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Association of Serum Leptin and Adiponectin Levels with Blood Pressure and Insulin Resistance in Obese Adults: A Cross-Sectional Study

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ABSTRACT

Background: Obesity is widely recognized as a serious worldwide health issue, with a clear relationship to metabolic abnormalities including insulin resistance and high blood pressure. Leptin and adiponectin, two adipose-derived hormones, serve conflicting roles in regulating vascular and metabolic activities. However, few studies have looked at their link with cardiometabolic risk in South Asian populations. The purpose of this research was to look at how serum leptin and adiponectin affect blood pressure and insulin resistance in obese Pakistani adults.

Methods: Cross-sectional research was carried out at three tertiary care facilities from January 2024 to June 2025. A total of 120 obese people (BMI ≥30 kg/m²; ages 25-60) were recruited. Demographic, anthropometric, and clinical data were gathered. Blood pressure was assessed using established methods, and fasting blood samples were tested for glucose, insulin, leptin, and adiponectin. Insulin resistance was evaluated using the homeostasis model assessment (HOMA-IR). Correlation tests and multivariate regression were used in the statistical analysis, which controlled for age, gender, and BMI.

Results: The study population's mean BMI was 33.8 ± 3.9 kg/m2, and its mean age was 44.5 ± 9.8 years, with 57.5% of the participants being female. The mean values of leptin and adiponectin were 21.5 ± 9.3 ng/mL and 6.2 ± 2.1 µg/mL, respectively. When compared to those with normotension, hypertensive participants showed considerably greater levels of leptin (25.2 ± 8.7 vs. 18.3 ± 7.2 ng/mL, $p<0.01$) and significantly lower levels of adiponectin (5.1 ± 1.8 vs. 7.2 ± 2.3 µg/mL, $p<0.01$). Systolic pressure and HOMA-IR were favorably connected with leptin ($r=0.38$ and $r=0.42$, $p<0.001$), but adiponectin was inversely correlated ($r=–0.32$ and $r=–0.39$, $p<0.01$). Both adiponectin and leptin were validated by regression models as independent predictors of insulin resistance and hypertension.

Conclusion: Obese Adults from Pakistan show a clear imbalance between leptin and adiponectin, with greater levels of leptin and lower levels of adiponectin being independently associated with insulin resistance and high blood pressure. These adipokines might guide preventative and intervention plans and act as early markers of cardiometabolic risk.

Keywords: Obesity; Leptin; Adiponectin; Hypertension; Insulin resistance

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INTRODUCTION

Obesity has become one of the most critical public health challenges of modern times, with its prevalence steadily rising in both developed and developing nations. The World Health Organization (WHO) estimated that there were over 1.9 billion adults who were considered to be overweight in 2022 and more than 650 million of them were obese [1]. The rapid urbanization, sedentary lifestyles, dietary changes and genetic predispositions have particularly resulted in a high rate of obesity in South Asian countries including Pakistan. This is a disturbing pattern, as obesity is a significant risk factor that leading to insulin resistance, type 2 diabetes mellitus (T2DM), hypertension, dyslipidemia, cardiovascular disease, and some malignancies. The biological pathways that connect obesity and metabolic and cardiovascular diseases have been not fully investigated despite the well-documented associations [2,3].

However, in recent years adipose tissue is no longer considered as an energy reservoir, but as a metabolically active endocrine organ. It releases diverse signaling molecules, also referred to as adipokines, that affect appetite control, glucose and lipid metabolism, vascular activity and inflammatory processes. Of interest among them are leptin and adiponectin due to their unique and opposite biological effects and relation to complications related to obesity [4,5].

