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The impact of obesity on metabolic and cardiovascular health: A morphometric retrospective cohort study

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Ethics Committee Approval
The study was approved by the Hamidiye Scientific Research Ethics Committee of the University of Health Sciences (Decision No: 2022/23-6) on October 14, 2022.
All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.

Conflict of Interest
No conflict of interest was declared by the authors.

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Abstract

Background/Aim: Obesity is recognized as a significant risk factor for various diseases, including cardiovascular disease. The link between obesity and adverse health outcomes, particularly cardiovascular ailments, underscores the need for a comprehensive understanding of the associated metabolic and cardiovascular markers. This study aimed to compare individuals with obesity and those without obesity in terms of markers related to metabolism and cardiovascular health.

Methods: 136 participants were enrolled in the analysis, comprising 83 individuals with obesity and 53 individuals without obesity. The participants' demographic data and biochemical test results were collected, including age, sex, fasting glucose, creatinine, cholesterol, triglyceride, and body mass index (BMI). Measurements were taken for various cardiac markers using echocardiographic images. Morphometric parameters of the heart, such as left ventricular (LV) and right ventricular (RV) end-systolic-and-diastolic diameter, ejection fraction, interventricular septum thickness, aortic ascending diameter, and epicardial fat tissue thickness were assessed. Statistical analyses were employed to identify significant differences. Independent-sample t-tests and Pearson correlation tests were used for comparisons between obese and non-obese individuals.

Results: Comparisons between obese and non-obese individuals revealed that individuals with obesity exhibited significantly higher levels of fasting glucose (P=0.021), triglycerides (P=0.014), and epicardial fat tissue thickness (P<0.001). LV ejection fraction was significantly higher in obese individuals than in non-obese participants (P<0.001) but remained within the normal range. Sex-associated differences in metabolic variables of obesity and non-obesity revealed that the obese male individuals had higher fasting glucose (P<0.001) and triglyceride levels (P<0.001) compared to obese female individuals. Moreover, BMI was positively correlated with epicardial fat tissue thickness (r^2=0.29, P<0.001), and triglyceride level was significantly correlated with fasting glucose level (r^2=0.19, P<0.001).

Conclusion: The study design allowed for a comparison between obese and non-obese individuals, providing valuable insights into the differences in these markers based on obesity status. The investigation of individuals with elevated BMI levels highlights significant deviations in crucial indicators compared to those with normal levels. These findings emphasize the urgent need to address obesity as a central contributor to the development of diverse diseases and advocate for proactive strategies aimed at mitigating associated health risks.

Keywords: obesity, metabolic profile, morphometric parameters, heart

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Introduction

Obesity, characterized by excessive accumulation of adipose tissue, has become a significant global health concern, predisposing individuals to various adverse health outcomes, particularly metabolic disturbances and cardiovascular disease [1]. Changes brought on by obesity can put the body at risk for alterations in heart shape and function, leading to cardiovascular disease [2]. While these changes may be less pronounced in overweight or moderately obese adults, they are most noticeable in highly obese individuals [3]. Experimental research suggests that metabolic anomalies may play a role in the heart structure and function changes associated with obesity [4]. Postmortem studies have provided information on the anatomy of heart chambers in highly obese individuals, showing increased epicardial fat tissue and thickened left and right ventricular walls [4]. Echocardiographic investigations can help determine how obesity impacts heart morphology [5]. However, these parameters alone do not fully explain the modifications in heart morphology related to obesity, especially those concerning left ventricle (LV) geometry. Speculation suggests that metabolic issues linked to obesity may contribute to altered heart shape in humans, based on experimental studies [6]. Previous research has highlighted the duration of obesity as a factor influencing LV morphology, demonstrating a direct correlation between LV diastolic chamber size and wall thickness with the duration of obesity [7].

One study found that only 6% of normal weight patients and 34% of obese patients had enlarged left atriums [8]. Studies have shown a significant correlation between body mass index (BMI) and left atrial dimensions measured in anteroposterior or longitudinal views [9]. LV diastolic dysfunction is thought to contribute to left atrium enlargement in obese individuals. The prevalence of left atrial enlargement in obesity may be underestimated by measuring the left atrial dimension [9].

