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Original Research Article

ASSOCIATION OF SERUM ADIPONECTIN AND INSULIN RESISTANCE WITH METABOLIC SYNDROME: A CROSS-SECTIONAL STUDY IN A TERTIARY CARE CENTER

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ABSTRACT

Background: Metabolic syndrome (MetS) is a cluster of interrelated metabolic abnormalities that significantly increases the risk of type 2 diabetes mellitus and cardiovascular disease. Insulin resistance is central to its pathogenesis, and adiponectin, an insulin-sensitizing adipokine, has been implicated in metabolic regulation. However, data on the association between serum adiponectin levels, insulin resistance, and MetS in the Indian population remain limited.
Objectives: To assess the association of serum adiponectin levels and insulin resistance with metabolic syndrome and to evaluate the correlation of adiponectin with individual components of MetS.
Materials and Methods: This hospital-based cross-sectional study was conducted at a tertiary care center and included 80 adult participants, divided into a MetS group (n = 40) and a non-MetS group (n = 40). Metabolic syndrome was diagnosed using the International Diabetes Federation criteria. Anthropometric measurements, blood pressure, fasting plasma glucose, lipid profile, fasting insulin, and serum adiponectin levels were assessed. Insulin resistance was calculated using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR). Statistical analysis was performed using SPSS version 25.
Results: Participants with MetS were significantly older and had higher body mass index, waist circumference, blood pressure, fasting plasma glucose, triglycerides, fasting insulin, and HOMA-IR values, along with lower HDL cholesterol levels compared to the non-MetS group ($p < 0.01$). Serum adiponectin levels were significantly lower in the MetS group (4.8 ± 1.9 µg/mL) than in the non-MetS group (9.6 ± 3.1 µg/mL; $p < 0.01$). Adiponectin showed a significant negative correlation with HOMA-IR, waist circumference, and fasting plasma glucose, and a positive correlation with HDL cholesterol ($p < 0.01$).
Conclusion: Lower serum adiponectin levels and increased insulin resistance were strongly associated with metabolic syndrome. Serum adiponectin may serve as a useful biomarker for early identification of individuals at high risk for MetS and related cardiometabolic complications.
Keywords: Metabolic syndrome; adiponectin; insulin resistance; HOMA-IR; cardiometabolic risk

INTRODUCTION

Metabolic syndrome (MetS) is a complex clinical entity characterized by a clustering of interrelated metabolic abnormalities that substantially increase the risk of type 2 diabetes mellitus (T2DM), atherosclerotic cardiovascular disease, and premature mortality.[1] The core components of MetS include central (abdominal) obesity, insulin resistance, dyslipidemia—typically elevated triglycerides and reduced high-density lipoprotein (HDL) cholesterol—and hypertension. Owing to rapid

urbanization, lifestyle transitions, physical inactivity, and dietary changes, the prevalence of MetS has increased markedly worldwide, with a particularly steep rise observed in developing countries such as India.[2] This growing burden has positioned MetS as a major public health challenge, underscoring the need for early identification and effective preventive strategies.[3]

Insulin resistance is widely regarded as the central pathophysiological mechanism underlying MetS. It is characterized by a reduced biological response of peripheral tissues to insulin, resulting in impaired glucose uptake, compensatory hyperinsulinemia, dyslipidemia, endothelial dysfunction, and a pro-inflammatory milieu.[4,5] The development of insulin resistance is multifactorial, influenced by genetic predisposition, excess adiposity—especially visceral fat accumulation—chronic low-grade inflammation, oxidative stress, and hormonal dysregulation.[6] Given its pivotal role in linking obesity to cardiometabolic complications, insulin resistance remains a key focus of ongoing metabolic research.