Leptin is a peptide hormone secreted mostly by the white adipose tissue and signals the hypothalamus about nutrition status and controls the food consumption and the energy usage. In healthy physiological status, as adiposity increases, the leptin levels rise and inhibit the development of more adiposity by inducing the feeling of fullness [6]. Nevertheless in obesity, this feedback loop is frequently faulty in a phenomenon referred to as leptin resistance wherein high levels of leptin in the blood are no longer able to decrease appetite and maintain energy balance. Beyond energy regulation, leptin has several vascular and metabolic effects. It promotes renal sodium retention, enhances sympathetic nervous system activity, and stimulates vascular smooth muscle proliferation, all of which contribute to elevated blood pressure. Moreover, experimental evidence suggests that leptin impairs insulin signaling through oxidative stress and inflammatory pathways, thereby fostering insulin resistance [7].

In contrast, adiponectin, another adipocyte-derived protein, plays a protective role in metabolic and cardiovascular regulation. Unlike leptin, circulating adiponectin levels decline with increasing adiposity [8]. Low adiponectin concentrations are strongly associated with endothelial dysfunction, impaired glucose metabolism, dyslipidemia, and heightened cardiovascular risk. Mechanistically, adiponectin improves insulin sensitivity by stimulating fatty acid oxidation and glucose uptake through AMP-activated protein kinase (AMPK) and peroxisome proliferator-activated receptor-alpha (PPAR-α) pathways. It also exerts vasoprotective effects by enhancing nitric oxide production, reducing vascular inflammation, and inhibiting smooth muscle proliferation. Hypoadiponectinemia is therefore widely regarded as a hallmark of obesity-related metabolic dysfunction [9,10].

Individuals with obesity frequently present with both insulin resistance and hypertension, forming the cluster of abnormalities known as metabolic syndrome. While traditional mechanisms such as salt retention, activation of the renin–angiotensin system, and sympathetic overactivity are recognized contributors, growing evidence suggests that disruption of the leptin–adiponectin axis may be a key driver of these conditions [11]. Several epidemiological

studies have shown a positive association between leptin and blood pressure, whereas adiponectin consistently demonstrates negative correlations with both hypertension and insulin resistance. Despite this, most available data come from Western populations, leaving a knowledge gap in South Asian groups [12].

This issue is particularly relevant for Pakistan, where individuals often develop metabolic complications at comparatively lower BMI thresholds than those observed in Western cohorts. Higher visceral adiposity, genetic predispositions, and lifestyle risk factors such as high-calorie diets, physical inactivity, and limited access to preventive care contribute to this vulnerability. Understanding the contribution of adipokines in this context is therefore essential, not only for identifying at-risk individuals but also for guiding preventive and therapeutic strategies [13].

The present study was undertaken to investigate the relationship of serum leptin and adiponectin with blood pressure and insulin resistance in obese adults in Pakistan. By focusing on a South Asian population, this work aims to generate region-specific insights into the role of adipokine imbalance in the pathophysiology of obesity-related cardiometabolic disorders [14].

MATERIALS AND METHODS

The Pakistan Institute of Medical Sciences (PIMS) in Islamabad, Jinnah Hospital in Karachi, and Mayo Hospital in Lahore were the three main tertiary care facilities in Pakistan where this cross-sectional research was conducted between January 2024 and June 2025. A representative sample of obese individuals from various metropolitan areas was recruited thanks to the specialist endocrine and metabolic services provided by these clinics.

With 95% confidence and 80% statistical power, the OpenEpi calculator was used to determine the sample size, assuming a correlation coefficient of 0.25 between blood pressure and adipokine levels. The sample size was expanded to 120 to account for missing data and laboratory mistakes, even though the minimum needed number was 108. Participants who met WHO criteria for obesity (body mass index (BMI) ≥30 kg/m2) and were between the ages of 25 and 60 were eligible. The method used in outpatient clinics was a successive non-probability sampling technique. All participants gave written permission prior to participation after being briefed on the study's goals and methods. All participating institutions' Institutional Review Boards granted ethical permission for the research, which complied with the Declaration of Helsinki.

