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Original Article

Early Metabolic Alterations and Predictors of Obesity Among Young Adults in Indonesia: Focus on Lipid Abnormalities and Cardiometabolic Risk

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Abstract

Background: The prevalence of obesity and metabolic syndrome (MetS) is rising among young adults, contributing to early cardiometabolic risk amid lifestyle transitions in Indonesia. This study examined the associations between obesity, MetS components, and body composition among Indonesian young adults.

Methods: A total of 99 participants were classified based on body mass index (BMI) into normoweight (n=51) and obese (n=48) groups. Anthropometric measurements, body composition, blood pressure, and lipid profiles were assessed. MetS was defined according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria. Bivariate and multivariable logistic regression analyses were performed to identify factors independently associated with obesity.

Results: Obesity was significantly associated with several MetS components. Abdominal obesity was observed only in the obese group (33.3%). Hypertriglyceridemia and low HDL cholesterol were more prevalent among obese participants compared with normoweight individuals (27.1% vs. 5.9% and 51.0% vs. 31.4%, respectively). MetS (≥3 NCEP ATP III criteria) was identified in 12.5% of obese participants and was absent in the normoweight group. Multivariable analysis identified family history of hypertension (adjusted OR 2.82; 95% CI 1.06–7.48) and elevated triglyceride levels (adjusted OR 4.78; 95% CI 1.13–20.22) as independent predictors of obesity.

Conclusions: Obese young adults exhibit early metabolic abnormality, particularly abdominal obesity, low HDL cholesterol, and hypertriglyceridemia. Early metabolic screening and targeted preventive strategies are important to reduce future cardiometabolic risk.

Keywords: Cardiometabolic risk, metabolic syndrome, obesity, triglycerides, young adults

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Introduction

The increasing prevalence of obesity and metabolic syndrome (MetS) among young adults has become a major global public health concern. In 2022, the World Health Organization (WHO) reported that over 2.5 billion adults were classified as overweight and over 890 million as obese.[1] Obesity is a key driver in the development of MetS, which substantially increases the risk of cardiovascular disease (CVD), type 2 diabetes mellitus (T2DM), and long-term metabolic dysfunction.[2] According to the National Cholesterol Education Program-Adult Treatment Panel (NCEP-ATP) III, MetS is diagnosed when three or more abnormalities are present, including central adiposity, dyslipidemia, elevated blood pressure, and impaired glucose regulation.[3] MetS in young adults often remains undiagnosed until T2DM or other complications develop, contributing to increased long-term morbidity and premature mortality from non-communicable disease.[4]

In Indonesia, rapid lifestyle transitions marked by increased consumption of energy-dense foods, reduced physical activity, and

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more sedentary behaviors have contributed to rising rates of obesity, MetS, and early-onset T2DM among the productive age group of 18–59 years.[5,6] National Survey data from 2023 reported that the proportion of hypertension cases accompanied by T2DM and central obesity is 3 to 3.4 fold higher than in individuals without central obesity, while the prevalence of T2DM is 1.3 fold higher among individuals with sedentary lifestyles.[5]

Young adulthood as a critical period for metabolic health as body composition during this stage strongly predicts future cardiometabolic outcomes.[7] Longitudinal cohort studies consistently show that younger adults experience the greatest rates of weight gain and have the highest risk of transitioning to a higher BMI categories compared with older age groups.[7,8] Notably, Asian populations, including Indonesians, tend to develop metabolic complications at lower levels of adiposity compared with Western population.[9] Despite this increased vulnerability, data on obesity and MetS among young adults in Indonesia remain limited. Most studies on MetS in Indonesia have focused on adult and elderly populations, while evidence involving adolescents and young adults is still limited.[10] This gap may partly due to the often subtle or absent clinical manifestations of MetS in younger individuals, leading to underdiagnosis in this age group.

Diagnosing metabolic syndrome only in adulthood may represent a delayed intervention, as many metabolic complications may already be present. Early identification of MetS allows timely lifestyle modifications and preventive interventions that may reduce the risk of future complications such as T2DM, CVD, and stroke.[11] Therefore, understanding the early manifestations of MetS and its associated risk factors among young adults is essential for improving prevention strategies and reducing the long-term burden of chronic metabolic diseases. Therefore, this study aimed to examine the association between obesity, MetS, and body composition among Indonesian young adults to support early identification and prevention of metabolic complications.

