

Elevated Carotid Intima-Media Thickness in Obesity is Unassociated with other Indices Used to Assess Cardiometabolic Risks

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ABSTRACT

This study set out to investigate carotid intima-media thickness (CIMT) and other cardiometabolic indices within those elevated body mass index, compare to controls, and evaluate the relationships between CIMT and other indices in patients with obesity.

The present cross-sectional study enrolled patients diagnosed with obesity between September 2019 and January 2022 and a control group without obesity. A broad range of metabolic markers were measured, including fasting glucose, HbA1c, serum fasting insulin, HOMA-IR index, used to estimate insulin resistance; blood lipid parameters; C-reactive protein (CRP); and thyroid function tests. Several cardiovascular risk indices were calculated: atherogenic index of plasma (AIP), visceral adiposity index (VAI), lipid accumulation product (LAP), and triglyceride-glucose index (TyG index). CIMT was measured following standardized protocols.

Obese and control groups included 107 and 108 participants, respectively. Patients with obesity were significantly older (39 [29-43] vs. 33.5 [28-40], $p = 0.040$), while sex distribution did not differ ($p = 0.089$). Anthropometric results aligned with anticipated differences between the obese and normoweight groups. Cardiometabolic parameters, including glucose ($p = 0.012$), HbA1c, insulin, HOMA-IR, triglycerides, CRP, CIMT, AIP, VAI, LAP, and TyG index, were found to be substantially elevated in the obesity group ($p < 0.001$). CIMT had weak correlations with age ($r = 0.362$, $p < 0.001$), total cholesterol ($r = 0.206$, $p = 0.034$), and low-density lipoprotein cholesterol ($r = 0.293$, $p = 0.002$).

Obese individuals had significantly higher CIMT, AIP, VAI, LAP, and TyG index values, but no statistically significant correlations were detected between CIMT and cardiometabolic indices.

Keywords: Carotid Intima-Media Thickness, Obesity, Cardiometabolic Risk Factors, Atherogenic Index of Plasma, Triglyceride-Glucose Index

Introduction

Defined by an abnormal increase in body fat, obesity represents a persistent and multifactorial health issue (1). It affects more than 890 million adults globally (~13%) (1). Obesity rates have surged nearly three times since 1975, with projections indicating that about 1.02 billion

adults (roughly 18%) will be living with obesity by 2030 (2).

Obesity contributes notably to the development of several metabolic and cardiovascular disorders, including osteoarthritis, obstructive sleep apnea, non-alcoholic fatty liver disease, polycystic ovary syndrome, diabetes mellitus, hypertension, dyslipidemia, insulin resistance, and atherosclerotic cardiovascular diseases (CVD) (3).

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Obesity contributes to endothelial dysfunction and chronic inflammation within the endothelium, thereby promoting the development of atherosclerosis, which is a major underlying mechanism in CVD pathogenesis (4-6).

Carotid intima-media thickness (CIMT) was largely identified as a reliable non-interventional marker for assessing subclinical atherosclerosis (7). The association between elevated CIMT and CVD risk has established CIMT as a useful predictor in clinical settings (8,9). Several other biomarkers and indices have also been explored for risk stratification and appear to exhibit reliable risk assessment, comprising the atherogenic index of plasma (AIP), visceral adiposity index (VAI), lipid accumulation product (LAP), and triglyceride-glucose (TyG) index (10-12). The strength of these parameters come from their ease of access and availability, as they are calculated based on anthropometric and metabolic parameters (10-13). Investigating the differences in these indices among obese patients and their relationship with CIMT may allow the assessment of the accuracy and utility of these markers in the context of cardiovascular risks. Furthermore, understanding these associations could enhance risk stratification and guide preventive strategies in clinical practice.

Despite their potential significance, there is limited research exploring the level of agreement between CIMT measurements and these metabolic indices in obese individuals. Many previous studies have lacked control groups and included small sample sizes, limiting the generalizability of their findings. Our study aims to compare CIMT, AIP, VAI, LAP, and TyG index between obese patients and a control group, while also evaluating the correlations between CIMT and these metabolic indices in obese individuals.

