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The study included forty MetS patients and forty age- and sex-matched healthy controls. Demographic and clinical data were collected, the serum preptin levels were measured by Enzyme-Linked Immunosorbent Assay (ELISA). The relationships between the serum preptin levels and clinic parameters were assessed.

Results

Serum preptin levels were significantly higher in MetS patients compared to the control group (53.77 pg/mL vs. 40.77 pg/mL, $p<0.001$). MetS patients also had higher triglyceride, cholesterol, low-density lipoprotein cholesterol (LDL-C), fasting glucose, Hemoglobin A1c (HbA1c), insulin, and Homeostatic model assessment of insulin resistance (HOMA-IR) values compared to controls (all $p<0.001$). A positive correlation was found between serum preptin, insulin ($r=0.432$, %95 CI:0.07-0.57, $p=0.0050$), and HOMA-IR ($r=0.426$, %95 CI:0.08-0.56, $p=0.0060$) in the MetS group.

Conclusions

The results indicate that serum preptin levels are significantly elevated in MetS patients. Furthermore, high serum preptin levels were positively correlated with insulin and insulin resistance. These results suggest that preptin may be involved in the insulin resistance pathway via insulin-like growth factor II (IGF-II) modulation. Therefore, further extensive studies are required to fully understand the precise role in MetS and underlying mechanisms of preptin.

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1. Introduction

Metabolic syndrome (MetS) is a complex clinical condition that encompasses a cluster of interrelated metabolic abnormalities, increasing susceptibility to diabetes and cardiovascular diseases. This condition includes risk factors such as dysglycemia, arterial hypertension, hypertriglyceridemia, low levels of high-density lipoprotein cholesterol (HDL-C), and obesity characterized particularly by fat accumulation in the abdominal region. [1]. These risk factors significantly increase the prevalence of type 2 diabetes (T2DM), cardiovascular diseases, non-alcoholic fatty liver, obesity, and the related metabolic disorders [1,2]. All over the world, MetS occurs at rates ranging from 10% to 40% and frequently in obese people [3]. According to the National Cholesterol Education Program Adult Treatment Panel (NCEP-ATP III) criteria, MetS in adults is defined by the presence of any three or more of the following components: abdominal obesity (waist circumference >102 cm in men, >88 cm in women), hypertriglyceridemia (triglycerides >150 mg/dL), low HDL cholesterol (<40 mg/dL in men, <50 mg/dL in women), hypertension (systolic blood pressure >130 mmHg or diastolic blood pressure >85 mmHg), and hyperglycemia (fasting blood glucose >110 mg/dL) [4].

According to the guideline published by the International Diabetes Federation (IDF) in 2005, abdominal obesity is a prerequisite for the diagnosis of MetS. Additionally, at least two of high triglycerides, low HDL-C, high blood pressure and high fasting glucose must be present in cases [5,6]. In this study, we selected cases based on the diagnostic criteria established by both NCEP and IDF.

Peptide hormones play a crucial role in food intake and carbohydrate metabolism and studies on these hormones have been quite remarkable in recent years. One of these

hormones is preptin. Preptin is a peptide containing 34 amino acid and it was first isolated from pancreatic β cells of rats in 2001 [7]. Preptin, the most recently discovered member of the insulin family, is derived from pro-insulin-like growth factor II (pro-IGF II) and it is secreted from pancreatic β-cells with insulin and amylin [7]. It is thought to increase glucose-mediated insulin secretion [7,8].

In recent years, some studies on the relationship between T2DM and preptin level have shown that preptin levels are higher in the T2DM patients compared to the healthy control group [9,10]. Wang et al. (2019) investigated the serum preptin levels in diabetic nephropathy patients and they found that the serum preptin levels were high in T2DM and diabetic nephropathy. They also showed a positive correlation between the serum preptin levels and gender, body mass index, blood urea nitrogen and creatinine [11]. There are also some studies showing the changes in serum or plasma preptin levels in patients with polycystic ovary syndrome (PCOS), cardiovascular diseases, osteoporosis, and osteopenia [12,13,14].

Based on our knowledge, the studies showing the direct relationship between MetS and serum preptin levels are limited. Accordingly, our hypothesis is that serum preptin levels are significantly higher in individuals diagnosed with MetS compared to healthy controls, and that this increase is positively associated with components of MetS such as obesity, insulin resistance, and arterial hypertension. We think that the obtained data could be beneficial to better understand MetS and the related diseases which have a significant effect on public health and high socioeconomic costs. Also, the data contribute to the limited literature on this subject.

2. Material and methods

2.1 Subjects

In this study, forty patients who applied to the internal medicine outpatient clinic, Kırsehir Training and Research Hospital and diagnosed with MetS and forty healthy volunteers were included. Patients under 18 years of age and over 70 years of age, patients who did not participate voluntarily or dropped out of the study, morbidly obese patients, patients receiving chemotherapy, patients with known cardiovascular or cerebrovascular disease, and patients with severe organ failure were excluded. The control group consisted of healthy volunteers who were non-morbidly obese, non-diabetic, age- and gender-matched, without comorbidities and medication use. Ethics committee approval was Clinical Research Ethics Committee, Faculty of Medicine, Kırsehir Ahi Evran University (Approval no: 2020-10/85, Date: 07.07.2020) before the study was undertaken. Informed consent was obtained from all participants.