Understanding the complex interactions between morphometric factors and metabolic profiles in obesity is crucial due to its widespread prevalence. Morphometric measures, such as BMI, provide insights into adipose tissue distribution and serve as important predictors of health concerns associated with obesity. The literature establishes a relationship between morphometric factors and metabolic health, with increased adiposity significantly increasing the risk of metabolic diseases like insulin resistance, type 2 diabetes, and dyslipidemia [1,10]. Morphometric approaches, particularly BMI, have emerged as non-invasive, affordable tools for estimating body fat [11,12].

This study aims to explore the relationship between morphometric variables and metabolic parameters in the context of obesity. By examining a comprehensive set of morphometric measurements and their associations with metabolic and cardiovascular markers, this research seeks to enhance our understanding of the mechanisms linking obesity, morphometrics, and metabolic outcomes. Investigating these relationships may offer insights for developing targeted interventions to reduce metabolic and cardiovascular disease risks associated with obesity and improve overall health.

Materials and methods

The sample population consisted of 136 participants, with 83 classified as obese (BMI ≥30) and 53 as non-obese. Echocardiographic data were obtained from the archives of Istanbul Health Sciences University Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital between June 2021 and January 2023. BMI was calculated by dividing weight in kilograms by the square of height. Quality control procedures included participant re-examination and blind re-reading to assess intra- and inter-sonographer variability and intra- and inter-reader variability. Metabolic profiles were determined through biochemical analysis, including fasting blood tests for parameters such as hemoglobin, cell count, HDL, LDL, cholesterol, creatinine, and uric acid. The doctor conducting echocardiographic recordings was blinded to the patients' metabolic characteristics. Measurements of LV dimensions in diastole and systole, as well as ventricular septum and posterior wall thickness, were taken.

Statistical analysis

Descriptive statistics, including means and standard deviations, were used to summarize participant characteristics by adiposity level. Pearson correlation analysis was used to assess differences in continuous and categorical characteristics. Normal distribution of data was checked, and two independent-sample t-tests were conducted for characteristics, adjusting for sex. The alpha level for significance tests was set at 0.05, and all analyses were performed using SPSS version 21.1 software.

Results

Clinical characteristics of obese and non-obese individuals are presented in Table 1. Apart from fasting glucose (P=0.021) and triglyceride levels, which were slightly higher in obese individuals compared to non-obese individuals (P=0.014), most of the biochemical variables in obese individuals were within normal ranges.

Table 1: Comparison of obese and non-obese in metabolic variables of obesity

Variable	Obese individuals (n=83) Mean (SD)	Non-obese individuals (n=53) Mean (SD)	*P*-value
Age (years)	45.0 (17.0)	48.9 (15.9)	0.588
Gender (Male/Female)	28 / 55	17 / 36	0.688
BMI (kg/m^2)	45.4 (11.1)	25.1 (3.1)	<0.001**
Fasting glucose (mg/dL)	117.4 (39.4)	102.5 (18.8)	0.021*
Creatinine (µmol/L)	0.9 (0.3)	0.9 (0.5)	0.122
eGFR (mL/min/1.73m^2)	90.7 (20.7)	94.2 (25.6)	0.190
Urea (mmol/L)	34.4 (22.1)	37.2 (31.4)	0.241
Uric acid (mg/dL)	6.2 (4.7)	5.0 (1.8)	0.381
CRP (mg/L)	6.3 (8.1)	8.2 (21.1)	0.069
HDL cholesterol (mg/dL)	47.9 (14.0)	53.4 (13.9)	0.836
LDL cholesterol (mg/dL)	128.6 (37.0)	132.8 (5.2)	0.753
Triglyceride (mg/dL)	175.9 (110.7)	134.4 (61.1)	0.014*
HGB (g/dl)	14.5 (7.8)	12.6 (2.1)	0.259
TSH (mIU/L)	2.2 (1.9)	2.0 (1.0)	0.184
Serbest T4 (pmol/L)	16.3 (2.21)	16.5 (2.4)	0.941
AST (U/L)	20.3 (10.4)	17.6 (7.7)	0.381
ALT (U/L)	25.1 (20.9)	16.1 (7.4)	0.002‡

SD: Standard deviation, BMI: Body mass index, eGFR: Estimated Glomerular Filtration Rate, CRP: C-reactive protein, HDL: high-density lipoprotein, LDL: low-density lipoprotein, HGB: Hemoglobin, TSH: thyroid stimulating hormone, AST: aspartate aminotransferase, ALT: alanine transaminase. *P<0.05, **P<0.001, ‡within normal value.