Material and Methods

Ethical Issues: Ethical clearance was granted by the Institutional Review Board of Necmettin Erbakan University, Meram Faculty of Medicine (Decision date 20.09.2024, decision no: 2024/5203), and the research was carried out in line with the principles of the Declaration of Helsinki.

Study Design, Setting, and Participants: The current retrospective cohort was designed at the Department of Endocrinology. The study included patients classified as obese based on clinical criteria who were monitored and treated in accordance with standard clinical protocols during

the timeframe of September 2019 through January 2022. The control group included participants who had not been diagnosed with obesity. The threshold for obesity, as outlined by WHO, was set at a BMI of 30 kg/m² or greater (1).

Participants under the age of 18 or over 65, as well as those with a known history of diabetes mellitus, hypertension, chronic kidney or liver disease, malignancy, or pregnancy were excluded from the study. The control group was selected using a random sampling method to ensure a comparable number of participants to the obesity group. Selection was performed through SPSS software.

Data Collection: The study utilized retrospective data obtained from electronic health records and patient files. Demographic, anthropometric, metabolic, and biochemical parameters were recorded from hospital records. Smoking status was determined as recorded in the medical files through patient-reported smoking status.

Anthropometric Measurements: Anthropometric data collection included height and weight measurements, BMI calculation, and the evaluation of waist and hip circumferences along with the waist-to-hip ratio (WHR). Participant height was measured using a standard stadiometer, while body weight was recorded with a digitally calibrated scale to ensure accuracy. BMI was derived by dividing an individual's weight in kilograms by the square of their height in meters. The measurement of waist circumference was taken at halfway point between the bottom edge of the rib cage and the top of the hip bone (iliac crest), whereas hip circumference was determined at the widest part of the hips. Participants were classified as having either normal or elevated WHR according to WHO standards (>0.85 for females, >0.90 for males) (14).

Metabolic and Biochemical Assessments:

Laboratory analyses were performed at the central biochemistry laboratory of our center, which is accredited and regularly participates in external quality control programs. The laboratory operates with certified, calibrated analyzers to ensure accurate and reliable measurements. Fasting glucose was measured using the glucose oxidase method, while glycated hemoglobin (HbA1c) was analyzed by high-performance liquid chromatography. Fasting insulin was measured using a chemiluminescent immunoassay technique, and insulin resistance was estimated using the HOMA-IR formula, which involves multiplying fasting glucose (mg/dL) by fasting insulin (μU/mL) and dividing the result by 405 (15). Cholesterol and triglyceride levels, including

HDL-C, were measured through enzymatic colorimetric assays; LDL-C was estimated using the widely accepted Friedewald equation. Thyroid-stimulating hormone and free thyroxine levels were assessed via electrochemiluminescence immunoassay.

Cardiovascular Risk Indices: Cardiovascular risk indices were calculated using validated formulas based on the collected anthropometric and biochemical data. The AIP was computed using the formula: $AIP = \log [Triglycerides (mg/dL) / HDL-C (mg/dL)]$ (16). The VAI was determined using sex-specific equations: For males: $VAI = [Waist\ circumference\ (cm) / (39.68 + (1.88 \times BMI))] \times [Triglycerides\ (mmol/L) / 1.03] \times [1.31 / HDL-C\ (mmol/L)]$, for females: $VAI = [Waist\ circumference\ (cm) / (36.58 + (1.89 \times BMI))] \times [Triglycerides\ (mmol/L) / 0.81] \times [1.52 / HDL-C\ (mmol/L)]$ (17). To calculate the LAP, the formula $[waist\ circumference\ (cm) - 65] \times triglycerides\ (mmol/L)$ was used for males, and $[waist\ circumference\ (cm) - 58] \times triglycerides\ (mmol/L)$ for females (13). The TyG index was obtained using the expression $\log [fasting\ triglyceride\ (mg/dL) \times fasting\ glucose\ (mg/dL) / 2]$ (18).