Methods

This cross-sectional study included young adults aged 18 to 24 years, who were grouped into obese and normoweight groups. Participants were selected using purposive sampling based on predefined inclusion and exclusion criteria. Obesity was defined as body mass index (BMI) >25 kg/m^2, whereas normoweight group was defined as BMI between 18.5 and 24.9 kg/m^2. Individuals who were using medications such as steroids, thyroid hormones, or appetite suppressants, as well as those who were pregnant or lactating or had eating disorders such as bulimia or anorexia nervosa, were excluded from the study.

Ethical approval for this study was obtained from the Health Research Ethics Committee of Cibabat Regional General Hospital, Cimahi City, Indonesia (No. 070/44/Ethical Clearance/RSUD Cibabat/VI/2023). After obtaining written informed consent, participants completed questionnaires to collect information on demographic characteristics, personal and family health history, and cardiovascular disease (CVD) risk factors. Physical examination were subsequently performed, including anthropometric measurements, body composition analysis, and blood pressure measurement.

Anthropometric measurements included height, weight, waist circumference, hip circumference, triceps skinfold thickness (TSFT), and upper arm circumferences. BMI was calculated as weight in kilograms divided by height in meters squared (kg/m^2).

Body composition analysis was conducted using a bioelectrical impedance analysis (BIA) device (Omron HBF 375, Omron Healthcare Co., Ltd., Kyoto, Japan) to assess body fat percentage, visceral fat percentage, and skeletal muscle percentage. Blood pressure was measured according to the recommendations of the American Heart Association.[12]

Fasting blood samples were collected, and serum from serum separator tube (SST) was isolated by centrifugation at 1,500 g for 15 minutes at 4°C. The serum samples were stored at −80°C until analysis (Biopath Laboratory, Bandung-Indonesia). Biochemical parameters measured included triglycerides (TAG), high density lipoprotein (HDL-c), low density lipoprotein (LDL-c), total cholesterol (TC) and fasting blood glucose (FBG). All analyses were performed using an automated chemistry analyzer (Mindray BS-180, Mindray Bio-Medical Electronics Co., Ltd., Shenzhen, China). FBG was measured using the glucose oxidase-peroxidase (GOD-POD) method, TC using the cholesterol oxidase- peroxidase (CHOD-POD) method, and TAG using the glycerokinase –peroxidase method. LDL-c and HDL-c were measured using direct enzymatic method.

MetS was defined according to the NCEP/ATP III criteria. Participants were classified as having MetS if they met three or more of the following five criteria: elevated waist circumference, high blood pressure, high fasting glucose, elevated triglycerides, and low HDL cholesterol.[3]

Data were analyzed using the Statistical Package for Social Science (SPSS, version 26.0; IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize participant characteristics, with continuous variables presented as mean ± standard deviation (SD) and categorical variables as frequencies and percentages.

Univariate analysis described the distribution of variables within the obese and normoweight groups. Bivariate analysis included independent t-tests or Mann-Whitney tests for continuous variables, depending on the normality of data distribution. For categorical variables, chi-square tests or Fisher's exact tests were applied as appropriate. Pearson or Spearman correlation tests were performed to evaluate associations between variables. Statistical significance was set at $p<0.05$.

In order to minimize potential confounding due to interrelated metabolic variables, multivariable logistic regression analysis was conducted. Two separate models were performed: one including demographic and behavioral variables, and another including metabolic syndrome components based on the NCEP ATP III criteria. Variables with $p<0.25$ in bivariate analysis were entered into the regression model using the Enter Method. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were reported. Model performance was evaluated using the Omnibus χ^2 statistic, Nagelkerke R^2, and Hosmer–Lemeshow goodness-of-fit test.

