevated TG levels. Parks & Hellerstein observed that individuals consuming a low-fat, high-carbohydrate diet experienced a ~60% elevation in fasting triglyceride concentrations and a 37% decrease in VLDL-TG clearance compared to baseline values. [19] These factors may disproportionately affect normal-weight individuals with a genetic predisposition to dyslipidemia. The findings of this study contrast with previous research, such as a study conducted among civil servants in Abakaliki, South Eastern Nigeria, where overweight and obese individuals had significantly higher TG levels than their normal-weight counterparts. [20] This discrepancy highlights potential population-specific variations in lipid metabolism, likely influenced by genetic, dietary, and environmental factors.

Another critical consideration is the reliance on BMI as a measure of adiposity. BMI fails to differentiate between subcutaneous and visceral fat, the latter being a stronger predictor of metabolic disturbances. Previous research has suggested that individuals with central obesity, even at a normal BMI, are more likely to develop dyslipidemia and other metabolic disorders. [21] Therefore, alternative markers such as waist-to-hip ratio or body fat percentage may provide more accurate assessments of metabolic risk. The inverse association between BMI and TG levels challenges the conventional view of obesity-driven dyslipidemia and shows the importance of comprehensive metabolic assessments. The findings suggest that lean individuals are not necessarily metabolically healthy and highlight the need for targeted screening strategies that extend beyond BMI.

Relationship between BMI and HDL in Patients with MetS

This study suggests that low HDL cholesterol is not exclusively associated with higher BMI, but is also prevalent among non-obese individuals ($\chi^2 = 24.153$, $df = 6$, $p < 0.05$). This can be attributed to the role of physical activity. Regular aerobic exercise has been shown to increase HDL levels, while a sedentary lifestyle is associated with reduced HDL concentrations. [22] The decreasing prevalence of low HDL from normal-weight to obese individuals in our study may reflect differences in lifestyle habits rather than inherent metabolic advantages. Some overweight and obese individuals may engage in more physical activity than their normal-weight counterparts, thereby improving their HDL profile. Additionally, paradoxical findings in obesity research suggest that certain obese individuals maintain a healthier metabolic profile, a phenomenon referred to as metabolically healthy obesity (MHO). [23]

This inverse relationship between BMI and low HDL levels suggests that HDL dysregulation extends beyond obesity-driven metabolic dysfunction, as discussed earlier, with genetic and lifestyle factors playing a key role. Comparing our findings with existing literature, similar

patterns have been reported in other populations. A study by Heianza et al. [24] found that lean individuals with MetS exhibited significantly lower HDL levels compared to overweight individuals without metabolic abnormalities. Similarly, a study conducted in South Asian populations showed that a high prevalence of low HDL was observed even in individuals with normal BMI, likely due to genetic predisposition and dietary habits. [25] These findings, again, support the argument that BMI alone is an inadequate predictor of metabolic health and that a more comprehensive assessment, including lipid profile evaluation, insulin sensitivity, and body composition analysis, is necessary.

CONCLUSION

This study exposes the inadequacy of BMI as a sole determinant of metabolic health, highlighting the significant proportion of non-obese individuals who exhibit metabolic syndrome (MetS). The presence of metabolically obese normal-weight (MONW) and metabolically unhealthy lean (MUL) phenotypes reinforces the complexity of metabolic dysfunction, which extends beyond conventional obesity markers. Factors such as insulin resistance, visceral adiposity, dysregulated adipokine secretion, and genetic predisposition contribute to increased cardiometabolic risk, even among individuals with normal BMI. A multifaceted approach to metabolic risk assessment, integrating easy anthropometric indices such as waist circumference, is essential for early detection and targeted intervention. This approach is particularly crucial in resource-limited settings, where early identification of at-risk individuals can improve preventive strategies and reduce the burden of cardiovascular disease and metabolic disorders. Future research should explore population-specific variations in lipid metabolism and the interplay between genetic and environmental factors in shaping metabolic risk.

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