Carotid Intima-Media Thickness: The CIMT values were measured using high-resolution B-mode ultrasonography following standardized protocols (7). Measurements were performed with patients in the resting on their back, the patients' heads were mildly extended and turned to the opposite side. CIMT was assessed on longitudinal images obtained from the posterior wall of the distal 1 cm segment of the common carotid artery. Measurements were taken synchronized with the electrocardiographic R wave, and the mean of at least three consecutive measurements was used for each patient. A Siemens Healthineers Acuson Juniper ultrasound system with a 12-L3 MHz linear probe was used for all measurements, which were performed by the same skilled radiologist/operator. To minimize intra-observer and inter-observer variability, repeated measurements were conducted by a single observer, and the averages were recorded.

Statistical Analysis: All data were systematically reviewed and verified before analyses, which were conducted using SPSS v25 (IBM, NY, USA). Normality assumption was evaluated using the histograms and Q-Q plots. Descriptive statistics were presented using mean and standard deviation or as median along with the interquartile range (IQR: 25th to 75th percentile) based on normality of distribution. Categorical data were summarized using counts and percentages within each study

group. Depending on the distribution characteristics, continuous variables were analyzed between groups with the Student's t-test or Mann-Whitney U test, while the chi-square test was applied to assess associations between categorical variables. Relationships between CIMT and relevant variables were evaluated using the Pearson correlation coefficient when both variables were normally distributed continuous variables or Spearman correlation coefficient when at least one of the continuous variables were not normally distributed or point biserial correlation coefficient when we evaluate one normally distributed continuous variable and one dichotomous variable. Results with p-values under 0.05 were interpreted as statistically significant. Statistical analysis methods were reviewed and validated by a qualified biostatistician to ensure appropriate test selection and interpretation.

Results

The obese group consisted of 107 participants, while the control group had 108 participants. The average age of the obese group was 39 (29 - 43), whereas the median age of the control group was 33.5 (28 - 40) years, with this difference being statistically significant ($p = 0.040$). With regard to sex, 72.90% of the obese group were female, while in the control group, 62.04% were female. However, this difference was not statistically significant ($p = 0.089$).

Compared to their non-obese counterparts, individuals in the obese group had significantly lower height ($p = 0.009$), alongside greater body weight and BMI values (both $p < 0.001$). Waist and hip circumferences, as well as waist-to-hip ratio (WHR), were also markedly elevated in the obese group (all $p < 0.001$).

Regarding metabolic parameters, those with obesity displayed significantly higher levels of glucose ($p = 0.012$), HbA1c ($p < 0.001$), insulin ($p < 0.001$), and HOMA-IR ($p < 0.001$). Additionally, they had elevated levels of triglycerides, CRP, and CIMT (all $p < 0.001$). Scores for AIP, VAI, LAP, and the TyG index were also significantly greater in this group (all $p < 0.001$) (Table 1).

Examination of internal relationships between numerical variables in the obesity group indicated a positive correlation between age and CIMT ($r = 0.362$, $p < 0.001$). Despite detecting significant correlations between CIMT and total cholesterol levels ($r = 0.206$, $p = 0.034$) and LDL-C ($r = 0.293$, $p = 0.002$), we did not find any statistically significant

Table 1: Summary of Demographics and Laboratory Measurements With Regard To Groups

