

Comparative Analysis of Serum Leptin and Adiponectin Levels in Obese and Non-Obese Individuals and their Association with Cardiovascular Risk

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Cite this paper as: Muhammad Omer Aslam, Ayesha Anwar, Ammara Abid, Sana Akram, Wajahat Ullah Khan, Zahira Bashir (2025) Comparative Analysis of Serum Leptin and Adiponectin Levels in Obese and Non-Obese Individuals and their Association with Cardiovascular Risk. Journal of Neonatal Surgery, 14, (32s) 10873-10876

ABSTRACT

Background: Obesity is a major global health concern strongly associated with cardiovascular diseases (CVD). Adipokines, particularly leptin and adiponectin, play crucial roles in energy homeostasis, inflammation, and vascular function. Alterations in these biomarkers may contribute to increased cardiovascular risk.

Objective: To compare serum leptin and adiponectin levels in obese and non-obese individuals and evaluate their association with cardiovascular risk factors.

Methods: A comparative cross-sectional study was conducted on 120 participants (60 obese, 60 non-obese). Serum leptin and adiponectin were measured by ELISA. Cardiovascular parameters were assessed and analyzed using SPSS.

Results: Mean serum leptin levels were significantly higher in obese individuals (32.8 ± 8.6 ng/mL) compared to non-obese (11.4 ± 4.2 ng/mL; $p < 0.001$). Mean adiponectin levels were significantly lower in obese individuals (4.6 ± 1.5 µg/mL) compared to non-obese (9.8 ± 2.7 µg/mL; $p < 0.001$). Obese participants had higher BMI (31.9 ± 2.8 vs 22.6 ± 1.9 kg/m²), systolic BP (138.5 ± 12.4 vs 118.2 ± 9.6 mmHg), and LDL levels (142.6 ± 28.3 vs 104.7 ± 22.5 mg/dL). Leptin showed strong positive correlation with BMI ($r = 0.72$) and LDL ($r = 0.61$), while adiponectin showed negative correlation with BMI ($r = -0.68$) and positive correlation with HDL ($r = 0.55$) ($p < 0.001$).

Conclusion: Elevated leptin and reduced adiponectin are significantly associated with increased cardiovascular risk in obese individuals..

Keywords: Leptin, Adiponectin, Obesity, Cardiovascular Risk, Adipokines, Dyslipidemia

INTRODUCTION

Obesity has emerged as a major global public health challenge, contributing significantly to morbidity and mortality, particularly through its strong association with cardiovascular diseases (CVD) (1,2). The pathophysiology of obesity-related cardiovascular risk involves complex interactions between metabolic dysregulation, chronic low-grade inflammation, and hormonal imbalance (3,4).

Adipose tissue is now recognized as a dynamic endocrine organ that secretes a wide range of bioactive molecules collectively termed adipokines (5). Among these, leptin and adiponectin play pivotal roles in metabolic homeostasis and cardiovascular regulation. Leptin, primarily involved in appetite regulation and energy balance, is often elevated in obese individuals, resulting in leptin resistance and subsequent metabolic dysfunction (6,7). In contrast, adiponectin, which possesses anti-inflammatory, insulin-sensitizing, and anti-atherogenic properties, is typically reduced in obesity (8,9)..

The imbalance between leptin and adiponectin contributes to endothelial dysfunction, oxidative stress, insulin resistance, and progression of atherosclerosis (10–12). Emerging evidence suggests that these adipokines are closely linked with traditional cardiovascular risk factors such as dyslipidemia, hypertension, and impaired glucose metabolism (13,14). Therefore, understanding alterations in leptin and adiponectin levels may facilitate early identification of individuals at increased risk of cardiovascular disease.

This study aims to compare serum leptin and adiponectin levels in obese and non-obese individuals and evaluate their association with cardiovascular risk factors

MATERIALS AND METHODS

A comparative cross-sectional study was conducted at a tertiary care hospital to evaluate differences in adipokine levels between obese and non-obese individuals and their association with cardiovascular risk factors. The study was carried out over a defined period following approval from the institutional ethical review committee, and all procedures were performed in accordance with standard research guidelines. A total of 120 participants aged between 20 and 50 years were enrolled in the study using a non-probability convenient sampling technique. The participants were divided into two equal groups based on body mass index (BMI), with Group A comprising 60 obese individuals (BMI ≥ 30 kg/m²) and Group B including 60 non-obese individuals (BMI 18.5–24.9 kg/m²). Participants of both genders who were willing to participate and provided informed consent were included in the study. Individuals with a known history of cardiovascular disease, diabetes mellitus, chronic inflammatory conditions, or endocrine disorders were excluded, and pregnant women were also excluded to avoid hormonal influences on adipokine levels.