2.2 Biochemical measurements

Blood samples were collected in serum separator tubes (BD vacutainer SST II Advance: Becton, Dickinson and Company, USA) after an overnight fast. Blood samples were allowed to clot for 30 minutes and subsequently centrifuged at 2000 × g for 10 minutes to obtain serum. The collected serum samples were stored at −80°C until analysis. Fasting glucose and lipid profile measurements were obtained using an autoanalyzer system (AU 5800; Beckman Coulter, USA). Hemoglobin A1c (HbA1c) levels were determined by high-performance liquid chromatography (HPLC) (Premier Hb9210; Trinity biotech, Ireland). Insulin levels were determined in an immunoassay (DXI 800; Beckman Coulter, USA). IR was expressed as Homeostatic model assessment of insulin resistance (HOMA-

IR). The HOMA-IR value was calculated using the following formula: Fasting Glucose (mg/dL) × Insulin (μIU/mL) / 405. Serum preptin levels were determined using the Human preptin ELISA kit (Elabscience Biotechnology Inc., China) according to the manufacturer's instructions. Serum preptin measurement was performed using the SPECTROstar Nano microplate reader (BMG LABTECH, Germany).

2.3 Statistical analysis

Median and quartile (Q_1-Q_3) values were given for quantitative variables, while frequencies (n) and percentages (%) were reported for categorical variables. Kolmogorov-Smirnov test was used for normality assumption, and Levene's test was applied for testing variance homogeneity assumption ($p<0.05$). Chi-Square test was used to determine the relationship between MetS patients and the control groups in categorical variables. Independent Samples t-test or Mann-Whitney test was performed for a comparison of values across MetS patients and the control group. Spearman correlation coefficients were calculated to analyze the relationship between the parameters in both the cases and the control group.

In this study, a Receiver Operating Characteristic (ROC) analysis was performed to evaluate the discriminative performance of preptin levels between the MetS and control groups. The ROC curve plotting and calculation of the area under the curve (AUC) were conducted using the scikit-learn library. The cut-off points for the preptin threshold value of the MetS group and the control group were determined based on the relationship according to the Youden index. Sensitivity and specificity at the optimal threshold were also reported. The AUC measure was calculated with 95% confidence interval (CI) for preptin. Binary logistic regression analysis was used to determine the factors affecting

MetS (Enter Model). Variables with P<0.10 in binary analyses were included in the model. MetS was used as the dependent variable in the models for waist circumference, triglyceride, cholesterol, fasting glucose, HbA1c, insulin, HOMA-IR and preptin. The explanatory power model was evaluated according to Nagelkerke R square (Nagelkerke R^2). IBM SPSS Statistics version 22.0 (IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 23.0. Armonk, NY: IBM Corp.) was used for all analysis. The significance level was considered as < 0.05. Since no similar study was found in the literature, the sample size was calculated using the G*Power 3.1.9.7 (University of Düsseldorf, Germany) software to detect a large effect size (d = 0.8) with a 5% margin of error and 95% power. Accordingly, a total of 84 participants, with a minimum of 42 individuals in both the control and intervention groups, was determined to be necessary. However, during the study, two patients and two control group participants were excluded two due to missing data and two for not meeting the study criteria. As a result, the study was conducted with 40 participants in each group.

3. Results

The mean ages of the MetS group and the control group were 36.5±9.5 and 33.6±8.1 years, respectively. In the MetS group, 18 (45%) of the cases were male, 22 (55%) were female, and in the control group, 19 (52.5%) of the cases were male and 21 (47.5%) were female. There was no statistical difference between the MetS and the control group in terms of gender, smoking and alcohol use. A statistical difference was determined between these groups considering the presence of additional disease such as hypertension, obesity and blood pressure values. While the presence of additional disease was 10.0% in the MetS group, it was 0% in the control group. Although the rate of high and stage 1

hypertension was 52.5% in the MetS group, there was no high and stage 1 hypertension in the control group individuals ($p<0.001$).

There was also no significant difference between the MetS and the control group with respect to the age (36.5±9.5 vs. 33.6±8.1) and height (167.8±9.8 vs. 171.1±9.5) ($p>0.05$). Weight (88.8±9.3 vs. 73.6±11.2; $p<0.001$) and waist circumferences of the MetS group [97.5 (92.2-102) vs. 80 (77.2-92); $p<0.001$] were found to be statistically higher than the control group (Table 1). In addition, the triglyceride, cholesterol, low-density lipoprotein cholesterol (LDL-C), fasting glucose, HbA1c, insulin and HOMA-IR values of the MetS group were found to be statistically higher than the control group ($p<0.001$, $p<0.001$, $p = 0.002$, $p<0.001$, $p<0.001$, $p<0.001$, $p<0.001$ respectively) (Table 1). Serum preptin levels were significantly higher in the MetS group than in the controls [53.77 (46.57-60.73) vs. 40.77 (36.29-49.66), ($p<0.001$)] (Figure 1).

A positive correlation was determined between serum preptin and insulin ($r=0.382$, %95 CI:0.07-0.64, $p=0.015$), HOMA IR ($r=0.326$, %95 CI:0.01-0.59, $p=0.040$), HbA1c ($r=0.367$, %95 CI:0.02-0.66, $p=0.020$) levels in the control group (Figure 2). The serum preptin level in the MetS group also shown a positive correlation with insulin ($r=0.432$, %95 CI:0.07-0.57, $p=0.0050$) and HOMA-IR ($r=0.426$, %95 CI:0.08-0.56, $p=0.0060$) levels (Figure 3). The preptin level did not show any correlation with other parameters in both groups.