	Groups		
	Obese (n=107)	Control (n=108)	p
Age	39 (29 - 43)	33.5 (28 - 40)	0.040‡
Sex			
Female	78 (72.90%)	67 (62.04%)	0.089§
Male	29 (27.10%)	41 (37.96%)	
Height, cm	164.33 ± 10.38	167.96 ± 9.83	0.009†
Weight, kg	100 (87 - 113.3)	72 (65 - 80)	<0.001‡
Body mass index, kg/m ²	36.72 (32.32 - 42.24)	25.87 (23.52 - 27.90)	<0.001‡
Waist circumference, cm	107.19 ± 14.26	83.62 ± 10.43	<0.001†
Hip circumference, cm	122.62 ± 14.26	100.07 ± 7.63	<0.001†
Waist to hip ratio	0.88 ± 0.08	0.83 ± 0.08	<0.001†
Normal	51 (48.11%)	75 (70.09%)	0.001§
High	55 (51.89%)	32 (29.91%)	
Smoking	22 (20.56%)	26 (24.07%)	0.649§
Glucose, mg/dL	96.32 ± 11.06	92.86 ± 8.81	0.012†
HbA1c, %	5.46 ± 0.37	5.24 ± 0.45	<0.001†
Insulin, µU/mL	14.93 (10.80 - 21.64)	9.81 (7.59 - 13.88)	<0.001‡
HOMA-IR	3.53 (2.52 - 5.51)	2.28 (1.73 - 3.30)	<0.001‡
Total cholesterol, mg/dL	183.96 ± 30.72	175.52 ± 33.09	0.054†
HDL, mg/dL	45.63 ± 10.56	50.06 ± 11.42	0.003†
LDL, mg/dL	108.12 ± 27.74	102.72 ± 28.99	0.166†
Triglyceride, mg/dL	133.8 (96 - 200)	105.5 (75.65 - 157.9)	<0.001‡
TSH, µIU/mL	1.96 (1.21 - 2.70)	1.75 (1.20 - 2.56)	0.597‡
T4, ng/dL	1.20 (1.05 - 1.32)	1.26 (1.11 - 1.42)	0.084‡
CRP, mg/L	3.57 (2.18 - 7.39)	1.20 (0.57 - 2.36)	<0.001‡
Carotid intima media thickness, mm	0.06 ± 0.01	0.05 ± 0.01	<0.001†
Atherogenic index of plasma	0.15 ± 0.25	-0.02 ± 0.28	<0.001†
Visceral adiposity index	2.22 (1.51 - 3.61)	1.48 (0.83 - 2.48)	<0.001‡
Lipid accumulation product	79.10 (49.19 - 109.24)	27.71 (13.52 - 46.29)	<0.001‡
TyG index	8.83 ± 0.51	8.50 ± 0.53	<0.001†

Continuous variables with normal distribution are expressed as mean ± standard deviation, while those not normally distributed are presented as median (interquartile range: 25th–75th percentile). Categorical variables are summarized using frequencies and percentages.

† Student's t test, ‡ Mann Whitney U test, § Chi-square test. Statistically significant p values are shown in bold.

Abbreviations: CRP: C-Reactive Protein, HbA1c: Hemoglobin A1c, HDL: High-Density Lipoprotein, HOMA-IR: Homeostatic Model Assessment of Insulin Resistance, LDL: Low-Density Lipoprotein, TSH: Thyroid-Stimulating Hormone, T4: Thyroxine, TyG Index: Triglyceride-Glucose Index

relationships between CIMT and the examined indices (Table 2).

Discussion

The data obtained in the current study revealed that obese individuals exhibited superior levels of blood glucose, HbA1c, insulin, triglycerides, CRP, CIMT measurements, LAP, TyG, AIP, VAI, and HOMA-IR scores, along with lower HDL-C levels, compared to

non-obese individuals. CIMT was not found to have any notable relationships with several indices used to assess cardiovascular risks, including AIP, VAI, LAP, and TyG.

Obesity has been a widely recognized contributing factor to both metabolic, cardiac and circulatory conditions due to its complex pathophysiological effects on multiple organ systems (3). One of the primary mechanisms linking obesity to these risks is chronic low-grade inflammation driven by excessive

Table 2: Correlations Between Carotid Intima Media Thickness and Other Variables

	r	p
Age	0.362‡	<0.001
Sex, Male	0.068§	0.485
Height, cm	0.042†	0.668
Weight, kg	0.136‡	0.162
Body mass index, kg/m2	0.124‡	0.205
Waist circumference, cm	0.100†	0.307
Hip circumference, cm	0.007†	0.946
Waist to hip ratio	0.145†	0.138
Smoking, Yes	-0.024§	0.810
Glucose	0.141†	0.148
HbA1c	0.137†	0.167
Insulin	-0.058‡	0.556
HOMA-IR	-0.031‡	0.755
Total cholesterol	0.206†	0.034
HDL	-0.037†	0.706
LDL	0.293†	0.002
Triglyceride	0.130‡	0.183
TSH	0.109‡	0.263
T4	-0.018‡	0.857
CRP	0.082‡	0.425
Atherogenic index of plasma	0.075†	0.440
Visceral adiposity index	0.085‡	0.384
Lipid accumulation product	0.180‡	0.065
TyG index	0.100†	0.304

† Pearson correlation coefficient, ‡ Spearman correlation coefficient, § Point biserial correlation coefficient. Statistically significant p values are shown in bold.

