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The relationship between thyroid functions, vitamin B12, and lipid profiles across different BMI categories in adults

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ABSTRACT

Aims: Obesity is a multifactorial condition characterized by abnormal or excessive fat accumulation that adversely impacts health and disrupts various metabolic processes. This study aimed to assess thyroid function tests, vitamin B12 levels, and lipid profiles in normal, overweight, and obese adults, and to elucidate the correlation with body-mass index (BMI) values.

Methods: This study was planned as a retrospective descriptive cross-sectional study. Within the scope of the study, age, gender, occupation, history of chronic diseases, smoking, alcohol use and drug use; weight, height, BMI, blood pressure; TSH, free T3, free T4, total cholesterol, LDL-cholesterol, HDL-cholesterol, VLDL-cholesterol, triglyceride, fasting blood glucose, vitamin B12, folate, ferritin, serum iron level, iron binding capacity and whole blood parameter values were retrieved and recorded by reviewing health records retrospectively. Patients were grouped according to BMI values, and the relationships between obesity, sociodemographic characteristics and blood parameters were analyzed.

Results: A total of 539 patients were analyzed, with 63.6% identified as women. The average age of the patients was 36.88±13.75 years (range: 13-79), and the average BMI was 26.92±6.37 kg/m². Analysis revealed that 40% of patients were classified as normal weight, 30.1% as overweight, 26.1% as obese, and 3.8% as underweight based on BMI criteria. The classification of obesity indicates that class 1 obesity accounts for 59.7%, while class 2 and class 3 obesity each represent 20.1% of the total cases. The obesity rate was 72.1% in women and 27.9% in men, with a statistically significant difference observed between genders and BMI groups ($p<0.001$). The prevalence of B12 deficiency was 1.2%, and no significant association was observed among BMI groups. The study identified a statistically significant difference in total cholesterol ($p<0.001$), HDL ($p=0.001$), LDL ($p<0.001$), VLDL ($p<0.001$), triglycerides ($p<0.001$), and BMI groups. Conversely, no significant relationship was observed between B12 values and TSH ($p=0.430$), fT3 ($p=0.462$), or fT4 ($p = 0.279$).

Conclusion: In conclusion, our findings indicate that BMI significantly influences the lipid profile of individuals; however, it does not exhibit a direct relationship with B12 levels or thyroid functions. Given the fact that obesity elevates cardiometabolic risks, particularly through heightened lipid levels, it is essential to monitor not only obese individuals but also those at risk for it as well, to reduce obesity and prevent its onset.

Keywords: BMI, obesity, vitamin B12, lipid, TSH, thyroid hormones

INTRODUCTION

Obesity is a complex condition characterized by abnormal or excessive body fat accumulation, adversely affect. Obesity typically results from an imbalance between energy intake and expenditure; nonetheless, genetic, environmental, psychological, and socioeconomic factors significantly contribute to its development.[1,2] The World Health Organization (WHO) classifies individuals with a body-mass index (BMI) of 25-29.9 kg/m² as overweight and individuals with a BMI of ≥30 kg/m² as obese according to the BMI, which is the most widely used criterion to evaluate obesity.[2] The global prevalence of obesity has increased rapidly in recent years. According to 2022 data, more than 1 billion people worldwide are obese, and approximately 880 million of these people are adults, and 159 million are children.[3,4] The effects of obesity on energy metabolism, hormonal balance, and absorption of nutrients lead to changes in metabolic and endocrine parameters. Thyroid hormones have a critical role in the regulation of energy metabolism. Various studies have shown that especially thyroid-stimulating hormone (TSH) levels show a positive correlation with BMI in obese individuals, and this has a significant effect on thyroid

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functions.[5] This suggests that thyroid dysfunction in obese individuals may worsen metabolic balance.

Vitamin B12 is crucial for DNA synthesis, hematopoiesis, and the functioning of the nervous system. Factors such as obesity, alterations in dietary habits, insufficient intake, and the use of proton pump inhibitors can contribute to an increased risk of vitamin B12 deficiency. This deficiency can result in elevated homocysteine levels, thereby heightening the risk of cardiovascular disease.[6]

Obesity induces dyslipidemia through its impact on lipid metabolism. Obese individuals often exhibit elevated triglyceride and low-density lipoprotein (LDL) cholesterol levels, alongside reduced high-density lipoprotein (HDL) cholesterol levels. Atherogenic dyslipidemia refers to these lipid profile changes and constitutes a modifiable risk factor for cardiovascular diseases.[7]

This study evaluates thyroid function tests, vitamin B12 levels, and lipid profiles in normal, overweight, and obese adults, aiming to determine their relationship with BMI values. In particular, we hypothesised that increasing BMI leads to significant changes in thyroid function (especially TSH levels), vitamin B12 levels and lipid profile parameters and that these changes reflect obesity-related metabolic disorders.

