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Association of Adiponectin and Leptin Levels with Endothelial Dysfunction in Patients with Metabolic Syndrome: A Cross-Sectional Clinical Study

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ABSTRACT

Background: Metabolic syndrome (MetS) is defined by a group of cardiometabolic disorders such as central obesity, insulin resistance, dyslipidemia, and hypertension, all of which lead to an increased cardiovascular risk. One early indicator of vascular injury in MetS is endothelial dysfunction. Two important adipokines released by adipose tissue, adiponectin and leptin, may affect endothelial function via pro- and anti-inflammatory pathways, respectively.

Objective: To assess the association between circulating adiponectin and leptin levels and endothelial dysfunction in adult patients diagnosed with metabolic syndrome.

Methods: This cross-sectional research was carried out between January 2023 and April 2024 at two tertiary care institutions in Pakistan. There were one hundred individuals with metabolic syndrome in all. Adiponectin and leptin levels in blood were measured using ELISA, and endothelial function was evaluated using flow-mediated dilatation (FMD) of the brachial artery. Endothelial dysfunction was indicated by a flow-mediated dilatation (FMD) of less than 6%. Associations were found using statistical techniques such as t-tests, Pearson's correlation, and multivariate regression.

Results: Patients exhibiting endothelial dysfunction (64%) had markedly reduced adiponectin levels (3.8 ± 1.1 µg/mL) and elevated leptin levels (24.7 ± 5.1 ng/mL) in comparison to those with normal endothelial function. Adiponectin had a favorable association with FMD ($r = 0.54$, $p < 0.001$), but leptin showed a significant negative correlation ($r = -0.48$, $p < 0.001$). Both adipokines continued to serve as independent predictors of endothelial function after adjustments for metabolic variables.

Conclusion: Adiponectin insufficiency and hyperleptinemia are significantly correlated with compromised endothelial function in metabolic syndrome. These adipokines may function as early indicators and therapeutic targets for the mitigation of cardiovascular risk.

Keywords: Adiponectin, Leptin, Metabolic Syndrome, Endothelial Dysfunction, Flow-Mediated Dilation

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INTRODUCTION

Metabolic syndrome (MetS) is a worldwide health issue caused by a group of interrelated metabolic disorders such as central obesity, insulin resistance, dyslipidemia and hypertension [1]. People that have this clustering of risk factors are far more likely to develop atherosclerosis, type 2 diabetes mellitus (T2DM) and cardiovascular events such myocardial infarction and stroke [2,3]. Endothelial dysfunction or an early reduction in the bioavailability of endothelial nitric oxide (NO) is an important underlying

mechanism that links MetS and cardiovascular disease. This brings about a decreased vasodilation, vascular inflammation and atherogenesis [4].

In addition to being a store of energy, adipose tissue, especially of the visceral compartment, is a dynamic endocrine gland secreting a variety of bioactive chemicals called adipokines [5]. Two of them, leptin and adiponectin, have been found to be important modulators of vascular homeostasis. A 30 kDa collagen-like protein named adiponectin stimulates endothelial nitric oxide synthase (eNOS) and decreases oxidative stress and vascular inflammation to have insulin-sensitizing, anti-inflammatory, and vasoprotective effects [6,7]. On the other hand, pro-inflammatory and pro-atherogenic processes such as endothelial activation, oxidative stress, and sympathetic overactivity have been linked to leptin, a 16-kDa hormone that is mainly engaged in energy control and satiety signaling [8,9].

The development from metabolic disturbances to cardiovascular dysfunction may be mediated by the dysregulation of these adipokine levels, which is often seen in individuals with MetS and is characterized by hypoadiponectinemia and hyperleptinemia [10,11]. Even while each of these adipokines has been linked to endothelium health in a number of separate investigations, integrative clinical data connecting their combined profiles to functional vascular outcomes is still needed, particularly in South Asian communities where the incidence of MetS is on the rise [12].

Finding early, non-invasive biomarkers that indicate vascular impairment is becoming more and more important due to the public health burden of cardiovascular disease caused by MetS. The brachial artery's flow-mediated dilatation (FMD), a proxy for endothelial function, has been utilized extensively to evaluate subclinical vascular injury and shows an inverse relationship with unfavorable metabolic states [13]. Examining the relationship between adiponectin and leptin and FMD in MetS may help identify new treatment targets and enhance risk assessment. Thus, the current study intends to investigate the relationship between endothelial dysfunction, as determined by FMD, and circulating levels of adiponectin and leptin in adult patients with MetS. This will make it clearer whether vascular dysfunction in this high-risk group is substantially caused by adipokine imbalance.