Results

A total of 99 young adults aged 18–24 years were included in the study, consisting of 51 participants with normoweight and 48 participants with obesity. As shown in Table 1, females were more prevalent in the normoweight group compared with the obese group (74.51% vs 54.17%, p=0.03). Metabolic syndrome (MetS) was observed only among obese participants (12.5%, p=0.01). Smoking history was also significantly higher in the

Table 1 Characteristics of Young Adults Aged 18–24 Years Based on Body Mass Index

Characteristic	Group		p-value	Correlation Coefficient (r)
	Normoweight (n= 51)	Obese (n=48)		
	n (%)	n (%)		
Gender				
Male	13 (25.49)	22 (45.83)	0.03**	0.23
Female	38 (74.51)	26 (54.17)		
Mets*				
Yes	0 (0)	6 (12.5)	0.01**	0.25
No	51 (100)	42 (87.5)		
Medical history				
Smoking	4 (7.84)	15 (31.25)	0.00**	0.29
CVD	0 (0)	0 (0)	1	-
Hypertension	4 (7.84)	4 (8.33)	1	-
T2DM	2 (3.92)	1 (2.08)	1	-
Family History				
CVD	7 (13.73)	12 (25)	0.2	-
Hypertension	14 (27.45)	24 (50)	0.02**	0.23
T2DM	12 (23.53)	15 (31.25)	0.5	-
Hypercholesterolemia	10 (19.61)	16 (33.33)	0.17	-
Hypertriglyceridemia	2 (3.92)	1 (2.08)	1	-

Note: *MetS (metabolic syndrome) was defined according to the NCEP/ATP III criteria as the presence of three or more of the following: elevated waist circumference, high blood pressure, elevated fasting glucose, elevated triglycerides, and low HDL cholesterol. **p<0.05 indicates statistical significance. CVD= cardiovascular disease; T2DM= type 2 diabetes mellitus.

Table 2 Correlation of Anthropometric, Body Composition, and Metabolic Parameters Among Young Adults Aged 18–24 Years Based on Body Mass Indexx

Parameters	Group		p-value	Correlation Coefficient (r)
	Normoweight (n= 51)	Obese (n=48)		
	Mean ± SD	Mean ± SD		
Age (years)	20.84±0.67	21.06±0.94	0.30	-
Anthropometric and body compositition				
Body age (years)	26.69±5.56	44.94±8	0.00*	0.10
Waist circumference (cm)	72.83±6.09	92.19±11.49	0.00*	0.13
Hip circumference (cm)	84.55±7.92	103.09±9.65	0.00*	-0.23
Waist hip ratio	0.87±0.08	0.89±0.07	0.02*	0.00
Upper arm circumference (cm)	25.85 ±2.71	32.94±4.11	0.00*	-0.07
TSFT (cm)	2.77±0.82	3.64±1.15	0.00*	0.25
Body fat (%)	27.29±5.78	33.34±5.41	0.0*	-0.11
Visceral fat (%)	3.73±1.91	12.99±6.46	0.00*	-0.02
Subcutaneous fat (%)				
Whole	22.12±5.44	27.97±7.48	0.00*	0.22
Trunk	18.90±4.86	24.74±6.96	0.00*	-0.08
Arms	37.12±10.03	41.25±11.27	0.04*	-0.1
Legs	33.57±8.43	39.97±10.68	0.00*	-0.12
Muscle mass/skeletal muscle (%)				
Whole	27.93±3.89	26.62±3.85	0.09	-
Trunk	22.79±3.35	19.68±3.57	0.00*	-0.11
Arms	32.03±5.06	28.58±7.65	0.03*	-0.11
Legs	40.99±6.44	41.7±5.93	0.00*	0.03
Metabolic parameters				
SBP (mmHg)	108.41±14.84	118.27±12.23	0.00*	-0.16
DBP (mmHg)	75.82±9.22	79.79±7.48	0.02*	-0.25
TC (mg/dl)	184.84±36.79	194.42±34.45	0.12	-
HDL-c (mg/dl)	52.51±8.16	45.77±9.10	0.00*	0.13
LDL-c (mg/dl)	103.22±33.43	113.17±28.60	0.02*	0.38
TAG (mg/dl)	88.65±45.21	126.79±67.04	0.00*	0.15
FBG (mg/dl)	72.22±6.88	73.17±7.45	0.41	-

Note: TSF= Triceps skinfold thickness, SBP= Systolic blood pressure, DBP= Diastolic blood pressure, TC= Total cholesterol, HDL-c = High-density lipoprotein cholesterol, LDL-c = Low-density lipoprotein cholesterol, TAG= Triglycerides, FBG = Fasting blood glucose, *Statistically significant at p<0.05.

obese group compared with the normoweight group (31.25% vs 7.84%, p<0.001).