METHODS

The study was approved by the Dicle University Non-interventional Ethics Committee (Date: 05.08.2014, Decision No: 306), and conducted by the Declaration of Helsinki. Patient data were anonymized by confidentiality principles.

This study was planned as a retrospective descriptive cross-sectional study. This study analyzed the records of patients aged 18 years and older who visited the Family Medicine, Endocrinology, and Dietary outpatient clinics at Dicle University Faculty of Medicine Hospitals from January 1, 2014, to December 31, 2014. Participants admitted to the outpatient clinics with recorded weight and height measurements, along with conducted investigations, were included in the study. Age, gender, occupation, history of chronic diseases, smoking, alcohol and drug use, weight, height, BMI, blood pressure, TSH, free T3, free T4, total cholesterol, LDL-cholesterol, HDL-cholesterol, VLDL-cholesterol, triglycerides, fasting blood glucose, vitamin B12, folate, ferritin, serum iron level, iron binding capacity, and whole blood parameter values were retrospectively scanned in the health records and entered into the data collection form. No additional laboratory or imaging methods were used, and only existing patient records were considered. Exclusion criteria were individuals aged <18 years and patients with incomplete or inaccurate laboratory results.

BMI calculations of the patients were made according to WHO, calculated by dividing their body weight (in kilograms) by their height (in meters squared), recorded, and classified. BMI value <18.5 kg/m^2 was categorized as underweight, 18.5-24.9 kg/m^2 as normal weight, BMI 25-29.9 kg/m^2 as overweight, and BMI ≥30 kg/m^2 as obese. In obesity classification, Obesity class I represents patients with a BMI of 30-34.9 kg/m^2, Obesity class I BMI 35-39.9 kg/m^2, and Obesity class I BMI ≥40 kg/m^2. The following reference ranges and units were used for laboratory parameters (Table 1).

Blood tests for hemoglobin, hematocrit, mean cell volume, and platelets were performed using the Sysmex XN-1000 SA-01 device with the laser method; glucose, total iron-binding capacity, iron, HDL, LDL, VLDL, total cholesterol, and triglycerides were analyzed using the Beckman Olympus AU5800 autoanalyzer with the photometric method; ferritin, folate, TSH, FT4, FT3, and vitamin B12 were measured using the Beckman DxI 600 device with the electrochemiluminescence method; and HbA1c was analyzed using the BIO-RAD Variant II device.

Statistical Analysis

Data were analyzed with IBM SPSS Statistics Version 23 software.

- The conformity of the data to normal distribution was evaluated with the Kolmogorov-Smirnov test.
- The Kruskal-Wallis H test was used to compare non-normally distributed data in three or more groups, and Dunn's test was used for multiple comparisons.
- One-way analysis of variance (ANOVA) was used to analyze the normally distributed data in three or more groups, and the Tukey test was used for multiple comparisons.
- The relationships between variables that did not fit the normal distribution were evaluated by Spearman's rho correlation.
- Quantitative data were presented as mean±standard deviation and median (minimum-maximum); categorical data were presented as frequency and percentage.
- The significance level was accepted as $p<0.05$ in all statistical analyses.

RESULTS

The data of 539 patients were analyzed, 63.6% of the patients were women and 36.4% were men. The mean age of the patients was 36.88±13.75 (13-79)/year, the mean height was 165.45±8.32 cm, the mean weight was 73.99±18.64 kg, and the mean BMI was 26.92±6.37 kg/m^2. Descriptive statistics of quantitative variables are given in Table 2.

The analysis revealed that the prevalence of chronic diseases among patients was 59.7%. Hypertension prevalence was 29.9%, diabetes 50.3%, hyperlipidemia 6.4%, coronary artery disease 10.8%, hyperthyroidism 7.6%, and hypothyroidism 29.9% among chronic diseases. The proportion of individuals who did not use medication regularly was 56.8%, whereas the proportion of those who did was 43.2%.