Detailed clinical and demographic data were obtained from all participants. Anthropometric measurements, including height and weight, were recorded using standardized protocols, and BMI was calculated accordingly. Blood pressure was measured using a calibrated sphygmomanometer under resting conditions. Venous blood samples were collected after an overnight fast of 10–12 hours to ensure accuracy of biochemical parameters. Serum leptin and adiponectin levels were measured using enzyme-linked immunosorbent assay (ELISA) kits according to the manufacturer's instructions. In addition, a complete lipid profile, including total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides, was assessed using standard laboratory techniques, and fasting blood glucose levels were also measured to evaluate metabolic status.

All collected data were entered and analyzed using SPSS version 25. Continuous variables were expressed as mean \pm standard deviation (SD). The independent t-test was applied to compare means between the two groups, while Pearson correlation analysis was used to determine the relationship between adipokines and cardiovascular risk parameters. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 120 participants were enrolled and equally distributed into obese (n=60) and non-obese (n=60) groups. The baseline characteristics of the study population are presented in Table 1. There was no statistically significant difference in mean age between the groups (36.2 \pm 8.1 vs 34.7 \pm 7.5 years; p=0.28), indicating baseline comparability. However, obese individuals exhibited significantly higher BMI (31.9 \pm 2.8 vs 22.6 \pm 1.9 kg/m²; p<0.001), systolic blood pressure (138.5 \pm 12.4 vs 118.2 \pm 9.6 mmHg; p<0.001), and diastolic blood pressure (88.7 \pm 8.3 vs 76.4 \pm 6.5 mmHg; p<0.001).

Biochemical parameters demonstrated a significantly adverse metabolic profile in obese participants, as shown in Table 2. Fasting blood glucose (108.3 \pm 14.6 vs 91.7 \pm 10.2 mg/dL), total cholesterol (212.5 \pm 34.2 vs 176.8 \pm 28.6 mg/dL), LDL cholesterol (142.6 \pm 28.3 vs 104.7 \pm 22.5 mg/dL), and triglycerides (186.2 \pm 45.7 vs 122.4 \pm 30.1 mg/dL) were significantly elevated in the obese group (all p<0.001). In contrast, HDL cholesterol levels were significantly lower in obese individuals (38.4 \pm 6.8 vs 51.6 \pm 8.2 mg/dL; p<0.001).

Adipokine analysis (Table 3) revealed a marked imbalance between the groups. Serum leptin levels were significantly higher in obese individuals (32.8 \pm 8.6 ng/mL) compared to non-obese individuals (11.4 \pm 4.2 ng/mL; p<0.001). Conversely, serum adiponectin levels were significantly reduced in the obese group (4.6 \pm 1.5 μ g/mL vs 9.8 \pm 2.7 μ g/mL; p<0.001).

Correlation analysis (Table 4) demonstrated that leptin exhibited a strong positive correlation with BMI (r=0.72), LDL cholesterol (r=0.61), and systolic blood pressure (r=0.58), along with a moderate negative correlation with HDL cholesterol (r=-0.44) (p<0.001 for all). In contrast, adiponectin showed a strong negative correlation with BMI (r=-0.68), LDL (r=-0.52), and systolic blood pressure (r=-0.49), while demonstrating a significant positive correlation with HDL cholesterol (r=0.55) (p<0.001).

The distribution of categorical cardiovascular risk factors is presented in Table 5. A significantly higher proportion of obese individuals were hypertensive (63.3% vs 15.0%), had elevated LDL levels (68.3% vs 20.0%), low HDL levels (60.0% vs 13.3%), and hypertriglyceridemia (65.0% vs 18.3%), further supporting the association between obesity, adipokine imbalance, and cardiovascular risk.