The Enter model is significant ($F= 13.37$, $p<0.001$) and these variables together explain 61.9% of the change in MetS ($R2= 0.670$). The waist circumference variable has a significant effect on MetS ($p<0.01$). When the factors affecting the MetS in the study were analyzed by logistic regression analysis, it was found that one cm increase in waist

circumference (OR: 0.018 %95 CI: 0.010-0.250) was found to significantly increase the risk of MetS (Table 2).

ROC analysis was applied for preptin and AUC value was calculated as 0.810 (%95 CI: 0.72–0.90) with 45.67 (%95 CI:40.11-53.38) cut-off value. In the ROC analysis, preptin was obtained to predict the clinical severity in the MetS group with a sensitivity and specificity of 82.5 (%95 CI= 0.68-0.93), and 70.0% (%95 CI= 0.55-0.85), respectively (Figure 4).

4. Discussion

MetS is a complex disease including abdominal obesity with elevations in serum triglyceride and glucose levels, increased blood pressure and low HDL-C levels [15,16]. MetS is a multifactorial risk factor for various diseases such as atherosclerotic cardiovascular disease and T2DM [9,17]. In this context, preptin, a 34-amino acid peptide secreted by pancreatic β-cells alongside insulin, has been suggested to play a potential role in insulin secretion and glucose metabolism [7,8,18].

Previous studies have demonstrated that preptin may influence glucose-mediated insulin secretion. Buchanan et al. (2001) showed that preptin infusion into the isolated rat pancreas increased the second phase of glucose-mediated insulin secretion by 30%, while the infusion of anti-preptin antibodies decreased the first phase of insulin secretion by 29% and the second phase of insulin secretion by 26% [7]. Cheng et al. (2012) suggested that preptin could activate insulin-like growth factor 2 receptor (IGF2R), which is associated with the protein kinase C (PKC) / phospholipase C (PLC) pathway, to induce calcium-dependent insulin secretion under high glucose conditions [8]. As the results of these studies indicate, the biological mechanism of preptin can be explained by the

activation of PLC and PKC pathways via IGF2R, leading to an increase in intracellular calcium levels. This mechanism suggests that preptin can be considered not only as a marker of glucose-mediated insulin release but also as an indicator of pancreatic β-cell function. Furthermore, an increase in preptin levels in conditions such as MetS or T2DM may represent an adaptive response to impaired glucose regulation. In this context, a more detailed examination of the molecular effects of preptin may provide important insights into the dysregulated insulin dynamics observed in metabolic disorders.

In this study, we measured serum preptin levels in patients with MetS and healthy control groups. Our findings revealed that serum preptin levels were notably elevated in the MetS group compared to the healthy controls. Our study findings suggest that, in addition to the elevated preptin levels observed, there were significant increases in clinical parameters such as triglycerides, cholesterol, LDL-C, HbA1c, and fasting glucose in the MetS group, which further support the role of preptin in lipid metabolism and glucose homeostasis. Preptin may have an effect that could enhance the role of insulin in glucose metabolism. Specifically, the strong positive correlation between preptin and HOMA-IR underscores its association with insulin resistance, suggesting that preptin could be a valuable marker for evaluating IR in MetS patients. Furthermore, the results of this study suggest that preptin could be involved in the pathophysiology of MetS.

Rahimi et al. (2019) investigated the effect of exercise on preptin levels in obese male adults with MetS on four different groups such as aerobic interval exercise (AIEX), resistance exercise (REX), concurrent aerobic interval and resistance exercise (CEX) and a non-exercise control group. They found that serum preptin levels after exercise significantly decreased in AIEX and CEX groups in comparison to the control [19]. However, the fact that the study was conducted solely on obese male individuals, along

with the limited evaluation of the direct relationship between preptin and metabolic parameters, restricts the ability to determine the causal relationship between preptin levels and MetS and limits the generalizability of the findings to the broader population.

In line with our findings, several studies have explored preptin levels in other conditions closely linked to MetS, including T2DM [9,10,20]. According to these studies, preptin levels were found to be significantly higher in patients compared to healthy controls. For example, Yang et al. (2009) compared plasma preptin levels of T2DM group, impaired glucose tolerance (IGT) group and healthy individuals and they showed that preptin levels were higher in patients with T2DM than IGT and healthy individuals. They also found that the plasma preptin level was positively correlated with diastolic blood pressure, triglyceride, total cholesterol, HbA1c and HOMA-IR index [9]. The cross-sectional design of this study, along with the fact that all participants were recruited from the same region, limits the ability to establish a causal relationship between elevated plasma preptin levels and T2DM, and restricts the generalizability of the findings to the wider population. Similarly, Rija et al. (2022) the levels of serum preptin, HbA1c, fasting blood glucose, urea and creatinine were investigated in women newly diagnosed with T2DM and it was found that they were significantly higher in the T2DM group than the control [20]. However, the fact that the study was conducted exclusively on female participants limits the generalizability of the findings to the broader population. The plasma preptin levels in T2DM patients were examined by Kalayci et al. (2019), who found that these patients had considerably higher plasma preptin levels than controls. They also demonstrated that although the levels of HDL-C were lower in the T2DM group compared to the control group, the levels of fasting glucose, HbA1c, insulin, HOMA-IR, total cholesterol, LDL-C, and triglycerides were higher in patients [10]. Similar to the results in our study, they