The classification based on BMI values revealed that 40% of patients were of normal weight, 30.1% were overweight, and 26.1% were classified as obese, while 3.8% fell into the underweight category. The classification of obesity indicates that class 1 obesity accounts for 59.7%, class 2 obesity for 20.1%, and class 3 obesity for 20.1%.

Table 1. Reference ranges and units were used for laboratory parameters

Glucose: 74-106 mg/dl	Triglyceride: 50-180 mg/dl	Vitamin B12: 211-911pg/ml
TIBC: 1550-3550 µg/L	Total cholesterol: 112-200 mg/dl	Hb: 12.9-14.2 g/dl
Iron: 600-1800 µg/L	HDL: 37-79 mg/dl	Htc: 37.7-53.7%
HbA1c: 4.3-6.1%	LDL: 60-160 mg/dl	MCV: 81.1-91.6 fL
TSH: 0.35-5.5 µIU/ml	VLDL: 10-32 mg/dl	Plt: 155-366 10e3/uL
FT4: 0.89-1.76 ng/dl	Ferritin: 11-306.8 µg/L	
FT3: 2.3-4.2 pg/ml	Folate: 5.9-24.8 µg/L	

Abbreviations: TIBC: Total iron binding capacity, HbA1c: Hemoglobin A1c, TSH: Thyroid-stimulating hormone, FT4: Free T4, FT3: Free T3, Vit. B12: Vitamin B12, Hb: Hemoglobin, Hct: Hematocrit, MCV: Mean corpuscular volum, Plt: Platelet, HDL: High density lipoprotein, LDL: Low density lipoprotein, VLDL: Very low density lipoprotein

Table 2. Descriptive statistics of quantitative variables

	Mean±SD*	Median (min-max)
Age (years)	36.888±13.751	35 (13-79)
Length (cm)	165.454±8.32	165 (145-195)
Weight (kg)	73.994±18.641	72 (30-210)
BMI value (kg/m^2)	26.916±6.367	25.9 (13.8-59.2)
SBP (mmHg)	112.936±18.533	110 (80-200)
DBP (mmHg)	65.483±12.654	60 (40-120)
TSH (µIU/ml)	5.195±22.744	1.54 (0.01-248)
FT4 (ng/dl)	17.011±9.979	15.9 (1.52-129.2)
FT3 (pg/ml)	5.089±3.292	4.745 (0.27-49.8)
Blood sugar (mg/dl)	115.476±63.815	96 (32-672)
Triglyceride (mg/dl)	148.449±101.424	119 (35-1110)
Total cholesterol (mg/dl)	196.656±116.952	184 (38-2221)
HDL (mg/dl)	45.207±12.563	43 (3-96)
LDL (mg/dl)	115.759±37.764	110 (27-299)
VLDL (mg/dl)	31.125±27.178	24 (7-313)
Ferritin (µg/L)	58.904±70.109	28.2 (1.46-464)
Folate (µg/L)	9.074±6.771	8.415 (2.6-89.6)
B12 (pg/ml)	325.492±154.612	291 (26.38-1244)
Iron (µg/L)	74.294±39.718	69 (8-215)
TIBC (µg/L)	269.281±79.772	271 (26-527)
Hemoglobin (g/dl)	13.929±1.925	14 (7.8-18.75)
Hematocrit (%)	42.207±5.126	42 (26.36-59.17)
MCV	84.984±7.24	86 (30-107)
Platelet x1000	274.162±72.888	264 (32.6-658.9)

Abbreviations: SD: Standard deviation, Min: Minimum, Max: Maximum, BMI: Body-mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, TSH: Thyroid-stimulating hormone, FT4: Free T4, FT3: Free T3, HDL; High density lipoprotein, LDL: Low density lipoprotein, VLDL: Very low density lipoprotein, Vit. B12: Vitamin B12, TIBC: Total iron binding capacity, MCV: Mean corpuscular volum

The comparison of BMI groups, age, and vitamin B12 deficiency is presented in Table 3. Obesity prevalence was identified at 72.1% in women and 27.9% in men, with a statistically significant difference observed between gender and BMI groups (p<0.001). The prevalence of B12 deficiency was determined to be 1.2%, with no significant correlation identified among BMI groups.