The echocardiographic features of the study population are outlined in Table 2. The thickness of epicardial fat tissue was greater in obese individuals than in non-obese participants (P<0.001). Obese individuals also had a higher LV ejection fraction than non-obese individuals (P<0.001), but it remained

within normal limits. Obese individuals exhibited a higher atrial diastolic filling wave velocity in terms of functional parameters (P=0.025). Triglyceride levels in obese individuals were positively correlated with fasting blood sugar levels (r2=0.19, P<0.001), and epicardial fat tissue thickness was strongly correlated with BMI (r2=0.29, P<0.001) (Table 3) (Figure 1).

Table 2: Comparison of obese and non-obese in cardiac variables of obesity

Variable	Obese individuals (n=83) Mean (SD)	Non-obese individuals (n=53) Mean (SD)	*P*-value
Left ventricular-end-systolic diameter (mm)	28.1 (8.7)	30.9 (14.5)	0.019‡
Left ventricular-end-diastolic diameter (mm)	48.1 (6.2)	49.7 (10.3)	0.026‡
Left ventricular posterior wall thickness (mm)	10.4 (2.1)	10.1 (1.3)	0.285
Interventricular septum thickness (mm)	10.3 (2.2)	10.1 (1.6)	0.845
Left atrium diameter (mm)	34.8 (7.1)	37.3 (7.9)	0.514
Right atrium-end-diastolic diameter (mm)	31.4 (4.8)	32.7 (4.9)	0.961
Right ventricular-end-diastolic diameter (mm)	29.8 (4.6)	29.3 (5.0)	0.678
Aortic ascending diameter (mm)	33.1 (3.9)	32.5 (4.8)	0.167
Aortic root diameter (mm)	20.3 (2.1)	20.4 (2.6)	0.079
ECG heart rate	77.8 (14.3)	72.9 (10.5)	0.094
Systolic blood pressure (mm Hg)	133.3 (20.6)	122.9 (19.7)	0.785
Diastolic blood pressure (mm Hg)	79.7 (12.2)	76.6 (9.4)	0.207
Ejection fraction (%)	60.0 (8.5)	56.5 (15.0)	<0.001**
Aortic valve flow rate	1.4 (0.2)	1.3 (0.2)	0.997
Pulmonary valve flow rate	1.0 (0.2)	0.9 (0.2)	0.986
Pulmonary arterial pressure (mm Hg)	18.4 (7.1)	22.0 (9.3)	0.091
Epicardial fat tissue thickness (mm)	6.8 (2.2)	3.6 (1.1)	<0.001**
Mitral A-wave (m/s)	0.7 (0.2)	0.7 (0.2)	0.025*
Mitral E-wave (m/s)	0.8 (0.2)	0.8 (0.2)	0.838
Mitral E/A-wave ratio	1.3 (0.5)	1.2 (0.4)	0.681

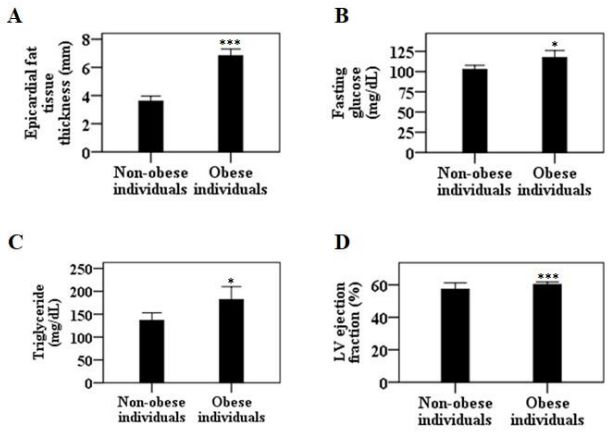
SD: Standard deviation, ECG: Electrocardiogram. *P<0.05, **P<0.001, ‡within normal value.