also determined that plasma preptin levels in the healthy control group and T2DM group showed a significant positive correlation with insulin, HOMA-IR, HbA1c and fasting glucose levels [10]. The study by Ozkan et al. (2013) investigated preptin levels based on body mass index (BMI) index and they found a positive correlation between BMI value and preptin levels. They also suggested that the increased IR in obesity may be associated with preptin level [21]. In another study, it was demonstrated that serum preptin levels were found to be significantly higher in the IR patients. In the same study, it was determined that serum preptin levels were positively correlated with HOMA-IR, fasting insulin level, body mass index and triglyceride level [22]. We also determined that serum preptin levels in the MetS patients had a significant positive correlation with insulin and HOMA-IR levels.

Bu et al. (2012) studied preptin levels in 63 patients with polycystic ovary syndrome (PCOS) [33 women with normal glucose tolerance (NGT) and 30 women with IGT], and 63 non-PCOS groups [35 women with NGT and 28 women with IGT] and they detected that preptin levels were higher in the PCOS group than the non-PCOS group [23]. They also reported that serum preptin levels were higher in IGT women compared to NGT women in the PCOS and non-PCOS groups [23]. Similarly, Mierzwicka et al. (2018) found that the preptin levels of the PCOS group were higher than the control group [24]. Based on all these data, the secretion of preptin in response to glucose and its enhancement of insulin secretion via the IGF2R pathway suggests that preptin may serve as a physiological regulator of pancreatic β-cell function. Furthermore, the positive correlation between the increase in preptin levels and insulin resistance observed in our study and other studies implies that preptin could serve as an early indicator of impaired insulin dynamics in the early stages of metabolic diseases, particularly before the onset

of T2DM. Therefore, preptin may play a critical role in the early diagnosis of MetS and could serve as a potential therapeutic target for the management of insulin resistance and its associated complications.

However, there are some limitations to our study. First, it was conducted in a single center, which may limit the generalizability of the findings. In addition, the relatively small sample size may reduce the statistical power and generalizability of the results. Furthermore, individual and environmental factors that could potentially influence preptin levels such as dietary habits, medication use, physical activity levels, age, ethnicity, and genetic predisposition could not be fully controlled. The inability to account for these variables may have impacted the outcomes. Moreover, due to the limited number of studies in the literature examining preptin levels specifically in patients with MetS, comparing our findings with those of other studies remains challenging. In future research, these limitations could be addressed by including larger sample sizes and utilizing multicenter study designs.

In conclusion, the findings of this study demonstrate increased serum preptin levels in patients with MetS, suggesting that preptin may play a role in the pathogenesis of MetS, and this condition may be associated with insulin and insulin resistance. Preptin, a peptide associated with insulin secretion, could be a potential target for the early diagnosis and treatment strategies of MetS. However, more comprehensive clinical studies are needed to fully understand the role of preptin in MetS and the underlying mechanisms involved. Future research should investigate the relationship between preptin and insulin resistance, obesity, inflammation, and other metabolic markers, as well as the impact of preptin levels on long-term metabolic outcomes. Furthermore, in future studies, evaluating C-peptide levels—both as a direct marker of insulin production and to allow for a more

detailed assessment of pancreatic β-cell function—alongside preptin may contribute to a better understanding of the pathophysiology of MetS. In addition, it is essential to evaluate how factors such as age, sex, ethnicity, and genetic predisposition influence preptin levels. Advanced studies addressing these aspects will help clarify the potential role of preptin in early diagnosis and therapeutic strategies for MetS.

Conflicts of Interest

The authors declare that they have no competing interests

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Tables and Figures

Tables

Table 1. Demographic and biochemical properties of the MetS and the control groups.

Variables	MetS (n=40) Mean±SD	Control (n=40) Mean±SD	p*	Cohen's d (effect size)
Age (years)	36.5±9.5	33.6±8.1	0,156	-0.3039
Height (cm)	167.8±9.8	171.1±9.5	0,119	0.3383
Weight (kg)	88.8±9.3	73.6±11.2	***<0,001***	-1.4251
LDL-C (mg/dL)	129.9±26.3	110.6±28.1	**0,002**	-0.6885
HDL-C (mg/dL)	50.8±8.9	52.3±10.6	0,49	0.1298
Variables	**Median (Q1-Q3)**	**Median (Q1-Q3)**	**p****	
Waist Circumference (cm)	97.5 (92.2-102)	80 (77.2-92)	***<0,001***	-1.5926
Triglyceride (mg/dL)	148.5 (103.2-207.2)	97.5 (67.2-126.7)	***<0,001***	-0.9182
Cholesterol (mg/dL)	200 (191.2-243)	175 (161-204.7)	***<0,001***	-0.8192
Fasting Glucose (mg/dL)	105 (101-116.5)	93 (89.2-98.7)	***<0,001***	-0.8184
HbA1c (%)	5.4 (5.2-5.8)	5.2 (5-5.4)	***<0,001***	-0.5959
Insulin (μIU/mL)	12 (9.2-16.5)	7 (5.2-8.7)	***<0,001***	-1.4348
HOMA-IR	3.2 (2.4-4.3)	1.6 (1.2-2)	***<0,001***	-1.6079
Preptin (pg/mL)	53.7 (46.5-60.7)	40.7 (36.2-49.6)	***<0,001***	-1.0824

* Independent Samples T test **Mann-Whintney U test
MetS: Metabolic Syndrome; HbA1c: Hemoglobin A1c; HDL-C: High-density lipoprotein Cholesterol; HOMA-IR: Homeostatic model assessment of insulin resistance; LDL-C: Low-density Lipoprotein Cholesterol

Table 2. Investigation of risk factors affecting MetS by logistic regression analysis*

Variables	Odds Ratio	95% CI	p-value
Waist circumference	1 0.018	0.010-0.250	**0.001**

CI: Confidence Interval, p: Logistic regression analysis enter model *No adjustments were made for potential confounding variables.