Interestingly, a higher proportion of obese young adults had a family history of hypertension (50%) compared with those in the normoweight group (27.45%) (p=0.02). However, no significant differences were observed between the two groups in terms of family history of cardiovascular diseases (CVD), type 2 diabetes mellitus (T2DM), hypercholesterolemia, or hypertriglyceridemia.

As expected, the obese group had significantly higher Anthropometric and body compositition compared with normoweight group (Table 2). These included body age (44.94±8 vs. 26.69±5.56 years, p<0.01), waist circumference (92.19±11.49 vs. 72.83±6.09 cm, p<0.01), hip circumference (103.09±9.65 vs. 84.55±7.92 cm, p<0.01), waist-hip ratio (0.89±0.07 vs. 0.87±0.08, p=0.024), and upper arm circumference (32.94±4.11 cm vs 25.85±2.71, p<0.01) Moreover, obese individuals showed significantly higher triceps skinfold thickness (TSFT), body fat percentage, and visceral fat percentage.

The distribution of subcutaneous fat, expressed as a percentage of the total body fat, including the trunk, arms, and legs, was also higher in the obese group. However, no noticeable difference was observed in whole-body muscle mass percentages between the

Table 3 Comparison of Metabolic Syndrome Components Among Young Adults Aged 18–24 Years Based on NCEP/ATP III Criteria

NCEP/ATP III Criteria	Group		p-value	Correlation Coefficient (r)
	Normoweight (n= 51)	Obese (n=48)		
	Mean ± SD	Mean ± SD		
Impaired glucose tolerance (FPG ≥100 mg/dL)	0 (0.0)	0 (0.0)	1.00	-
Abdominal obesity (WC >02 cm in men; >88 cm in women)	0 (0.0)	17 (33.33)	0.00*	0.43
Hypertriglyceridemia (≥150 mg/dL)	3(5.88)	13 (27.08)	0.00*	0.28
Low HDL-c (<40 mg/dL in men; <50 mg/dL in women)	16 (31.37)	26 (50.98)	0.03*	0.23
High blood pressure (≥130/85 mmHg)	3 (5.88)	3 (6.25)	1.00	-
Number of NCEP/ATP III criteria:				
0	30 (58.82)	12 (25.00)	0.00*	0.32
1	20 (39.22)	19 (39.58)	1.0	-
2	1 (1.96)	11 (22.92)	0.00*	0.31
≥3 (Metabolic syndrome)	0 (0.0)	6 (12.50)	0.01*	0.25

Note: NCEP/ATP III = National Cholesterol Education Program Adult Treatment Panel III, FPG = Fasting plasma glucose, WC= Waist circumference, HDL-c = High-density lipoprotein cholesterol, *Statistically significant at $p < 0.05$.

two groups (p=0.09).

In terms of metabolic parameters, both systolic and diastolic blood pressure were significantly higher in the obese group (p<0.01 and 0.022, respectively). Additionally, lipid profile components, including HDL-c, LDL-c, and triglycerides (TAG), showed notable differences between the groups.

A comparative analysis in young adults revealed a statistically significant association between obesity and a higher prevalence of abdominal obesity (33.33% vs. 0%, p<0.01), hypertriglyceridemia (27.08% vs. 5.88%, p=0.00), and reduced HDL-c levels (50.98%

Table 4 Family History of Hypertension as an Independent Demographic Predictor of Obesity in Young Adults

Predictor	Adjusted OR	95% CI	p-value
Gender (Female vs Male)	0.76	0.24–2.44	0.641
Metabolic syndrome (Yes vs No)	-	-	0.999
Smoking (Yes vs No)	3.18	0.76–13.31	0.116
Family history of hypertension – Present	2.82	1.06–7.48	0.038*
Family history of hypertension – Unknown	1.19	0.16–8.86	0.864