Table 3: Correlation (r^2-value) of BMI with cardiovascular indicators, and fasting glucose level with triglyceride in obesity.

Variable		r^2	*P*-value
Epicardial fat tissue thickness	BMI	0.29	<0.001**
Fasting glucose level	triglyceride	0.19	<0.001**

BMI: Body mass index. **P<0.001.

Figure 1: Metabolic and cardiac parameters of obese and non-obese individuals were compared. Significant findings were observed in epicardial fat tissue thickness (A), fasting glucose level (B), triglyceride level (C), and LV ejection fraction (D) in obese individuals compared to non-obese individuals.



The data are presented as the mean (SD). **P<0.01, ***P<0.001.

Sex-related differences in metabolic factors between obese and non-obese individuals are displayed in Table 4. Fasting glucose (P<0.001) and triglyceride (P<0.001) levels were higher in obese males compared to obese females.

Sex-related differences in cardiac variables of obese individuals are depicted in Table 5. The interventricular septum thickness (P=0.004), left ventricular end-diastolic diameter (P=0.002), left ventricular end-systolic diameter (P=0.002), left atrial diameter (P=0.022), and aortic root diameter (P=0.030) were all higher in obese men but still within normal ranges. Conversely, obese women had greater left ventricular posterior wall thickness (P<0.001), left ventricle ejection fraction (P=0.013), and mitral E/A-wave ratio values than obese males, but within normal limits (Figure 2, 3).

Table 4: Sex-associated differences in metabolic variables of obesity

Variable	Obese group		
	Male (n=28) Mean (SD)	Female (n=55) Mean (SD)	*P*-value
Fasting glucose (mg/dL)	130.4 (54.6)	110.8 (27.1)	<0.001**
Creatinine (µmol/L)	0.9 (0.2)	0.8 (0.3)	0.213
eGFR (mL/min/1.73m2)	90.1 (17.4)	91.0 (22.4)	0.371
Urea (mmol/L)	38.5 (33.7)	32.3 (12.7)	0.068
Uric acid (mg/dL)	6.5 (1.9)	6.0 (5.6)	0.370
CRP (mg/L)	5.0 (6.0)	6.9 (8.9)	0.039*
HDL cholesterol (mg/dL)	45.4 (16.0)	51.6 (15.0)	0.837
LDL cholesterol (mg/dL)	125.3 (36.1)	135.8 (39.8)	0.488
Triglyceride (mg/dL)	234.6 (155.2)	148.6 (69.0)	<0.001**
HGB (g/dl)	13.8 (3.5)	14.9 (8.4)	0.561
TSH (mIU/L)	1.9 (1.7)	2.3 (2.0)	0.448
Free T4 (pmol/L)	16.3 (2.8)	16.3 (1.9)	0.128
AST (U/L)	24.7 (14.4)	18.0 (6.7)	0.001‡
ALT (U/L)	35.5 (29.6)	19.8 (11.9)	0.003‡

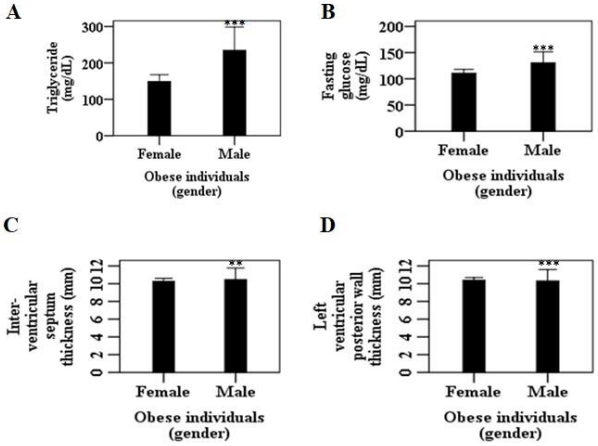
SD: Standard deviation, eGFR: Estimated Glomerular Filtration Rate, CRP: C-reactive protein, HDL: high-density lipoprotein, LDL: low-density lipoprotein, HGB: Hemoglobin, TSH: thyroid stimulating hormone, AST: aspartate aminotransferase, ALT: alanine transaminase. *P<0.05, ***P <0.001, ‡within normal value.