Figures

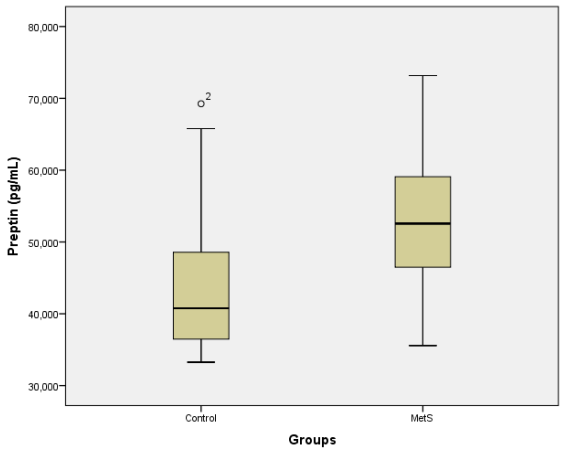


Figure 1. Serum preptin levels of the MetS (n=40) and control (n=40) groups.

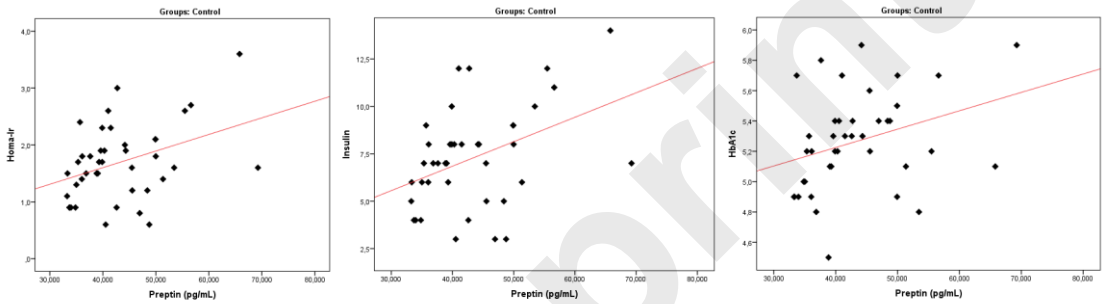


Figure 2. Serum preptin level correlation with HOMA-IR, HbA1c, and insulin in the control group. HbA1c: Hemoglobin A1c; HOMA-IR: Homeostatic model assessment of insulin resistance.

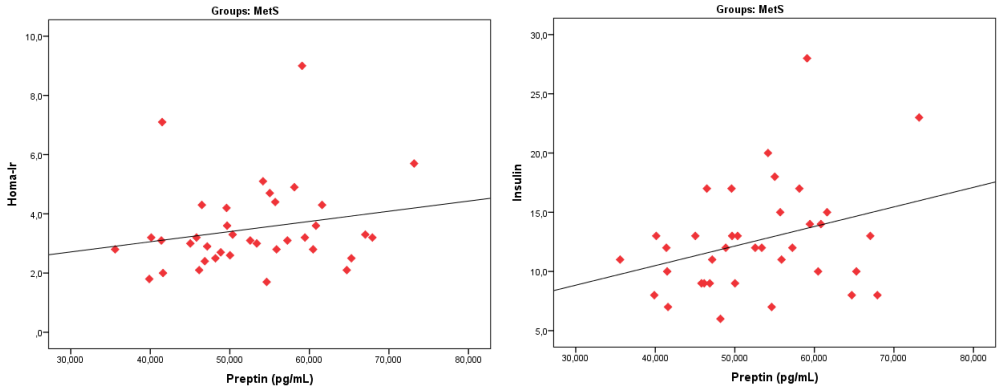


Figure 3. Serum preptin level correlation with insulin and HOMA-IR in the MetS group. HOMA-IR: Homeostatic model assessment of insulin resistance.

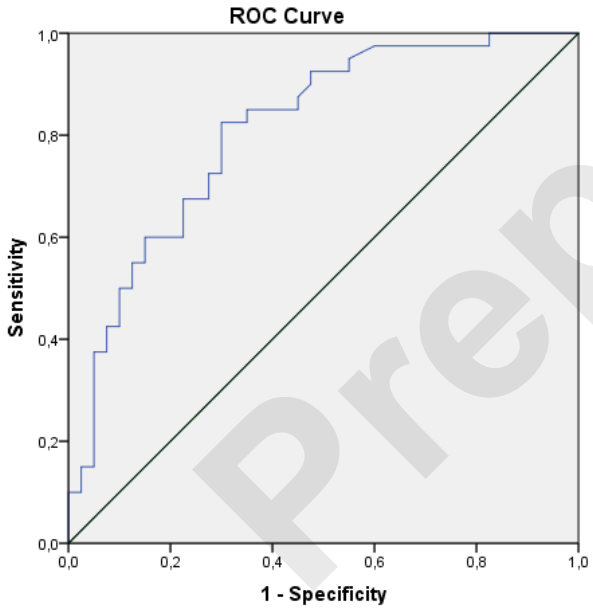


Figure 4. Receiving operating characteristic (ROC) analysis for preptin.