The quantitative data of the patients were compared across groups based on BMI, as presented in Table 4. A statistically significant difference was observed across age, blood pressure values (systolic and diastolic), blood sugar, blood lipids, blood glucose, ferritin, iron, iron binding capacity, B12 value, hemoglobin, hematocrit, MCV, platelet level, and BMI groups (p<0.001 for all values). Obese patients exhibited higher age, blood pressure values (systolic and diastolic), blood glucose, and blood lipids (excluding HDL), while HDL levels were lower compared to non-obese patients. The overweight group exhibited elevated levels of ferritin, iron, hemoglobin, hematocrit, and MCV values.

The analysis of the correlation between BMI and quantitative variables revealed statistically significant positive correlations with systolic blood pressure (r=0.440; p<0.001), diastolic blood pressure (r=0.400; p<0.001), blood sugar (r=0.320; p<0.001), triglyceride level (r=0.381; p<0.001), total cholesterol level (r=0.227; p<0.001), LDL level (r=0.204; p<0.001), VLDL level (r=0.375; p<0.001), ferritin (r=0.265; p<0.001), iron (r=0.159; p=0.016), hemoglobin (r=0.119; p=0.009), and hematocrit level (r=0.161; p<0.001). Conversely, HDL level exhibited a negative correlation (r=-0.208; p<0.001). The correlation analysis revealed no statistically significant results between BMI and other quantitative variables (Table 5).

In addition, in the correlation analysis of smoking with quantitative variables, significant relationships were found between HDL level (r=-0.144, p=0.025), ferritin level (r=0.181, p=0.035), hematocrit level (r=0.208, p<0.001), hemoglobin level (r=0.190, p<0.001) and MCV level (r=0.229, p<0.001). In the correlation analysis of alcohol use with quantitative variables, significant relationships were found between total cholesterol level (r=0.167, p=0.021), LDL level (r=0.167, p=0.029), hemoglobin level (r=0.197, p=0.003), hematocrit level (r=0.177, p=0.007) and MCV level (r=0.184, p=0.005). No statistically significant results were found in the correlation analysis between smoking and alcohol use and other quantitative variables.

DISCUSSION

This study examined a sample group of 539 individuals, revealing an obesity rate of 26.1% among participants. Obesity prevalence in women was 2.5 times greater than in men. A statistically significant correlation was observed between blood lipids and BMI categories; however, no correlation was found between thyroid function tests and B12 levels.

This study found that 40% of patients were classified as normal weight, 30.1% as overweight, and 26.1% as obese, while

Table 3. Comparison of BMI groups and categorical demographic characteristics

		Underweight	Normal	Overweight	Obese	Total	Test statistic	p
		n (%)	n (%)	n (%)	n (%)	n (%)		
Gender	F	15 (75)^{abc}	145 (69.4)^{c}	71 (45.2)^{b}	98 (72.1)^{ab}	329 (63)	30.965	**<0.001**^{x}
	M	5 (25)^{abc}	64 (30.6)^{c}	86 (54.8)^{b}	38 (27.9)^{ac}	193 (37)		
Vitamin B12 deficiency	Yes	0 (0)	3 (1.5)	1 (0.7)	2 (1.6)	6 (1.2)	0.955	0.819^{y}
	No	19 (100)	193 (98.5)	145 (99.3)	121 (98.4)	478 (98.8)		

x: Pearson Chi-square test, y: Fisher's Exact test with Monte Carlo correction, a-c: There is no difference between the rates, n (%). Abbreviations: BMI: Body-mass index, F: Female, M: Male