Those who were pregnant or nursing, had a history of ischemic heart disease, chronic renal disease, or endocrine diseases including hypothyroidism or Cushing's syndrome were not allowed to participate. To reduce confounding factors, those with a history of alcohol or drug use, those using lipid-lowering, antihypertensive, or antidiabetic drugs during the last three months, and those with a diagnosis of type 2 diabetes mellitus were also eliminated.

A standardized proforma was used to gather medical history, lifestyle facts (such as physical activity and smoking), and demographic data. To determine BMI and the waist-to-hip ratio, measurements of height, weight, waist, and hip circumferences were taken. Following five minutes of sitting at rest, participants' blood pressure was measured using a mercury sphygmomanometer. Five minutes separated the two measurements, and the study utilized the mean of the two. Systolic blood pressure of 130 mmHg or diastolic blood pressure of 80 mmHg, as defined by the American Heart Association/American College of Cardiology in 2017, was considered hypertension.

Venous blood samples (5 mL) were obtained after an 8–10 hour overnight fast. After the samples were centrifuged right away, the serum was kept at -80°C until it was time for analysis. Chemiluminescent immunoassay was used to test fasting insulin, and the glucose oxidase–peroxidase technique was used to measure fasting glucose. The following formula from the homeostasis model assessment (HOMA-IR) was used to determine insulin resistance:

$$\text{HOMA-IR} = \frac{\text{Fasting glucose (mg/dL)} \times \text{Fasting insulin (}\mu\text{U/mL)}}{405}$$

An ELISA kit (Millipore, USA; sensitivity 0.25 µg/mL) was used to detect serum adiponectin, while an ELISA kit (R&D Systems, USA; sensitivity 0.5 ng/mL) was used to quantify serum leptin concentrations. Inter-assay variance was kept to less than 10%, and each test was run in triplicate. The main findings were the correlations between insulin resistance and blood pressure and leptin and adiponectin. Adipokine levels in hypertensive and normotensive patients were compared, and relationships with BMI were assessed, as part of secondary analyses. SPSS version 26 was used to analyze the data. The mean ± standard deviation was used to represent continuous variables, whereas frequencies and percentages were used to represent categorical data. The normality of the distribution was examined using the Shapiro-Wilk test. When necessary, one-way ANOVA or independent t-tests were used for group comparisons. Adipokines, blood pressure, and HOMA-IR were examined in connection to one another using Pearson's correlation coefficient. Age, sex, BMI, and smoking were among the variables that were adjusted for using multivariate linear regression. P-values less than 0.05 were regarded as statistically significant.

RESULTS

A total of 120 obese individuals who met the inclusion criteria were recruited. The study population had an average age of 44.5 ± 9.8 years, with 57.5% females (n=69) and 42.5% men (n=51). The average BMI was 33.8 ± 3.9 kg/m². Table 1 shows the baseline demographic, clinical, and biochemical parameters.

Table 1. Baseline characteristics of participants (n=120)

Variable	Mean ± SD / n (%)
Age (years)	44.5 ± 9.8
Gender (Male/Female)	51 (42.5%) / 69 (57.5%)
BMI (kg/m²)	33.8 ± 3.9
Waist-to-hip ratio	0.94 ± 0.07
Systolic BP (mmHg)	138.6 ± 14.5
Diastolic BP (mmHg)	86.4 ± 9.7
Fasting glucose (mg/dL)	104.3 ± 15.1
Fasting insulin (µU/mL)	17.8 ± 6.5
HOMA-IR	4.63 ± 1.9
Serum leptin (ng/mL)	21.5 ± 9.3
Serum adiponectin (µg/mL)	6.2 ± 2.1