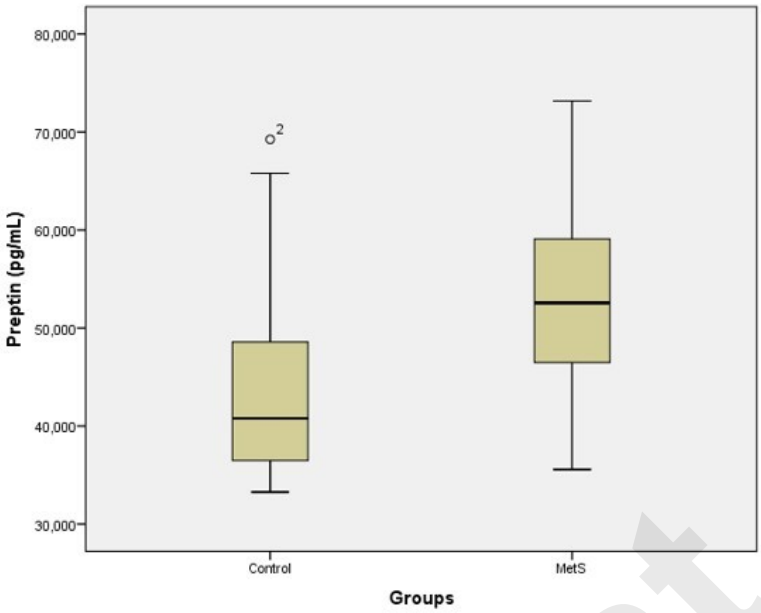


Figure 1. Serum preptin levels of the MetS (n=40) and control (n=40) groups.

Tables

Table 1. Demographic and biochemical properties of the MetS and the control groups.

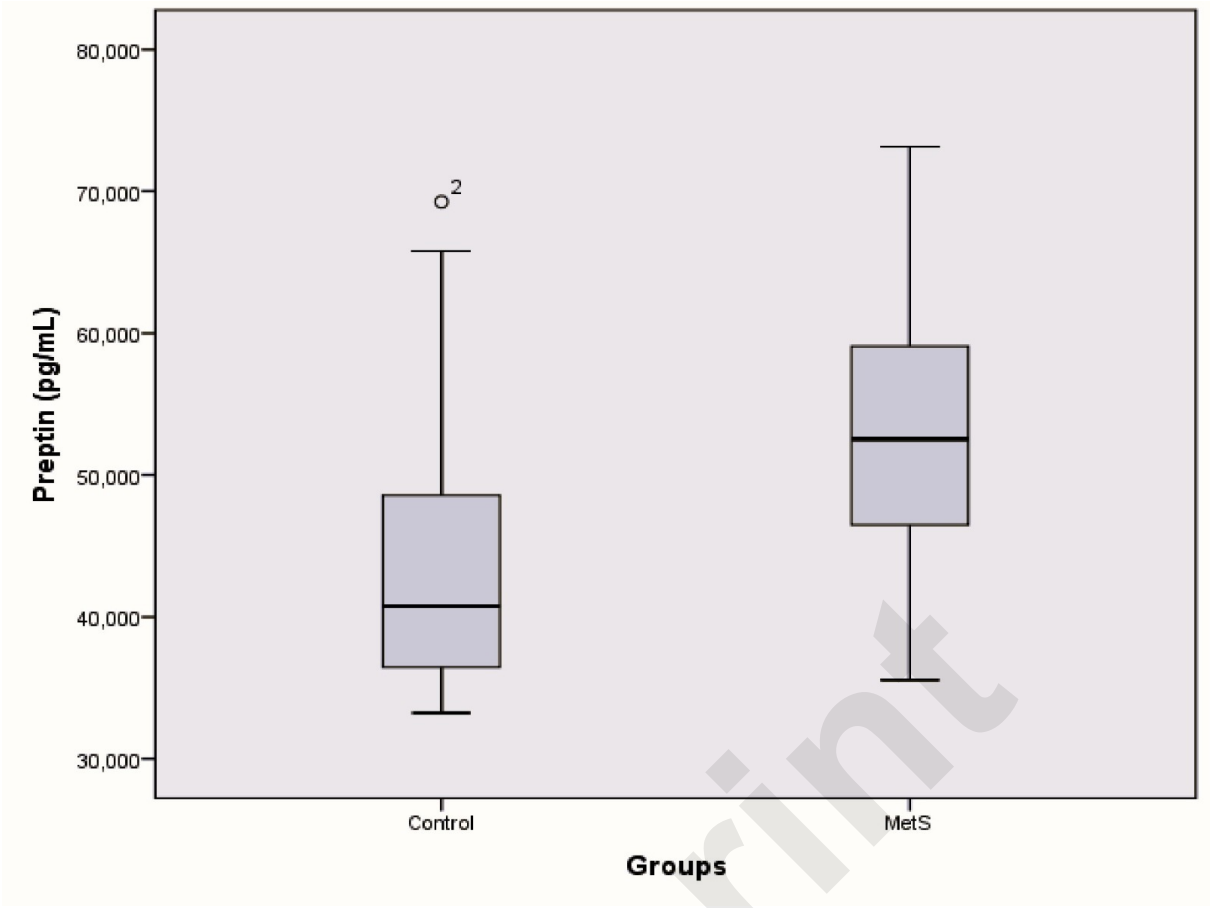
Variables	MetS (n=40) Mean±SD	Control (n=40) Mean±SD	p*	Cohen's d (effect size)
Age (years)	36.5±9.5	33.6±8.1	0,156	-0.3039
Height (cm)	167.8±9.8	171.1±9.5	0,119	0.3383
Weight (kg)	88.8±9.3	73.6±11.2	***<0,001***	-1.4251
LDL-C (mg/dL)	129.9±26.3	110.6±28.1	**0,002**	-0.6885
HDL-C (mg/dL)	50.8±8.9	52.3±10.6	0,49	0.1298
Variables	**Median (Q1-Q3)**	**Median (Q1-Q3)**	**p****	
Waist Circumference (cm)	97.5 (92.2-102)	80 (77.2-92)	***<0,001***	-1.5926
Triglyceride (mg/dL)	148.5 (103.2-207.2)	97.5 (67.2-126.7)	***<0,001***	-0.9182
Cholesterol (mg/dL)	200 (191.2-243)	175 (161-204.7)	***<0,001***	-0.8192
Fasting Glucose (mg/dL)	105 (101-116.5)	93 (89.2-98.7)	***<0,001***	-0.8184
HbA1c (%)	5.4 (5.2-5.8)	5.2 (5-5.4)	***<0,001***	-0.5959
Insulin (μIU/mL)	12 (9.2-16.5)	7 (5.2-8.7)	***<0,001***	-1.4348
HOMA-IR	3.2 (2.4-4.3)	1.6 (1.2-2)	***<0,001***	-1.6079
Preptin (pg/mL)	53.7 (46.5-60.7)	40.7 (36.2-49.6)	***<0,001***	-1.0824