Table 5: Sex-associated differences in cardiac variables of obesity

Variable	Obese group		
	Male (n=28) Mean (SD)	Female (n=55) Mean (SD)	*P*-value
Left ventricular-end-systolic diameter (mm)	32.3 (12.1)	25.9 (5.3)	0.002‡
Left ventricular-end-diastolic diameter (mm)	51.7 (7.6)	46.2 (4.3)	0.004‡
Left ventricular posterior wall thickness (mm)	10.3 (3.3)	10.4 (1.1)	0.000‡
Interventricular septum thickness (mm)	10.5 (3.4)	10.3 (1.3)	0.004‡
Left atrium diameter (mm)	35.0 (10.3)	34.6 (4.9)	0.022‡
Right atrium-end-diastolic diameter (mm)	30.7 (5.3)	31.7 (4.6)	0.258
Right ventricular-end-diastolic diameter (mm)	29.4 (5.1)	30.0 (4.5)	0.686
Aortic ascending diameter (mm)	33.8 (3.4)	32.8 (4.1)	0.431
Aortic root diameter (mm)	20.4 (4.5)	19.9 (1.9)	0.030‡
ECG heart rate	77.1 (14.4)	78.2 (14.4)	0.478
Systolic blood pressure (mm Hg)	136.4 (21.3)	131.8 (20.2)	0.683
Diastolic blood pressure (mm Hg)	83.0 (12.5)	78.0 (11.9)	0.800
Ejection fraction (%)	58.0 (11.1)	61.4 (2.9)	0.013‡
Aortic valve flow rate	1.4 (0.2)	1.4 (0.2)	0.236
Pulmonary valve flow rate	1.0 (0.2)	1.0 (0.2)	0.393
Pulmonary arterial pressure (mm Hg)	17.6 (5.0)	19.3 (8.0)	0.123
Epicardial fat tissue thickness	8.0 (1.9)	6.3 (2.1)	0.590
Mitral A-wave (m/s)	0.7 (0.2)	0.7 (0.3)	0.023‡
Mitral E-wave (m/s)	0.8 (0.2)	0.8 (0.2)	0.022‡
Mitral E/A-wave ratio	1.3 (0.3)	1.4 (0.6)	0.025‡

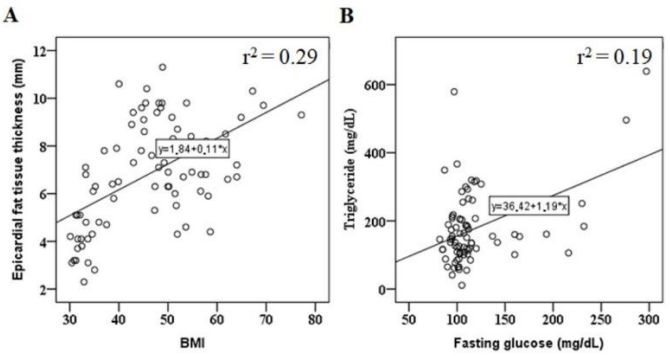
SD: Standard deviation, ECG: Electrocardiogram, ‡within normal value

Figure 2. Metabolic and cardiac parameters of obese men and obese women individuals were compared. Significant findings were observed in triglyceride level (A), fasting glucose level (B), and interventricular septum thickness (C) in obese men compared to obese women individuals. However, a significant difference was observed in obese women compared to obese men in terms of left ventricular posterior wall thickness (D).



The data are presented as the mean (SD). **P<0.01, ***P<0.001.

Figure 3: Correlation analysis on metabolic and cardiac parameters of obese individuals is shown. Significant correlations were observed between BMI and epicardial fat tissue thickness (r^2=0.29, P<0.001) (A), and between fasting glucose and triglyceride (r^2=0.19, P<0.001) (B).