Abbreviations: CRP: C-Reactive Protein, HbA1c: Hemoglobin A1c, HDL: High-Density Lipoprotein, HOMA-IR: Homeostatic Model Assessment of Insulin Resistance, LDL: Low-Density Lipoprotein, r: Correlation coefficient, TSH: Thyroid-Stimulating Hormone, T4: Thyroxine, TyG Index: Triglyceride-Glucose Index

adipose tissue (19,20). Among adipose tissues, visceral fat exhibits endocrine activity by releasing a range pro-inflammatory cytokines and acute-phase proteins, such as TNF- α , IL-6, and CRP. This inflammatory state plays a critical contribution to promoting endothelial dysfunction, oxidative stress, that are key contributors to atherosclerosis and increased cardiovascular risk (19-21). Beyond inflammation, obesity disrupts glucose and lipid metabolism. Increased adiposity leads to insulin resistance by impairing insulin signaling pathways, resulting in elevated glucose levels, compensatory hyperinsulinemia, and higher HbA1c values over time. Lipid metabolism dysregulation causes elevated triglyceride levels and altered lipid transport, further predisposing individuals to metabolic syndrome and CVD (22,23). The present study demonstrated that obese patients exhibited unfavorable alterations in inflammatory markers (such as CRP), metabolic

parameters, lipid markers (such as HDL-C and triglycerides), and cardiometabolic indices (AIP, VAI, LAP, and TyG). These results align with those reported in earlier studies (24-28). In particular, alterations in cardiometabolic indices may serve as early indicators of increased cardiovascular risk in obese individuals; however, evidence from this study points out these relationships might not be clearly linked to the results of CIMT measurement.

It has been established that metabolic disturbances are accompanied by structural and functional changes in the vasculature, including increased CIMT, which serves serving as a preliminary indicator of atherosclerotic changes (7,29). CIMT reflects the combined thickness of the intima and media layers of the carotid artery and is widely recognized as a non-invasive indicator of subclinical vascular damage that allows risk assessment before the manifestation of adverse cardiovascular outcomes (7,30). In this study,

we focused on CIMT because it offers a direct assessment of vascular health and is influenced by multiple the metabolic dysregulation and chronic low-grade inflammation linked to obesity. Increased CIMT has been implicated in the development of insulin resistance, dyslipidemia, low-grade inflammatory activity and vascular endothelial damage, both of which play pivotal roles in the progression of atherosclerosis (31-33). By investigating the relationship between CIMT and various cardiometabolic indices, we aimed to identify potential contributors to early atherosclerosis in obesity and assess whether novel metabolic markers, such as the TyG index, AIP, VAI, and LAP, are associated with vascular impairment. In the presented study, obese patients showed markedly elevated CIMT levels; however, no strong correlation was observed between CIMT and other metabolic parameters, similar to prior literature (29).

In a comprehensive study with a cross-sectional design, the authors founded a marked association between the frequency of carotid atherosclerosis and TyG-waist circumference, TyG-waist-to-height ratio, Chinese LAP, and VAI in normoweight. Nevertheless, analysis revealed no notable relationship between the occurrence of carotid atherosclerosis and TyG, TyG-BMI, or VAI values. (34), agreeing with our data. Kotsis et al. reported that CIMT increases with rising BMI. Overweight individuals in their study exhibited significantly higher CIMT values than their normoweight counterparts, and multivariable regression in obese individuals revealed an independent association between fasting blood glucose levels and CIMT (35). It has also been demonstrated that weight loss can yield CIMT decline (29). In the study by Rychter et al., specific micronutrients, including folate, folic acid, vitamins K, B12, D, and magnesium, were found to have beneficial effects on CIMT, suggesting their potential role in reducing atherosclerosis risk. The authors also found that specific diets and bariatric surgery (potentially in relation with weight loss), could effectively reduce CIMT and atherosclerosis risk in obese patients (30). A meta-analysis reported an average weight loss of 23.26 kg and an average CIMT reduction of 0.06 mm in such patients (7). Even in studies where CIMT appears to be similar in obese and normoweight individuals, correlation analyses still show that CIMT is positively correlated with age and BMI. These findings suggest that CIMT is more closely associated with age and BMI relative to metabolic phenotype (36), which provides an explanation for the lack of directional relationships with other cardiometabolic risk indices in our study.