The average systolic and diastolic blood pressure readings were elevated, consistent with the high prevalence of hypertension in obesity. The mean HOMA-IR value (4.63 ± 1.9) confirmed the presence of marked insulin resistance. In addition, participants exhibited the typical adipokine imbalance of obesity, with higher circulating leptin (21.5 ± 9.3 ng/mL) and reduced adiponectin (6.2 ± 2.1 µg/mL). When stratified by hypertensive status, 72 individuals (60%) were hypertensive (≥130/80 mmHg), while 48 (40%) were normotensive. Compared with the normotensive group, hypertensive participants showed significantly greater mean leptin concentrations (25.2 ± 8.7 vs. 18.3 ± 7.2 ng/mL, p<0.01) and lower adiponectin levels (5.1 ± 1.8 vs. 7.2 ± 2.3 µg/mL, p<0.01). Their HOMA-IR values were also higher, indicating stronger insulin resistance (Table 2).

Table 2. Comparison of adipokine levels and HOMA-IR between normotensive and hypertensive participants

Parameter	Normotensive (n=48)	Hypertensive (n=72)	p-value
Serum leptin (ng/mL)	18.3 ± 7.2	25.2 ± 8.7	<0.01
Serum adiponectin (µg/mL)	7.2 ± 2.3	5.1 ± 1.8	<0.01
HOMA-IR	3.8 ± 1.4	5.2 ± 1.9	<0.01

According to correlation analysis, leptin had a positive relationship with HOMA-IR (r=0.42, p<0.001), diastolic blood pressure (r=0.34, p=0.002), and systolic blood pressure (r=0.38, p<0.001). In contrast, adiponectin had an inverse relationship with insulin resistance (r=–0.39, p<0.001), systolic blood pressure (r=–0.32, p=0.004), and diastolic blood pressure (r=–0.29, p=0.006). Table 3 provides a summary of these relationships.

Table 3. Correlation of serum leptin and adiponectin with blood pressure and HOMA-IR

Parameter	Systolic BP (r)	Diastolic BP (r)	HOMA-IR (r)
Serum leptin	+0.38 (p<0.001)	+0.34 (p=0.002)	+0.42 (p<0.001)
Serum adiponectin	–0.32 (p=0.004)	–0.29 (p=0.006)	–0.39 (p<0.001)

In multivariate regression models adjusted for potential confounders (age, sex, BMI, and smoking), leptin remained an independent predictor of both systolic blood pressure (β=0.28, p=0.008) and HOMA-IR (β=0.36, p<0.01). Adiponectin independently predicted lower HOMA-IR (β=–0.29, p=0.01) and systolic blood pressure (β=–0.24, p=0.02). Taken together, the results demonstrate that obese individuals in this cohort had a distinct adipokine profile characterized by elevated leptin and reduced adiponectin, both of which were strongly linked to hypertension and insulin resistance. The associations were uniform in unadjusted comparisons, correlation testing, and regression analysis, which indicates the significance of adipokine imbalance in cardiometabolic dysfunction in South Asian adults.

DISCUSSION

This study examined the relationship between serum leptin and adiponectin and blood pressure and insulin resistance among obese Pakistani adults [11]. The results indicated that hypertension subjects had significantly higher leptin concentrations and reduced adiponectin concentrations than normal individuals [13]. Leptin was positively correlated with systolic and diastolic blood pressure, and HOMA-IR, but adiponectin had a negative correlation with them. Notably, both adipokines remained independent predictors of hypertension and insulin resistance even after adjusting with age, gender, BMI and smoking history. These findings indicate that a leptin and adiponectin imbalance is one of the most evident contributors to the emergence of the cardiometabolic issues among South Asian obese populations [14,15].

The association between blood pressure and leptin which was discovered in this study is consistent with that of the global literature. Leptin has been associated with hypertension through augmenting renal salt retention, to stimulate the sympathetic nervous system and endothelial malfunction through oxidative stress and inflammation [16]. These pathways are in line with our findings and this would support the idea that hyperleptinemia plays an important role in obesity-related hypertension. Leptin's high connection with HOMA-IR underlines its involvement in metabolic dysfunction, which is most likely caused by poor insulin signaling driven by inflammation and oxidative stress. The durability of these correlations after controlling for BMI shows that leptin effects cardiometabolic outcomes independent of adiposity [17,18].