Note: NCEP/ATP III = National Cholesterol Education Program Adult Treatment Panel III, FPG = Fasting plasma glucose, WC = Waist circumference, HDL-c = High-density lipoprotein cholesterol, *Statistically significant at p<0.05

Table 5 Elevated Triglycerides as the Only Metabolic Component Independently Associated With Obesity

Predictor	Adjusted OR	95% CI	p-value
Waist circumference (High vs Normal)	-	-	0.998
Triglycerides (High vs Normal)	4.78	1.13–20.22	0.033
HDL-c (Low vs Normal)	1.97	0.76–5.15	0.166

Note: HDL-c= high-density lipoprotein cholesterol. Logistic regression was adjusted for waist circumference (WC), triglycerides (TG), and HDL-c categories based on the NCEP ATP III criteria. The extremely large odds ratio for waist circumference indicates quasi-complete separation. Model fit statistics: Omnibus χ^2 p<0.001; Nagelkerke R^2=0.414; Hosmer–Lemeshow test p=0.994; classification accuracy=73.7%. Statistically significant at p<0.05

vs. 31.37%, p=0.03). However, no statistically significant differences were observed for impaired glucose tolerance or elevated blood pressure between the groups (Table 3). Obese young adults were also more likely to present multiple NCEP/ATP III criteria compared with the normoweight group.

The first logistic regression model examining sociodemographic and clinical predictors of obesity was statistically significant ($\chi^2(6)=21.02$, p=0.002), explaining approximately 25.5% of the variance in obesity (Nagelkerke R^2 of 0.255). Model calibration was adequate (Hosmer–Lemeshow p=0.801). Among the predictors included, only family history of hypertension was independently associated with obesity. Participants with a family history of hypertension had higher odds of being obese (OR=2.82, 95% CI: 1.06–7.48, p=0.038). Gender, smoking status, and metabolic syndrome status were not significantly associated with obesity (p>0.05). The overall classification accuracy of the model was 67.7% (Table 4).

A second logistic regression model examining metabolic risk factors was also statistically significant ($\chi^2(3)=36.75$, p<0.001). This model explained approximately 41.4% of the variance in obesity (Nagelkerke $R^2=0.414$) and demonstrated excellent calibration (Hosmer–Lemeshow p=0.994). Among the predictors included, only triglycerides levels were independently associated with obesity. Participants with elevated triglycerides had 4.78 times higher odds of being obese compared with those with normal triglycerides levels (OR=4.78; 95% CI: 1.13–20.22; p=0.033).

HDL cholesterol was not significantly associated with obesity (OR = 1.97; 95% CI: 0.76–5.15; p=0.166). Waist circumference showed an extremely large and unstable odds ratio (OR $>2.2\times10^9$) with a non-significant p-value (p=0.998). This occurred due to quasi-complete separation, as nearly all obese participants had elevated waist circumference, whereas almost all normoweight participants had normal values. This pattern prevented the model from estimating a stable regression coefficient for waist circumference despite its clear clinical relevance. The model achieved an overall classification accuracy of 73.7%, correctly classifying 94.1% of normoweight participants and 52.1% of obese participants (Table 5).

Discussion

This study demonstrates that obesity in young adults is strongly associated with early alterations in body composition and metabolic parameters, including the presence of metabolic syndrome (MetS) components. Although the participants were within a relatively narrow age range (18–24 years), clear differences were observed between obese and normoweight individuals, indicating that metabolic deterioration may begin earlier than often recognized.

One of the key findings is that 12.5% of obese young adults have met the NCEP/ATP III criteria for MetS, whereas none of the normoweight participants met the threshold. This finding supports existing evidence that obesity, particularly central adiposity, is a primary driver of clustered metabolic abnormalities and future cardiometabolic diseases. Central adiposity is strongly linked to dysregulated adipokine secretion, chronic low-grade inflammation, and hepatic lipid accumulation, which ultimately promote insulin resistance and dyslipidemia.[2,13] These mechanisms likely explain the elevated triglycerides, reduced HDL-c, and higher blood pressure observed among obese participants.