MATERIALS AND METHODS

This cross-sectional observational study was carried out from January 2023 to April 2024 in two tertiary care centers of Pakistan. The aim of this study was to determine the association between circulating leptin and adiponectin levels and endothelial dysfunction in subjects with metabolic syndrome. Using non-probability sequential sampling method, a total of one hundred participants between the ages of thirty and sixty-five were selected.

The International Diabetes Federation (IDF) criteria for metabolic syndrome which includes central obesity and at least two metabolic abnormalities were used to determine eligibility. Central obesity is defined as waist circumference of 80 cm and 90 cm or more for males and females respectively. Other criteria included high triglycerides (>=150 mg/dL), increased fasting blood glucose (>=100 mg/dL), hypertension (>=130/85 mmHg) or low HDL cholesterol (<40 mg/dL in males and <50 mg/dL in women).

Cancer, serious liver or renal disease, cardiovascular disease, chronic inflammatory or autoimmune disorders, and individuals who had used steroids, statins, or immune-suppressive medications in the past two months were not eligible for inclusion in the study. Written informed consent was obtained from all participants before the experiment began and ethical approval was provided by the institutional review boards of the two participating sites (ERC/18A/01/2023). A comprehensive clinical evaluation included taking vital signs and a medical history. The body mass index (BMI) was calculated using recognized methods after the waist circumference was measured midway between the iliac crest and the lower ribs.A manually calibrated sphygmomanometer was used to monitor blood pressure at rest five minutes after sitting.Blood samples were obtained after a 10- to 12-hour overnight fast. Triglycerides, HDL, LDL, total cholesterol, and fasting plasma glucose were measured using an automated biochemistry analyzer in the lab. Chemiluminescent immunoassay was used to measure insulin levels while fasting.

Insulin resistance was measured using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) rather to the traditional approach of dividing fasting insulin (μIU/mL) by fasting glucose (mg/dL) by 405). Utilizing commercially available enzyme-linked immunosorbent assay (ELISA) kits, the serum levels of leptin and adiponectin were determined. For accuracy, each sample was inspected twice.Brachial artery flow-mediated dilatation (FMD), a non-invasive method that replicates nitric oxide-dependent vascular relaxation, was used to assess endothelial function. High-resolution Doppler ultrasonography using a linear array transducer operating at 7–12 MHz was used for this procedure. Each participant's blood pressure cuff was elevated to 50 mmHg over their systolic level for five minutes in order to induce occlusion after a resting imaging of the brachial arteries. Post-occlusion pictures were obtained 60 seconds after the cuff was removed.

The percentage increase in artery diameter from the baseline measurement was used to calculate FMD. Less than 6% was considered to be a sign of impaired endothelial function. IBM SPSS version 26.0 was used for all statistical analyses. While frequencies and percentages were used to depict categorical variables, averages and standard deviations were used to represent continuous

data. Using independent sample t-tests for quantitative measures and chi-square testing for categorical data, groups with and without endothelial dysfunction were compared. The associations between the levels of FMD, leptin, and adiponectin were assessed using Pearson correlation coefficients. Potential confounding factors, such as age, gender, BMI, insulin resistance, and lipid profile, were investigated using multivariable linear regression. In the research, a two-tailed p-value of less than 0.05 was considered statistically significant.

RESULTS

The research examined 100 adult patients with metabolic syndrome to determine the relationship between blood adiponectin and leptin levels and endothelial function. There were 45 men and 55 women among the participants, with an average age of 51.6 ± 7.9 years. The incidence of certain metabolic abnormalities was recorded in order to calculate the clinical burden of metabolic syndrome features in the population being studied. Table 1 displays the subjects' initial clinical and demographic information.

The mean waist circumference was 99.2 ± 6.7 cm, and the overall mean BMI was 30.8 ± 4.5 kg/m², indicating widespread central obesity. The majority of patients (72%) had hypertension, and 66% had fasting blood glucose levels ≥100 mg/dL. The diastolic and systolic blood pressure readings were 84.9 ± 8.3 mmHg and 137.5 ± 14.1 mmHg, respectively. The mean fasting glucose was 113.7 ± 23.6 mg/dL, and the mean HOMA-IR was 3.3 ± 1.1, indicating a significant prevalence of insulin resistance. The lipid profile showed that 68% of the participants had low HDL levels and 74% had hypertriglyceridemia.

Flow-mediated dilatation (FMD) was used to assess endothelial function. The FMD in the general study population was 5.3 +- 2.1%. Sixty-four percent of the patients had endothelial dysfunction and 36 percent retained endothelium function, based on a cut-off value of less than 6 percent. As shown in Table 2, compared with the placebo group, the contents of adipokines and the metabolic parameters were statistically significantly different in the two groups.