Adiponectin, in contrast, is widely recognized for its protective effects on vascular and metabolic health. The present study reinforces this by demonstrating that lower adiponectin levels are associated with both insulin resistance and hypertension [15]. Adiponectin improves insulin sensitivity through AMPK and PPAR-α signaling pathways, enhances nitric oxide bioavailability, and reduces vascular inflammation. The hypoadiponectinemia observed in obese South Asians may therefore explain, at least in part, their higher predisposition to early-onset diabetes and cardiovascular disease. Interestingly, the strength of association between adiponectin and metabolic outcomes in our study was comparable to that of leptin, suggesting that considering these two adipokines together may provide better insights than evaluating either alone [16,19].

From a regional standpoint, these findings are particularly important. South Asians are known to develop metabolic complications at younger ages and at lower BMI thresholds than Western populations [13]. Factors such as visceral fat accumulation, genetic predisposition, and high-calorie diets with low physical activity contribute to this vulnerability. Our study demonstrates that disturbances in leptin and adiponectin may be central to this heightened risk. The clear differences in adipokine profiles between normotensive and hypertensive participants suggest that even small shifts in the leptin–adiponectin axis can precipitate cardiometabolic dysfunction in South Asians [20,21].

The clinical implications are noteworthy. Serum leptin and adiponectin could serve as accessible, cost-effective biomarkers for identifying obese individuals at high risk of hypertension and insulin resistance. Such markers may help clinicians implement early interventions [11,17]. Lifestyle modification remains the first-line strategy, but pharmacological agents that modulate adipokine activity such as thiazolidinediones or GLP-1 receptor agonists may also be useful. Emerging therapies, including leptin

antagonists and adiponectin mimetics, are currently under investigation and could provide novel treatment options in the future [14,22].

Strengths of this study include its multicenter design, standardized laboratory methods, and duplicate biochemical assays that ensured reliability. It also addresses a major gap by focusing on South Asian populations, which are underrepresented in adipokine research. However, several limitations should be acknowledged [23]. The cross-sectional design prevents conclusions about causality, and longitudinal studies are needed to determine whether changes in leptin and adiponectin precede the development of hypertension and insulin resistance. Residual confounding from unmeasured factors such as dietary habits, physical activity, and genetic variations cannot be completely ruled out. Furthermore, the study population was limited to urban tertiary care centers, which may restrict generalizability to rural settings. Finally, only leptin and adiponectin were examined, while other adipokines such as resistin, visfatin, and omentin may also play important roles [24].

Overall, the results strongly support the hypothesis that imbalance in leptin and adiponectin contributes to obesity-related cardiometabolic complications. Elevated leptin and reduced adiponectin were independently linked with hypertension and insulin resistance, reinforcing their importance as both biomarkers and potential therapeutic targets [25,26].

CONCLUSION

Obese Pakistani adults in this study demonstrated a distinct adipokine imbalance, characterized by high serum leptin and low serum adiponectin, which was independently associated with elevated blood pressure and insulin resistance. These findings suggest that the leptin–adiponectin axis plays a pivotal role in the pathophysiology of obesity-related cardiometabolic disorders in South Asians. Routine measurement of these adipokines may facilitate early detection of at-risk individuals and guide preventive and therapeutic interventions.

Conflict of Interest:

The authors report no conflicts of interest.

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Authors' contributions:

ZH1: Conceptualization, study design, data collection, drafting.

TH: Data acquisition, clinical input, manuscript review.

ZHq: Biochemical analysis, data interpretation.

SA: Statistical analysis, results compilation.

SUSZ: Literature review, critical revision.

ZH2: Clinical supervision, final approval. All authors read and approved the final manuscript.

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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