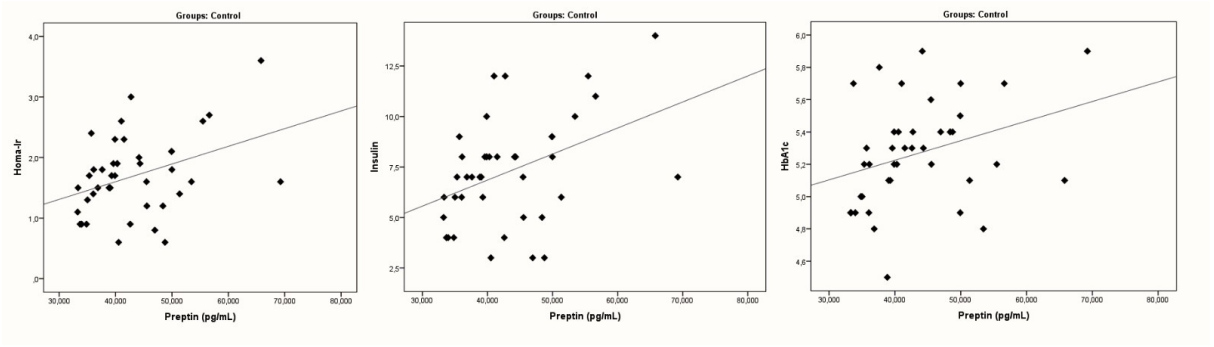
* Independent Samples T test **Mann-Whintney U test
MetS: Metabolic Syndrome; HbA1c: Hemoglobin A1c; HDL-C: High-density lipoprotein Cholesterol; HOMA-IR: Homeostatic model assessment of insulin resistance; LDL-C: Low-density Lipoprotein Cholesterol

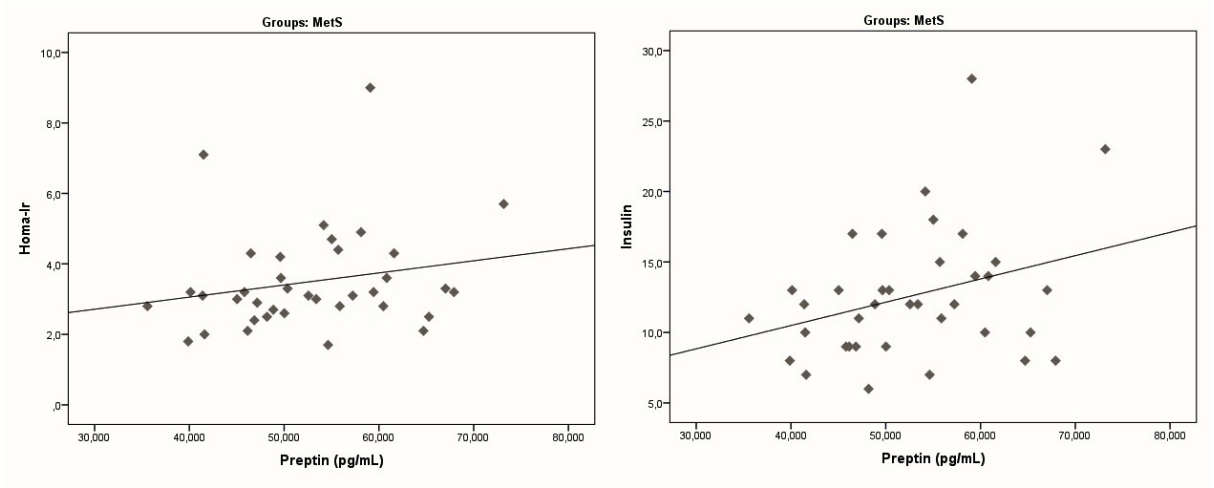
Table 2. Investigation of risk factors affecting MetS by logistic regression analysis