The mean serum adiponectin was significantly lower (3.8 +- 1.1 ug/mL) in the patients with endothelial dysfunction than in the people with normal endothelial function (6.3 +- 1.4 ug/mL) ($p < 0.001$). Conversely, the dysfunctional group had significantly greater leptin levels (24.7 ± 5.1 ng/mL) than the normal group (15.9 ± 4.6 ng/mL), which was likewise statistically significant ($p < 0.001$). With an average of 4.1 ± 1.0% vs 7.9 ± 1.2% in the group with intact function, FMD values were significantly lower in the group with decreased vascular function ($p < 0.001$). A more severe metabolic profile was also indicated by the higher BMI, fasting insulin levels, and HOMA-IR scores of people with endothelial impairment (Table 2).

The findings point to a consistent trend in which the anthropometric, glycemic, and adipokine profiles of individuals with endothelial dysfunction were poorer. Notably, there was a substantial correlation between reduced vascular reactivity and high leptin and low adiponectin levels. To learn more about the linear link between adipokine levels and FMD, correlation analyses were conducted. Serum adiponectin and FMD showed a considerable positive connection ($r = 0.54$, $p < 0.001$), whereas leptin and FMD showed a strong negative correlation ($r = -0.48$, $p < 0.001$), as shown in Table 3. Additionally, there was an inverse relationship between HOMA-IR and FMD ($r = -0.41$, $p = 0.002$), indicating that insulin resistance may be partially responsible for the vascular damage seen in metabolic syndrome.

FMD was used as the dependent variable in a multiple linear regression analysis to find independent predictors of endothelial function. Both adiponectin and leptin were still significant after age, gender, BMI, blood pressure, insulin resistance, and lipid factors were taken into account. Leptin was shown to be a negative predictor of FMD ($\beta = -0.35$, $p = 0.003$), but adiponectin was found to be a positive independent predictor ($\beta = 0.39$, $p = 0.001$). In addition, HOMA-IR independently accounted for variability in FMD (beta = -0.30, $p = 0.007$), indicating that the combination of these three signs may account for a substantial amount of vascular dysfunction in persons with metabolic syndrome.

Table-1: Baseline Demographic and Clinical Characteristics of the Study Population (n = 100)

Variable	Mean ± SD / n (%)
Age (years)	51.6 ± 7.9
Gender (Male / Female)	45 (45%) / 55 (55%)
BMI (kg/m²)	30.8 ± 4.5
Waist Circumference (cm)	99.2 ± 6.7
Systolic Blood Pressure (mmHg)	137.5 ± 14.1
Diastolic Blood Pressure (mmHg)	84.9 ± 8.3
Fasting Plasma Glucose (mg/dL)	113.7 ± 23.6
HOMA-IR	3.3 ± 1.1
HDL Cholesterol (mg/dL)	38.5 ± 9.4
Triglycerides (mg/dL)	184.3 ± 42.7
Central Obesity	100 (100%)
Elevated BP	72 (72%)
Elevated Fasting Glucose	66 (66%)
Low HDL Cholesterol	68 (68%)
Hypertriglyceridemia	74 (74%)

Table-2: Comparison Between Patients With and Without Endothelial Dysfunction (Based on FMD <6%)

Parameter	Endothelial Dysfunction (n = 64)	No Dysfunction (n = 36)	p-value
BMI (kg/m²)	31.5 ± 4.3	29.2 ± 3.7	0.013
Waist Circumference (cm)	100.4 ± 6.1	96.8 ± 5.9	0.007
Systolic BP (mmHg)	139.6 ± 13.3	133.1 ± 12.7	0.026
Fasting Glucose (mg/dL)	117.6 ± 21.9	106.4 ± 20.1	0.011
Fasting Insulin (µIU/mL)	18.1 ± 5.3	13.6 ± 4.1	<0.001
HOMA-IR	3.9 ± 1.1	2.9 ± 0.8	<0.001
Adiponectin (µg/mL)	3.8 ± 1.1	6.3 ± 1.4	<0.001
Leptin (ng/mL)	24.7 ± 5.1	15.9 ± 4.6	<0.001
FMD (%)	4.1 ± 1.0	7.9 ± 1.2	<0.001

Table-3: Pearson's Correlation Coefficients Between Flow-Mediated Dilation and Selected Variables

Variable	Correlation Coefficient (r)	p-value
Adiponectin	0.54	<0.001
Leptin	-0.48	<0.001
HOMA-IR	-0.41	0.002
BMI	-0.29	0.010
Fasting Insulin	-0.37	0.004
HDL Cholesterol	0.22	0.036

These results underscore the strong and independent association between the reduction of endothelial reactivity in metabolic syndrome and adipokine dysbalance. The evidence for the vascular involvement of leptin and adiponectin is enhanced by the constant association in regression modeling, correlation analysis, and unadjusted comparisons. These findings also suggest the potential therapeutic application of these biomarkers in the early evaluation and treatment of cardiovascular risk for a person with metabolic syndrome.