Table 4. Comparison of quantitative data between groups according to BMI

	Underweight	Normal	Overweight	Obese	Total	Test statistic	p
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)		
Age	20.5 (15-50)[a]	29 (13-77)[b]	37.5 (15-71)[c]	43 (15-79)[c]	35 (13-79)	78.140	**<0.001**[x]
SBP (mmHg)	110 (90-120)[abc]	100 (80-200)[a]	110 (80-155)[b]	120 (90-190)[c]	110 (80-200)	36.352	**<0.001**[x]
DBP (mmHg)	60 (60-80)[abc]	60 (40-120)[a]	62.5 (45-90)[b]	75 (50-100)[c]	60 (40-120)	34.778	**<0.001**[x]
TSH (µIU/ml)	1.185 (0.01-5.31)	1.55 (0.01-207)	1.585 (0.01-181)	1.51 (0.01-248)	1.54 (0.01-248)	2.759	0.430[x]
FT4 (ng/dl)	17 (13.6-32.68)	15.98 (9.16-129.2)	15.945 (6.93-29.92)	15.5 (1.52-120)	15.9 (1.52-129.2)	3.842	0.279[x]
FT3 (pg/ml)	5.36 (4.17-25.58)	4.69 (3.23-49.8)	4.78 (2.37-18.42)	4.73 (0.27-6.19)	4.745 (0.27-49.8)	2.576	0.462[x]
Blood sugar (mg/dl)	91 (64-384)[ab]	92 (32-558)[a]	98 (67-672)[bc]	101 (70-382)[c]	96 (32-672)	43.213	**<0.001**[x]
Triglyceride (mg/dl)	70 (41-123)[a]	98 (35-1110)[a]	135 (44-481)[b]	147.5 (61-487)[b]	119 (35-1110)	45.741	**<0.001**[x]
Total cholesterol (mg/dl)	156 (104-226)[a]	179 (58-380)[a]	190 (115-2221)[ab]	194 (38-634)[b]	184 (38-2221)	18.127	**<0.001**[x]
HDL (mg/dl)	51 (45-72)[ab]	48 (15-96)[a]	42 (8-80)[c]	43 (25-88)[bc]	43 (3-96)	27.708	**<0.001**[x]
LDL (mg/dl)	78 (67-156)[a]	101 (34-299)[ab]	113.5 (42-293)[bc]	114.5 (27-228)[c]	110 (27-299)	16.130	**0.001**[x]
VLDL (mg/dl)	14 (8-25)[a]	19.5 (7-222)[a]	27 (9-128)[b]	29 (13-313)[b]	24 (7-313)	43.849	**<0.001**[x]
Ferritin (µg/L)	22.68 (4.07-87.14)[ab]	19.88 (1.46-199)[a]	67.5 (3.7-464)[b]	35. 89 (3.82-222)[ab]	28.2 (1.46-464)	23.841	**<0.001**[x]
Folate (µg/L)	7.585 (5-10.94)	8.5 (2.6-18.17)	7.96 (3.69-20)	8.995 (5.09-15.71)	8.415 (2.6-89.6)	1.659	0.646[x]
Vit. B12 (pg/ml)	262.8 (165.7-346)	291.3 (140.4-778)	288.1 (26.38-1244)	299.9 (113.9-1083)	291 (26.38-1244)	1.931	0.587[x]
Demir (µg/L)	66.5±32.667[ab]	67.382±35.044[a]	91.906±46.698[b]	69.638±34.979[a]	74.294±39.718	4.472	**0.010**[y]
TIBC	293.875±68.265[ab]	276.99±80.909[ab]	230.919±76.891[b]	294.933±66.895[a]	269.281±79.772	7.460	**<0.001**[y]
Hemoglobin	14.02 (10-16.7)[ab]	13.045 (8.52-18.75)[a]	14.85 (7.8-18.33)[b]	14 (9.86-18.25)[ab]	14 (7.8 -18.75)	22.688	**<0.001**[x]
Hematocrit	41.74 (36-48.5)[ab]	40.59 (26.36-54.93)[a]	44 (27.6-59.17)[b]	42.8 (30.4-53.5)[b]	42 (26.36-59.17)	26.340	**<0.001**[x]
MCV	86.4 (69-91.73)[ab]	85.985 (31-97.72)[ab]	86.95 (58-107)[a]	84 (30-96.98)[b]	86 (30-107)	8.864	**0.031**[x]
PlateletX1000	238.8 (186-376.7)	268.3 (43.4-513)	261 (121.6-442)	278.9 (89.3-658.9)	264 (32.6-658.9)	7.183	0.066[x]

[x]: Kruskal-Wallis H test, [y]: One-way analysis of variance, (a-c): There is no difference between groups with the same letter. Mean±standard deviation, Median (minimum-maximum). Abbreviations: BMI: Body-mass index, SD: Standard deviation, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, TSH: Thyroid-stimulating hormone, FT4: Free T4, FT3: Free T3, HDL; High density lipoprotein, LDL: Low density lipoprotein, VLDL: Very low density lipoprotein, Vitamin B12, TIBC: Total iron binding capacity, MCV: Mean corpuscular volum

Table 5. Examination of the relationship between BMI value and quantitative variables