A significant difference in gender distribution was also observed, with female being more represented in the normoweight group. This finding contrasts with global data showing a higher prevalence of obesity among female, which may be influenced by hormonal factors, sociocultural norms, and lifestyle behaviors.[14] However, gender likely operates within a multifactorial framework involving genetic, environmental, and behavioral contributors to obesity.[15]

The observed association between obesity and MetS components further supports the well-established pathophysiological link between these conditions. The absence of MetS among normoweight participants, underscores the important role of obesity in the early development of this metabolic cluster, which significantly elevates the risk of CVD and T2DM. Furthermore, a significant association between obesity and smoking history observed in this study may reflect clustering of unhealthy behaviors, where smoking coexist with other lifestyle factors such as poor diet and physical inactivity, thereby compounding metabolic risk.[16]

Family history of hypertension also emerged as an important factor associated with obesity. A previous study has shown that obesity is a well-established risk factor for hypertension and T2DM, largely through mechanisms involving insulin resistance,

endothelial dysfunction, and systemic inflammation.[17] Genetic predisposition and shared family environmental factors may contribute to the early development of these conditions.[18,19] Although the family history of hypercholesterolemia and hypertriglyceridemia did not differ between the two study groups, the weak positive correlation suggests a potential link with obesity that needs further investigation. These findings highlight the importance of monitoring lipid profiles in obese individuals as part of a comprehensive strategy for managing cardiovascular risk.

Obese individuals exhibit adverse alterations in body composition and metabolism. This significant difference in body age suggests that obesity is associated with accelerated biological aging, indicating that increased adiposity can contribute to premature aging through mechanism such as systemic inflammation and oxidative stress.[20] Moreover, obese individuals showed higher body age, waist and hip circumferences, and waist-to-hip ratios, reflecting increased central adiposity, a well-established risk factor for metabolic syndrome. Moreover, our data revealed that one-third of obese young adults had abdominal obesity, a critical factor in the MetS diagnosis , highlighting the central role of adiposity in abdominal and peripheral fat distribution among obese individual in several populations.[21,22] The significant difference in upper arm circumference further supports the notion that fat accumulation in the upper body is also indicative of overall obesity.

Consistent with these observations, obese participants showed significantly higher body fat percentage and visceral fat levels compared with normoweight individuals. Visceral fat is metabolically active and strongly associated with cardiovascular and metabolic diseases.[13] Other factors besides BMI, such as fat distribution, bone density, age, muscle mass, as well as gender and genetic factors, may also influence overall body composition.[23] More precisely, the proportion of visceral fat is higher in obese individuals, leading to chronic low-grade inflammation, which is a key driver of insulin resistance and endothelial dysfunction.[24] Adipose tissue secretes pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which interfere with insulin signalling pathways, leading to hyperinsulinemia and subsequent glucose intolerance. Additionally, increased free fatty acid flux from visceral fat to the liver promotes hepatic steatosis, contributing to dyslipidemia characterized by elevated triglycerides and reduced HDL-c levels.[25] These mechanisms are consistent with the lipid profile alterations observed in the obese participants in this study.

Analysis of lipid profiles showed that obese individuals exhibited lower HDL-c levels and higher LDL-c and triglyceride (TAG). Moreover, based on NCEP/ATP III criteria, hypertriglyceridemia and low HDL-c levels were more prevalent among obese young adults in this study. These findings indicate that young individuals with obesity are at increased risk of developing CVD, influenced by both central adiposity and dyslipidemia.

Interestingly, although obese participants demonstrated significantly higher blood pressure levels, fasting blood glucose levels did not differ between groups. This finding may reflect the relatively early stage of metabolic changes in this young population. However, the elevated visceral fat in obese participants indicates a latent risk for future glucose dysregulation, as visceral fat is a well-known contributor to insulin resistance and impaired glucose metabolism.[26] Furthermore, a widespread increase in subcutaneous fat highlights the systemic nature of fat accumulation in obesity, emphasizing the importance of addressing overall body fat reduction, not only visceral fat, to improve health outcomes in obese individuals.[27] The increase in subcutaneous adipose tissue, especially in the trunk area, reflects greater fat accumulation in central areas, thereby contributing to the elevated metabolic risk observed in the obese group.