Adipose tissue, once viewed primarily as a passive energy storage organ, is now recognized as an active endocrine organ that secretes a wide array of bioactive peptides known as adipokines. These adipokines play a critical role in regulating glucose and lipid metabolism, insulin sensitivity, inflammation, and vascular function.[7] Among them, adiponectin is one of the most abundant and biologically significant adipokines. It exerts potent insulin-sensitizing, anti-inflammatory, and anti-atherogenic effects by suppressing hepatic gluconeogenesis, enhancing peripheral glucose uptake, improving lipid oxidation, and protecting vascular endothelium.[8,9]

Paradoxically, circulating adiponectin levels are reduced in conditions characterized by excess adiposity, particularly visceral obesity, as well as in insulin resistance and MetS.[10,11] Lower serum adiponectin concentrations have been consistently associated with increased visceral fat mass, impaired insulin sensitivity, and a higher risk of T2DM and cardiovascular disease.[12] The decline in adiponectin levels in obesity and insulin-resistant states has been attributed to adipose tissue dysfunction, inflammatory cytokine activity, oxidative stress, and epigenetic regulation affecting adiponectin gene expression.[13] Moreover, adiponectin has been shown to influence pancreatic β-cell function and insulin secretion, suggesting a bidirectional relationship between adiponectin deficiency and insulin resistance.[14]

A growing body of evidence supports an inverse relationship between serum adiponectin levels and insulin resistance, indicating that hypoadiponectinemia may play a contributory role in the initiation and progression of MetS.[15,16] However, the strength and clinical relevance of this association appear to vary across populations, influenced by ethnic, genetic, and environmental factors. While several international studies have demonstrated the association of low adiponectin levels with MetS and its components, data from Indian populations—particularly from tertiary care settings—remain limited and inconsistent.[17]

Given the high and rising prevalence of MetS, diabetes, and cardiovascular disease in India, there is an urgent need to identify reliable biochemical markers that can facilitate early detection, risk stratification, and targeted intervention. In this context, evaluating the relationship between serum adiponectin levels, insulin resistance, and MetS may provide valuable insights into the metabolic and hormonal disturbances underlying this syndrome.

Therefore, the present cross-sectional study was conducted at a tertiary care center to assess the association of serum adiponectin levels and insulin resistance with metabolic syndrome

MATERIALS AND METHODS

Study Design and Setting

This study was conducted as a hospital-based cross-sectional observational study at a tertiary care center. The study was carried out in the Departments of Biochemistry.

Study Population

A total of 80 participants were included in the study. Adult individuals attending the outpatient or inpatient services of the tertiary care hospital were screened for eligibility and enrolled after fulfilling the inclusion criteria.

Inclusion Criteria

- Adults aged ≥18 years
- Participants willing to provide written informed consent
- Individuals diagnosed with metabolic syndrome as per standard criteria and individuals without metabolic syndrome (controls)

Exclusion Criteria

- Known cases of type 1 diabetes mellitus
- Patients with acute or chronic inflammatory diseases
- Individuals with chronic liver disease, chronic kidney disease, malignancy, or endocrine disorders affecting metabolism
- Pregnant or lactating women
- Patients on medications known to influence adiponectin levels or insulin sensitivity (such as corticosteroids or thiazolidinediones)

Diagnostic Criteria for Metabolic Syndrome

Metabolic syndrome was diagnosed based on the International Diabetes Federation (IDF) criteria. Central obesity was defined by waist circumference ≥90 cm in men and ≥80 cm in women (South Asian cut-offs), along with the presence of any two of the following:

- Raised triglycerides (≥150 mg/dL) or treatment for hypertriglyceridemia
- Reduced HDL cholesterol (<40 mg/dL in men and <50 mg/dL in women) or treatment for low HDL

- Elevated blood pressure (≥130/85 mmHg) or previously diagnosed hypertension
- Raised fasting plasma glucose (≥100 mg/dL) or previously diagnosed type 2 diabetes mellitus

Data Collection

After obtaining informed consent, detailed demographic and clinical data were collected using a pre-structured proforma. Information regarding age, sex, medical history, medication use, and lifestyle factors was recorded.

Anthropometric Measurements

Anthropometric measurements were obtained using standard techniques. Body weight was measured using a calibrated digital weighing scale, and height was measured using a stadiometer. Body mass index (BMI) was calculated as weight divided by height squared (kg/m²). Waist circumference was measured at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest using a non-stretchable measuring tape.

Blood Pressure Measurement

Blood pressure was measured in the sitting position after at least 5 minutes of rest using a standard sphygmomanometer. Two readings were taken at an interval of 5 minutes, and the average of the two readings was considered for analysis.