	BMI value	
	r^x	p
Systolic blood pressure	0.440	<0.001
Diastolic blood pressure	0.400	<0.001
TSH level	0.039	0.436
FT4 level	-0.059	0.284
FT3 level	-0.086	0.165
Blood sugar	0.320	**<0.001**
Triglyceride level	0.381	**<0.001**
Total cholesterol level	0.227	**<0.001**
HDL level	-0.208	**<0.001**
LDL level	0.204	**<0.001**
VLDL level	0.375	**<0.001**
Ferritin level	0.265	**<0.001**
Folate level	-0.009	0.913
Vitamin B12 level	0.008	0.904
Iron level	0.159	**0.016**
Total iron binding capacity	-0.048	0.486
Hemoglobin level	0.119	**0.009**
Hematocrit level	0.161	**<0.001**
MCV level	-0.073	0.111
Platelet levelx1000	0.056	0.222

[x]: Spearman's rho correlation, Abbreviations: BMI: Body-mass index, TSH: Thyroid-stimulating hormone, FT4: Free T4, FT3: Free T3, HDL: High density lipoprotein, LDL: Low density lipoprotein, VLDL: Very low density lipoprotein, MCV: Mean corpuscular volume

3.8% fell into the underweight category based on BMI. The classification of obesity indicates that class 1 obesity accounts for 59.7%, while both class 2 and class 3 obesity each represent 20.1% of the total cases. The obesity rate was 72.1% in women and 27.9% in men, with a statistically significant difference observed between genders and BMI groups (p<0.001). The prevalence of B12 deficiency was 1.2%, and no significant association was observed between BMI categories. The study identified a statistically significant difference in total cholesterol (p<0.001), HDL (p=0.001), LDL (p< 0.001), VLDL (p<0.001), triglycerides (p<0.001), and BMI groups. However, no significant relationship was observed between B12 values and TSH (p=0.430), fT3 (p=0.462), or fT4 (p=0.279).

It was noted that 63.6% of the participants were female. Literature indicates that women typically exhibit higher participation rates.[8,9] Analysis of the BMI distribution in our study revealed that 40% of participants were classified as normal weight, 30.1% as overweight, and 26.1% as obese. These rates corroborate research indicating a rising prevalence of obesity in developing nations, including Turkey.[1,10] Data from the Turkish Statistical Institute (TÜİK) in 2022 indicates that the obesity rate among individuals aged 15 years and older was 20.2%. This rate is lower than the 26.1% obesity rate reported in our study. This data aligns with the 2023 report from the World Obesity Federation, released in 2022, which predicts that over half of adults in Turkiye may be obese by 2035.[11] The elevated obesity rates observed in our study compared to national data may be attributed to regional variations or specific characteristics of the study population, given that Turkey is situated in the eastern Anatolia region and has a dietary pattern that includes frequent consumption of fat and meat products. This situation highlights the significance of regional strategies in combating obesity. The combined rates of Obesity in class 2 and class 3 obesity classification stand at 40.2%, highlighting the significance of severe obesity as a critical issue.

In our study, significant positive correlations were found between systolic and diastolic blood pressure and BMI. This finding is consistent with existing data in the literature. For

example, in a study conducted on sedentary women, it was determined that systolic and diastolic blood pressure showed significant positive relationships with BMI. In Mendeş and Mendeş's[13] study, a weak positive relationship was found between BMI and systolic and diastolic blood pressure in adult individuals.[12,13] These data emphasize that increasing body weight has negative effects on blood pressure, and obesity is an important risk factor in the development of hypertension. Therefore, the positive association between BMI and blood pressure suggests that obesity may impair blood pressure regulation and increase cardiovascular risks.

In our study, a significant positive association was found between BMI and blood glucose. This finding is consistent with existing data in the literature. For example, in a study conducted at Gaziantep University, it was determined that fasting blood glucose levels increased as BMI increased in obese individuals.[14] Similarly, a study conducted on university students in Istanbul showed that as anthropometric measurements such as BMI, waist circumference, and waist/height ratio increased, random blood glucose levels also increased.[15] These data support that increasing body fat leads to insulin resistance and increases blood glucose levels, and thus there is a positive relationship between BMI and blood glucose.