In a broad study similarly designed to ours, Bažadona et al. examined the relationships between obesity, CIMT, and conventional cardiovascular risk factors. It found significant associations between CIMT and chronological age, vascular age, systolic blood pressure, fasting glucose, total cholesterol, triglyceride levels, WHR, waist circumference, BMI, and skeletal muscle mass index in obese individuals (31). A positive correlation was observed between CIMT and both atherosclerotic cardiovascular disease (ASCVD) risk assessment and SCORE (Systematic Coronary Risk Evaluation), although the correlations were weak to moderate (31). Similar to the conclusion of these authors, we recommend further research to validate risk indices in terms of their real-world outputs for cardiovascular risk prediction in obese individuals. It is important to note that the absence of statistically significant correlations in our study does not definitively exclude the possibility of clinically meaningful relationships between CIMT and metabolic indices. Larger studies with greater statistical power may be needed to detect smaller effect sizes that could still be clinically relevant. Also, it may be prudent to assess whether these markers / indices can be utilized in the general population, as potential relationships without the confounding impacts of obesity could be crucial to understand the roles of these markers in disease prevention or early diagnosis. There are very few such studies, one of which reported no marked relationship between the TyG index and CIMT in non-obese patients (37). Despite the strong relationship between age and CIMT that has been established by multiple studies, it is crucial to note that studies involving obese children also detect elevated CIMT results relative to normoweight children (32). The similarities also extended to other analyses, including the fact that CIMT correlated with anthropometric measures (waist circumference, WHR) and was unassociated with insulin resistance (HOMA-IR) –despite being associated with elevated uric acid, LDL-C, triglyceride levels, and decreased HDL-C and vitamin D levels (32). Contrasting results concerning the relationships exist, for instance one pediatric study identified that CIMT was markedly correlated with HOMA-IR and endocan (the latter used as a measure of endothelial dysfunction) (33). Taken together, it is evident that age and obesity have strong impacts on CIMT which might be marginally reflected by some metabolic measures, but it also appears that the extent of these effects cannot be fully captured by other cardiovascular health-related parameters.

In our study, we observed significantly higher CIMT values in obese individuals; however, no strong correlation was found between CIMT and other

metabolic parameters. This finding suggests that the effect of obesity on CIMT is complex and multifactorial. Compared to other studies, our results indicate that CIMT is associated with traditional risk factors such as age, BMI, blood pressure, and lipid parameters but does not show a consistent relationship with novel cardiometabolic indices (TyG, AIP, VAI, LAP).

A few important limitations must be considered when interpreting these findings.. First, as a single-center study, its generalizability is limited. Secondly, being a retrospectively conducted study limits the capacity to draw causal inferences. Third, since only obese patients were included, the findings may not be generalizable to broader populations. Additionally, atherosclerosis risk assessment was based solely on CIMT measurements without the use of other imaging modalities such as ultrasound. Factors including dietary habits and menopausal status were not accounted for, that may have influenced the findings. Moreover, changes in CIMT and other metabolic parameters after obesity treatment were not evaluated, limiting the ability to assess longitudinal effects. Lastly, participants in the control group were chosen through random selection, and although significant age-related variation and anthropometric measurements were found between groups, this may have affected between-group comparisons. The narrow range of CIMT values prevented regression analysis, limiting the capacity to assess unique role of unmatched factors.

Our study demonstrated that obese individuals have significantly higher CIMT, AIP, VAI, LAP and TyG index values. However, no statistically significant correlations were detected between CIMT and these cardiometabolic indices, suggesting that its relationship with metabolic parameters is complex and multifactorial or that these markers cannot reflect the endothelial status. CIMT is a well-recognized tool for assessing cardiovascular risk, there may still be value in assessing whether other metabolic / cardiovascular markers can support the data obtained from CIMT measurement. Additional research is required to elucidate the pathways connecting obesity with metabolic dysfunction and vascular damage.

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