Discussion

Our study found strong evidence emphasizing the importance of addressing obesity as a serious health issue. Obese subjects had higher epicardial fat tissue, consistent with previous studies [2,13]. Increased epicardial fat tissue is linked to serious consequences like coronary inflammation, accelerated atherosclerosis, and fibrosis, as noted by Packer et al. [14]. These findings highlight the significance of elevated epicardial fat tissue as a potential indicator of cardiovascular disease. Additionally, our study showed that obese participants had elevated fasting glucose levels, signaling increased intracellular calcium concentrations and a higher risk of cardiovascular disease [15]. Impaired fasting glucose levels also raise concerns about a higher risk of type 2 diabetes, as the spectrum of obesity-related diseases expands. Furthermore, our results indicated significantly higher levels of triglycerides in the peripheral blood of obese individuals, which are independently associated with increased cardiovascular risk [16].

In our study, significant differences in left ventricular ejection fraction were observed between obese and non-obese individuals, aligning with recent research comparing various measures of LV in these groups [17]. Though our findings revealed differences between obese and non-obese individuals, they were within the normal range.

Gender-related differences play a crucial role in metabolic dynamics and health outcomes. Obese men in our study had higher triglyceride and fasting glucose levels than women, indicating a higher susceptibility to metabolic and cardiovascular diseases. These findings, inconsistent with current research, underscore the importance and reliability of our study [18]. These unique findings emphasize the originality of our study, as these patterns have not been previously documented. The complexity of these gender-based variations in BMI could stem from genetic diversity, dietary habits, physical activity levels, and environmental factors [19,20]. These distinctions also vary based on women's pre-menopausal, pregnant, and post-menopausal statuses. While well-documented, these gender-related differences contribute to the intricate web of metabolic dynamics and health outcomes across diverse populations. Our study highlights BMI as a crucial indicator of metabolic and cardiovascular disease risk due to its role in determining adiposity levels and its strong correlation with fasting glucose and triglyceride levels in obese patients [21].

Our findings emphasize the link between increased BMI and cardiovascular shifts among obese individuals, stressing the importance of weight management in reducing cardiovascular risk [22]. We observed a positive correlation between elevated BMI and epicardial fat tissue thickness, suggesting morphological heart alterations as weight increases in obese individuals. Surprisingly, non-obese individuals had higher LV-end-systolic and end-diastolic diameters than obese individuals, likely due to half of the non-obese group being overweight individuals with slightly higher ages than obese individuals.

Central adiposity assessment in human populations often relies on BMI estimation [23]. Among obese participants, metabolic assessments revealed significant correlations between fasting glucose and triglyceride levels [21], indicating BMI's importance as a metabolic disease risk indicator.

Limitations

The study's limitations include potential influences of various variables on observed differences between obese and non-obese individuals. Factors like age, gender, lifestyle habits, and underlying medical conditions may introduce bias and impact metabolic and cardiovascular markers. Without proper control for these factors, attributing differences solely to obesity status becomes challenging. Additionally, the study's cross-sectional nature limits establishing causality or understanding the temporal relationship between obesity and outcomes. Longitudinal studies or randomized controlled trials would offer more robust evidence on obesity's impact on metabolic and cardiovascular health over time. Future prospective clinical studies with larger patient populations are essential.

Conclusions

Our study underscores the urgency of addressing obesity as a serious health concern, with compelling evidence linking increased BMI to adverse cardiovascular outcomes. Higher epicardial fat tissue in obese individuals, known precursor to coronary inflammation and atherosclerosis, was observed. Obese participants also displayed elevated fasting glucose and triglyceride levels, indicating heightened cardiovascular and metabolic risks. Gender-related differences highlight the complexity of these associations, with obese men showing greater susceptibility to metabolic and cardiovascular diseases compared to women. Our study emphasizes BMI's significance as a key indicator of metabolic disease risk, particularly among obese individuals, as evidenced by its correlations with epicardial fat thickness and metabolic parameters. By elucidating these intricate relationships, our research stresses the importance of effective weight management strategies in reducing cardiovascular risk and improving overall health outcomes in obese populations.

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