In the study, a significant positive correlation was found between triglyceride levels and BMI. This indicates that as BMI increases, triglyceride levels also increase. Similarly, a significant positive relationship was observed between total cholesterol levels and BMI. This finding suggests that an increase in BMI may affect total cholesterol levels. A significant negative relationship was found between HDL level and BMI. This suggests that HDL levels decrease with increasing BMI, which is a significant factor increasing cardiovascular risks. A significant positive relationship was also found between LDL levels and BMI, suggesting that higher BMI contributes to an increase in LDL levels. These findings are consistent with existing studies in the literature. For example, a study conducted in 2018 reported that there were positive and negative associations between BMI and triglyceride and HDL levels, respectively, but no significant association was found with LDL.[16-18] These results suggest that increasing BMI has significant effects on lipid profile and should be considered in terms of cardiometabolic health. In the study in which metabolic parameters of obese and coronary heart disease patients were evaluated, it was shown that both obese and coronary heart disease patients were more dyslipidaemic, with higher LDL and TG levels and lower HDL levels compared to healthy controls.[19] The risks of cardiometabolic comorbidities should not be reduced to the assessment of BMI alone. It should be kept in mind that in the presence of normal BMI but high body fat percentage, which is called normal weight obesity, lipid profile disorder may be seen more frequently compared to people without normal weight obesity, and in this case, a worse cardiometabolic profile may be encountered.[20]

In this study, no significant relationship was found between TSH level and BMI. This finding is consistent with some studies in the literature. For example, in a study conducted by Manji et al.[21] in 2006, no significant relationship was found between serum TSH or free T4 levels and BMI in euthyroid individuals. Similarly, in another study conducted in 2010, TSH levels were found to be within normal reference ranges in obese individuals.[5] However, there are also studies in the literature that found a positive correlation between TSH levels and BMI. For example, Knudsen et al.[22] in 2005 reported that even small changes in thyroid function in the general population may have an effect on BMI and the incidence of obesity. In a large population-based study conducted in Spain investigating the prevalence of abnormal thyroid function and potential modulatory factors, mean serum TSH levels in euthyroid individuals were found to be higher in those with obesity (BMI ≥30 kg/m²).[23] The impact of thyroid hormones on lipid profiles has been widely investigated. In Wang Y.'s[24] study, the role of BMI in modulating the relationship between thyroid hormones (TH) and lipid parameters in euthyroid healthy adults was examined. The findings suggest that high-normal FT3 and TSH levels, as well as low-normal FT4 levels, are associated with an unfavorable lipid profile. Moreover, BMI mediates the effect of thyroid function on lipid metabolism in euthyroid adults.[24] Given the clinical significance of the interaction between metabolism and thyroid hormones, it is crucial to determine whether obesity leads to elevated TSH levels or whether higher TSH levels contribute to obesity. In Wang X.'s[25] study, this relationship was examined, and the findings indicated that genetically predicted serum TSH levels do not directly influence BMI or obesity risk. However, TSH levels were significantly elevated as a result of genetically determined high BMI. A study evaluating the association between TSH levels and metabolic profiles in obese individuals demonstrated that obesity complicated by mildly elevated TSH is associated with higher fasting insulin levels, more severe chronic low-grade inflammation, and lower HDL levels compared to obesity with normal TSH levels.[26] In Marzullo's[27] study, the impact of metabolic phenotype on thyroid function in obesity was evaluated. The findings indicated that FT4 levels were inversely associated with BMI, insulin resistance, and triglyceride levels, while showing a direct correlation with HDL-cholesterol levels. Additionally, TSH was found to be associated with FT4, total cholesterol, and BMI. Significant predictors of FT4 included BMI, TSH, and age.[27]

In clinical practice, ferritin is used for diagnosing iron deficiency; however, it can also serve as a marker of inflammation. In Khan's[28] study, the role of ferritin in overweight and obese individuals was investigated, either as an indicator of inflammation or iron deficiency. The findings revealed that ferritin levels were highest in obese patients and showed a strong positive correlation with BMI. The study concluded that ferritin serves as a marker of inflammation rather than iron status in overweight and obese individuals.[28] In our study, a positive correlation was found between BMI and ferritin levels. In a nationwide population-based study conducted in South Korea, serum ferritin levels were found to be positively associated with metabolically obese normal-weight individuals.[29] In a study conducted among the adult population in the United States, a linear positive association was observed between BMI and serum ferritin levels.[30] In a study evaluating the relationship between the percentage of visceral fat mass and serum ferritin levels, a significant

negative association was found between visceral fat mass and serum ferritin. Additionally, serum ferritin showed a significant positive correlation with BMI and the waist-to-hip ratio.[31]