DISCUSSION

The current study examined the relationship between endothelial function and adiponectin and leptin levels in people with metabolic syndrome (MetS). The major conclusions were that, when compared to those with intact endothelial function, the concentrations of leptin in blood were much greater and the concentrations of adiponectin in blood were much lower in the people with endothelial dysfunction. Additionally, even after taking into consideration confounding factors such as insulin resistance, body mass index and cholesterol levels, both adipokines were found to be independent predictors of the health of the endothelium [5].

Adipocytes are the main source of adiponectin, an anti-inflammatory adipokine of vasoprotective properties, including enhancement of activity of endothelial nitric oxide synthase (eNOS), inhibition of inflammatory cytokines, and inhibition of oxidative stress [6]. The role of adiponectin in vascular health was also supported by this study which revealed that the subjects with low levels of adiponectin had significantly reduced flow-mediated dilatation (FMD). Similar results have been found in previous studies of an inverse link between adiponectin levels and evidence of endothelial dysfunction and arterial stiffness in high-risk groups with metabolic disorders [7,8].

By increasing oxidative stress, boosting vascular smooth muscle proliferation, and encouraging sympathetic activity, leptin, on the other hand, is known to have pro-atherogenic actions. Additionally, it has been shown to activate and malfunction endothelium cells via a variety of inflammatory pathways [9, 10]. In our investigation, those with endothelial dysfunction had substantially higher leptin levels, and this association remained even after controlling for insulin resistance and body mass index. These results are consistent with previous study showing that endothelial dysfunction is linked to increased leptin, especially in obese and insulin-resistant individuals [11,12].

The negative relationship between HOMA-IR and FMD provided more evidence for the connection between insulin resistance and vascular dysfunction. In insulin-resistant conditions, this signaling is disrupted, resulting in decreased nitric oxide bioavailability and endothelial dysfunction. Normally, insulin activates eNOS by activating the PI3K/Akt pathway, which promotes vasodilation [13]. The intricate pathophysiology of MetS, where endocrine, inflammatory, and metabolic problems intersect, is highlighted by this molecular connection between metabolic changes and vascular dysfunction [14,15].

These results are especially pertinent to South Asians, who, while having lower BMI thresholds than those in Western countries, are more likely to suffer from visceral adiposity, insulin resistance, and early-onset cardiovascular illnesses [16]. According to a number of studies, South Asians have a distinct cardiometabolic risk profile that is marked by a greater incidence of hyperleptinemia and hypoadiponectinemia, two conditions that accelerate vascular aging [17]. This finding is supported by our study, which also shows that adipokines are early indicators of vascular risk in this high-risk group [18].

Finding non-traditional biomarkers like leptin and adiponectin may be useful from a therapeutic perspective for cardiovascular risk classification, particularly in individuals with borderline conventional risk scores. It has

been shown that dietary changes, cardiovascular activity, and weight reduction are examples of lifestyle treatments that may alter adipokine levels and enhance endothelial function [19,20]. Although their long-term vascular advantages are still being studied, pharmacological treatments such as thiazolidinediones and GLP-1 receptor agonists may also have a favorable impact on adiponectin expression and vascular health [21].

This study's strengths include a well-defined MetS sample, a thorough correction for pertinent confounding variables, and a standardized evaluation of endothelial function using FMD. Nonetheless, it is important to recognize certain limits. Despite being statistically powered, the sample size limits subgroup analysis, and the cross-sectional design limits causal inference. Furthermore, additional vascular biomarkers that can provide further light on mechanistic processes, such as interleukin-6, tumor necrosis factor-alpha, and oxidative stress indicators, were excluded [22]. To determine if lowering adipokine levels results in long-lasting enhancements in endothelial function and cardiovascular outcomes, further longitudinal and interventional study is necessary [23].

In summary, our study offers strong evidence that endothelial dysfunction in individuals with metabolic syndrome is substantially correlated with lower adiponectin and higher leptin levels. These adipokines may be useful markers for the early identification and prevention of cardiovascular disease and seem to play a significant role as mediators in the cascade that connects metabolic imbalances with vascular damage [24,25].

CONCLUSION

In patients with metabolic syndrome, low adiponectin and high leptin levels were significantly associated with endothelial dysfunction. These adipokines may serve as early biomarkers of vascular risk and offer potential targets for preventive strategies. Monitoring and managing adipokine imbalance could play a vital role in reducing cardiovascular complications in this high-risk group.

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Authors' contributions: IA: Conceptualization, drafting, final approval. MM: Data collection, analysis. MFSM: Literature review, editing. NS: Supervision, critical revision.

Data Availability Statement: The data used in this study are available upon reasonable request from the corresponding author, subject to ethical and institutional guidelines.

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