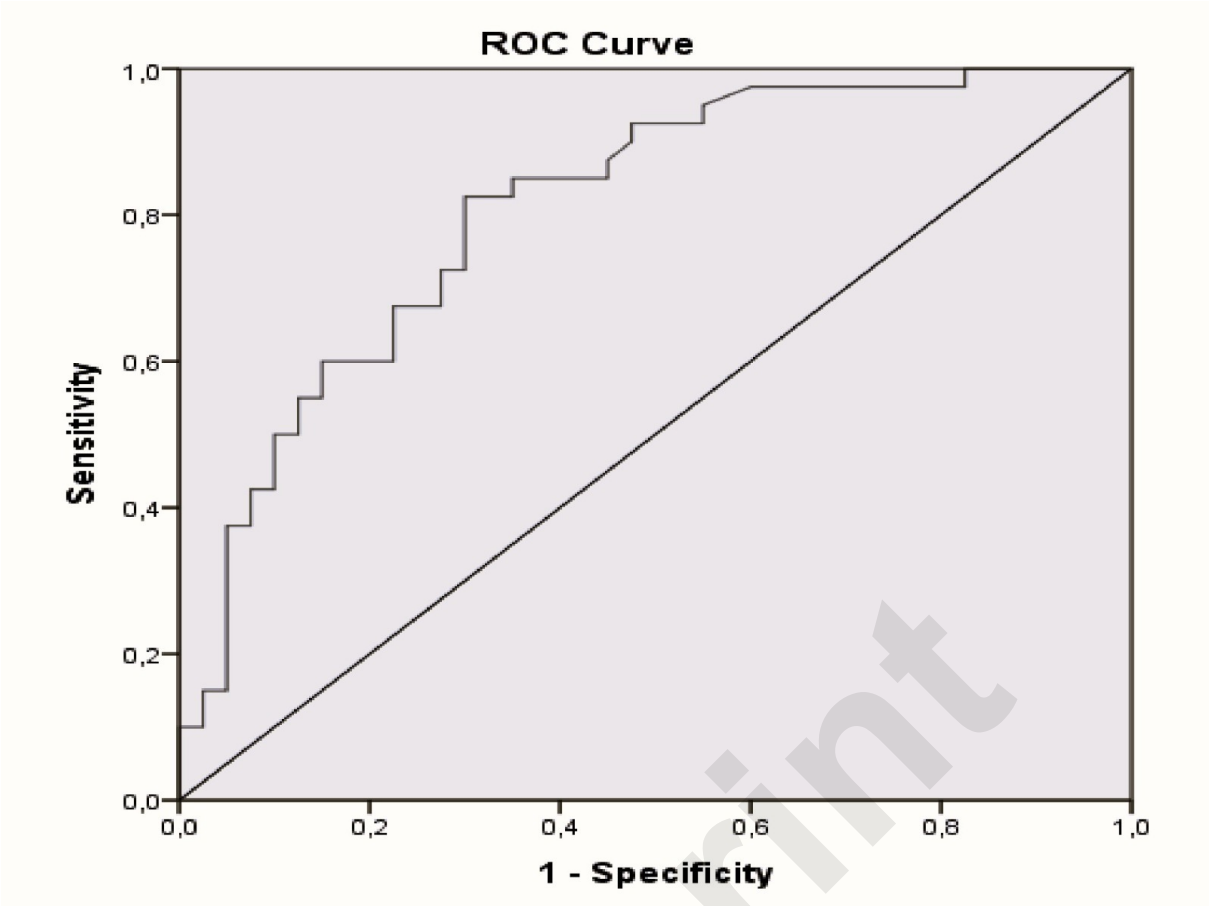
Variables	Odds Ratio	95% CI	p-value
Waist circumference	1 0.018	0.010-0.250	**0.001**

CI: Confidence Interval, p: Logistic regression analysis enter model *No adjustments were made for potential confounding variables.


80,000
70,000
60,000
50,000
40,000
30,000
Preptin (pg/mL)
2
Control
MetS
Groups


Groups: Control
Homa-Ir
Preptin (pg/mL)
Groups: Control
Insulin
Preptin (pg/mL)
Groups: Control
HbA1c
Preptin (pg/mL)


Groups: MetS
Homa-Ir
10,0
8,0
6,0
4,0
2,0
,0
30,000
40,000
50,000
60,000
70,000
80,000
Preptin (pg/mL)
Groups: MetS
Insulin
30,0
25,0
20,0
15,0
10,0
5,0
30,000
40,000
50,000
60,000
70,000
80,000
Preptin (pg/mL)


ROC Curve
Sensitivity
1,0
0,8
0,6
0,4
0,2
0,0
0,0
0,2
0,4
0,6
0,8
1,0
1 - Specificity