The findings of this study also suggest the presence of sarcopenic obesity, as muscle mass in the trunk and arms tended to be lower among obese participants compared with the normoweight group. Sarcopenic obesity is characterized by excess adiposity accompanied by reduced muscle mass, which may impair physical function and metabolic regulation.[28] Reduced skeletal muscle mass can limit glucose uptake, thereby exacerbating insulin resistance and metabolic dysfunction. These results highlight the importance of interventions that promote both fat reduction and preservation of muscle mass in young adults.

Our study emphasizes the significant presence of MetS components in young obese adults, particularly abdominal obesity, hypertriglyceridemia, and low HDL-c levels. Atherogenic dyslipidaemia, especially low HDL-c levels, was the most common MetS

component observed, emphasizing the important of lipid monitoring in young adults with obesity. [29]

The increasing prevalence of abdominal obesity and dyslipidemia in young populations is concerning because it signals an elevated risk of cardiovascular and metabolic diseases earlier in life.[30] This trend may be linked to modern lifestyle factors such as sedentary behavior and dietary patterns high in processed foods and sugars.[6]

In multivariable analysis, family history of hypertension emerged as the only significant demographic predictor of obesity. This result reinforces the role of genetic predisposition and shared familial lifestyle patterns in obesity development. Several mechanisms may underlie this association, including inherited cardiometabolic susceptibility, early-life metabolic imprinting, and household behavioral clustering such as dietary habits, sodium intake, and physical inactivity.[17,18] Interestingly, variables that differed in the unadjusted analysis, such as HDL-c, blood pressure, and smoking remain insignificant after adjustment. This may reflect overlapping and interrelated metabolic mechanisms, in which lipid metabolism, central adiposity, and inflammatory pathways interact closely and attenuate individual effects in adjusted models.[13,25,30] These findings highlight the importance of considering familial cardiovascular risk when evaluating early determinants of obesity and underscore the need for preventive strategies among young adults with a family history of hypertension.

Furthermore, elevated triglyceride levels is the only independent metabolic predictor of obesity. Individuals with hypertriglyceridemia had nearly fivefold higher odds of being obese. This finding aligns with the established pathophysiological links between dyslipidemia, visceral adiposity, and insulin resistance. Elevated triglycerides reflect increased free fatty acid flux, hepatic very-low-density lipoprotein (VLDL) overproduction, and early metabolic inflexibility.[13,25,30] These results highlight triglycerides as a potential early metabolic marker associated with obesity in young adults.

This study has several limitations. First, the cross-sectional design prevents causal inference. Second, potential confounding variables such as dietary intake, physical activity, and socioeconomic factors were not included in the analysis. These factors may influence metabolic health and obesity risk..

In conclusion, young adults with obesity already demonstrate early metabolic abnormalities, particularly abdominal adiposity, low HDL-c, and elevated triglycerides. Elevated triglycerides and family history of hypertension have been identified as important predictors of obesity, indicating that metabolic risk begins to developat a young age. These findings emphasize the need for early screening, including lipid profiling and assessment of familial cardiovascular risk, to prevent long-term cardiometabolic complications. Future studies should incorporate additional lifestyle and genetic factors to better understand the development of metabolic syndrome in young adults. Early interventions focusing on weight management, lifestyle modification, and targeted health education are essential to reduce the long-term burden of cardiometabolic disease.

Authors' Contributions

MP contributed to conceptualization and design study, methodology development, investigation, supervision, validation, and both drafting and critical revision of the manuscript. ERI and YFHH were responsible for data curation, project administration, and preparation of the original draft. RSP contributed to methodology, formal analysis, data curation, visualization, and participated in both drafting and revising the manuscript.

Conflict of Interest

The authors declare no conflict of interest.

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Generative AI Disclosure Statement

During the preparation of this work, the authors used ChatGPT to assist with paraphrasing. The authors subsequently reviewed and edited the generated content as necessary and assume full responsibility for the final content of the publication.

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