In our study, significant positive correlations were found between alcohol consumption and total cholesterol, LDL, hemoglobin, hematocrit, and MCV levels. These findings are consistent with studies in the literature showing the effects of alcohol consumption on various hematologic and biochemical parameters. Similarly, in a study conducted by Demir and Özsoy[33], it was found that liver enzymes as well as lipid profiles were affected in patients with alcohol use disorder; especially when total cholesterol and LDL levels increased. In a study conducted by Kulu[34], MCV values were found to be significantly higher in individuals with alcohol use disorder compared to the control group. In addition, it was determined that hemoglobin levels were higher but platelet levels were lower in alcohol addicts compared to the control group.[33-34]

Limitations

This study utilized data obtained retrospectively from the hospital registration system. Some individuals were excluded from the study due to incomplete records or incorrect entries. The potential use of non-standardized methods for measuring clinical and laboratory values in individuals is acknowledged as a limitation that may influence the results. The cross-sectional design of the study presents a significant limitation, as it precludes the evaluation of causal relationships between BMI and biochemical parameters. A relationship exists between obesity and changes in lipid profiles; however, the direction of this relationship and the underlying mechanisms remain unexplained.

The study did not evaluate critical variables that may influence obesity, including socioeconomic status, dietary habits, physical activity levels, and other environmental factors. The study was limited to individuals admitted to a single center. This restricts the applicability of the results to the broader population and raises concerns about the validity of the findings for individuals in different regions.

While the study incorporated lifestyle habits like smoking and alcohol consumption, it lacked comprehensive data regarding the frequency and duration of these behaviors. This constituted a limitation in the comprehensive evaluation of the impacts of smoking and alcohol on biochemical parameters. The study was limited to data from a specific timeframe, preventing the longitudinal analysis of changes in weight, blood values, and other individual parameters over time. This constrains the assessment of the outcomes from a dynamic viewpoint.

A commission established through a real collaboration involving 56 leading experts from a wide range of disciplines, representing high-income, middle-income, and low-income countries, announced on January 14, 2025, a new definition and diagnostic framework that clarifies when obesity is a risk factor (preclinical obesity) and when it is a disease on its own (clinical obesity).[35] The new, evidence-based definition distinguishes "clinical obesity," a chronic, systemic disease condition directly caused by excess fat, from "preclinical obesity," a condition of excess fat with no current organ dysfunction or limitations in daily activities but with an increased future health risk. Since our study was planned and data were collected prior to the establishment of this definition, patients were categorized according to BMI and evaluated by comparing with laboratory parameters. A specific assessment regarding clinical or preclinical obesity was not provided.

Finally, the study did not assess factors that may influence obesity, such as psychological stress, cortisol levels, or other hormonal parameters. All these limitations stand out as important issues that should be accounted for when interpreting the study findings.

The strength of this study is that the relationships between obesity and important metabolic and biochemical parameters such as thyroid function, vitamin B12 levels, and lipid profile were comprehensively evaluated with a large sample group. A detailed analysis of different BMI categories provided a better understanding of the effects of obesity on biochemical parameters. In addition, the fact that the data used in the study were obtained from the hospital registration system allowed the evaluation of data obtained under real-life conditions and made it possible to directly reflect the results in clinical practice.

CONCLUSION

This study assessed the associations between obesity and several biochemical and metabolic parameters, yielding significant findings. Obesity elevates cardiometabolic risks through the rise in blood glucose, triglycerides, total cholesterol, LDL cholesterol, and VLDL cholesterol levels, while simultaneously reducing HDL cholesterol levels as BMI increases. Additionally, elevated ferritin levels have been noted in obese individuals, suggesting potential inflammation or alterations in iron metabolism. Nonetheless, no significant correlation was identified between BMI and TSH, FT3, and FT4 levels, suggesting that the discussion regarding the impact of obesity on thyroid function remains unresolved.

The data obtained in the study once again emphasize the negative effects of obesity on metabolic health and reveal the importance of strategies to combat obesity. It is concluded that metabolic and biochemical changes associated with obesity should be regularly monitored, and individualized treatment plans should be established in health policies and clinical practices. In the future, more comprehensive and prospective studies should be conducted to investigate the biochemical effects and cause-effect relationships of obesity in more detail.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was approved by the Dicle University Non-interventional Ethics Committee (Date: 05.08.2014, Decision No: 306).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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