Table 1: Baseline Characteristics

Parameter	Obese (n=60) Mean \pm SD	Non-Obese (n=60) Mean \pm SD	p-value
Age (years)	36.2 \pm 8.1	34.7 \pm 7.5	0.28

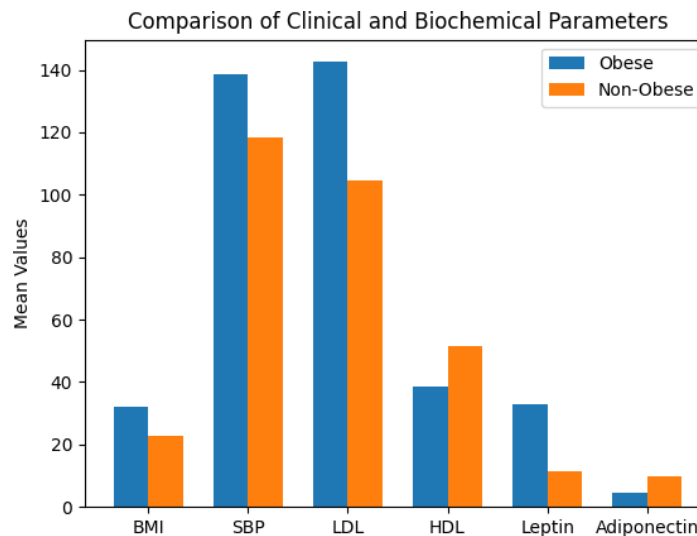
BMI (kg/m ²)	31.9 ± 2.8	22.6 ± 1.9	<0.001
SBP (mmHg)	138.5 ± 12.4	118.2 ± 9.6	<0.001
DBP (mmHg)	88.7 ± 8.3	76.4 ± 6.5	<0.001

Table 2: Biochemical Parameters

Parameter	Obese (Mean ± SD)	Non-Obese (Mean ± SD)	p-value
Fasting Glucose (mg/dL)	108.3 ± 14.6	91.7 ± 10.2	<0.001
Total Cholesterol (mg/dL)	212.5 ± 34.2	176.8 ± 28.6	<0.001
LDL (mg/dL)	142.6 ± 28.3	104.7 ± 22.5	<0.001
HDL (mg/dL)	38.4 ± 6.8	51.6 ± 8.2	<0.001
Triglycerides (mg/dL)	186.2 ± 45.7	122.4 ± 30.1	<0.001

Table 3: Correlation Analysis (r values)

Variable	Leptin (r)	Adiponectin (r)
BMI	+0.72	-0.68
LDL	+0.61	-0.52
HDL	-0.44	+0.55
SBP	+0.58	-0.49



DISCUSSION

The present study demonstrates a significant imbalance of adipokines in obese individuals, characterized by elevated leptin and reduced adiponectin levels. These findings are consistent with recent studies that highlight the crucial role of adipokines in the development and progression of cardiovascular disease (15,16).

Hyperleptinemia observed in obese individuals is associated with increased sympathetic nervous system activity, oxidative stress, and endothelial dysfunction, all of which contribute to the pathogenesis of atherosclerosis (17,18). Additionally, leptin has been shown to promote vascular inflammation and smooth muscle cell proliferation, further exacerbating cardiovascular risk (19).

Conversely, adiponectin exerts cardioprotective effects through multiple mechanisms, including enhancement of insulin sensitivity, inhibition of inflammatory pathways, and protection against endothelial injury (20,21). Reduced adiponectin levels in obesity are therefore linked with increased risk of metabolic syndrome and cardiovascular complications (22).

The observed inverse relationship between leptin and adiponectin in this study further supports their complementary roles in modulating metabolic and cardiovascular homeostasis (23). This imbalance may serve as a more sensitive indicator of cardiometabolic risk than either biomarker alone.

Overall, these findings suggest that monitoring serum leptin and adiponectin levels could provide valuable insights for early risk stratification and targeted preventive interventions in obese individuals (24,25).

CONCLUSION

This study demonstrates a significant imbalance in adipokine levels among obese individuals, characterized by elevated serum leptin and reduced adiponectin levels compared to non-obese individuals. These alterations are closely associated with adverse cardiometabolic parameters, including increased BMI, hypertension, and dyslipidemia. The findings suggest that leptin and adiponectin may serve as useful biomarkers for early identification of cardiovascular risk in obesity. Further large-scale studies are recommended to validate their clinical utility.

Disclaimer: None

Conflict of Interest: None

Funding: None

Authors' Contribution:

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Final Approval of Version: All authors approved the final version

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