Laboratory Investigations

After an overnight fast of 8–12 hours, venous blood samples were collected under aseptic precautions. The samples were analyzed for the following parameters:

- Fasting plasma glucose
- Serum triglycerides
- High-density lipoprotein (HDL) cholesterol
- Fasting serum insulin
- Serum adiponectin levels

Fasting plasma glucose and lipid profile parameters were analyzed using standard enzymatic methods on an automated analyzer. Fasting serum insulin and serum adiponectin levels were measured using enzyme-linked immunosorbent assay (ELISA) kits, following the manufacturer's instructions.

Assessment of Insulin Resistance

Insulin resistance was assessed using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) index. HOMA-IR was calculated using the formula:

HOMA-IR = (Fasting insulin × Fasting glucose) / 405

Higher HOMA-IR values indicated greater degrees of insulin resistance.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) software 25. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, depending on data distribution. Categorical variables were expressed as frequencies and percentages. Comparisons between groups were performed using the independent Student's t-test or Mann–Whitney U test for continuous variables and Chi-square test for categorical variables. Correlation between serum adiponectin levels and components of metabolic syndrome, including insulin resistance, was assessed using Pearson or Spearman correlation coefficients as appropriate. A p-value <0.05 was considered statistically significant.

Ethical Considerations

The study was conducted in accordance with the Declaration of Helsinki. Institutional Ethics Committee approval was obtained prior to the commencement of the study, and written informed consent was obtained from all participants before enrollment.

RESULTS

A total of 80 participants were included in the study and were categorized into two groups: participants with metabolic syndrome (MetS group, n = 40) and participants without metabolic syndrome (Non-MetS group, n = 40).The mean age of the study population was higher in the MetS group compared to the Non-MetS group. The mean age in the MetS group was 52.4 ± 8.6 years, while in the Non-MetS group it was 44.9 ± 7.9 years. The median age was 53 years (IQR: 47–58) in the MetS group and 45 years (IQR: 39–50) in the Non-MetS group. The difference in age between the two groups was statistically significant ($p < 0.01$), as assessed using the independent Student's t-test. Among the MetS group, 24 participants (60%) were males and 16 (40%) were females. In the Non-MetS group, 22 participants (55%) were males and 18 (45%) were females. There was no statistically significant difference in sex distribution between the two groups ($p = 0.65$), as analyzed using the Chi-square test.

Participants with MetS had significantly higher anthropometric measurements compared to those without MetS.The mean body mass index (BMI) in the MetS group was 28.6 ± 3.4 kg/m², compared to 23.9 ± 2.8 kg/m² in the Non-MetS group. The median BMI was 28.4 kg/m² (IQR: 26.1–31.2) in the MetS group and 23.7 kg/m² (IQR: 22.1–25.4) in the Non-MetS group. This difference was statistically significant ($p < 0.01$), as determined by the independent Student's t-test. Waist circumference was significantly higher in the MetS group (102.8 ± 8.9 cm) compared to the Non-MetS group (88.6 ± 7.4 cm). The difference was statistically significant ($p < 0.01$), as analyzed using the independent Student's t-test.

The MetS group exhibited significantly higher systolic and diastolic blood pressure values. The mean systolic blood pressure in the MetS group was 138.6 ± 14.2 mmHg, compared to 118.9 ± 11.6 mmHg in the Non-MetS group ($p < 0.01$). The mean diastolic blood pressure was 88.4 ± 9.8 mmHg in the MetS group and 76.2 ± 8.4 mmHg in the Non-MetS group ($p < 0.01$). Comparisons were made using the independent Student's t-test.

Fasting plasma glucose, triglyceride levels, and HDL cholesterol levels differed significantly between the two groups. The mean fasting plasma glucose level in the MetS group was 118.6 ± 26.4 mg/dL, while it was 92.8 ± 12.3 mg/dL in the Non-MetS group. The median fasting glucose levels were 114 mg/dL (IQR: 102–132) and 90 mg/dL (IQR: 84–98), respectively. This difference was statistically significant ($p < 0.01$), as assessed using the independent Student's t-test. Serum triglyceride levels were significantly higher in the MetS group (186.2 ± 52.8 mg/dL) compared to the Non-MetS group (118.4 ± 36.1 mg/dL) ($p < 0.01$). HDL cholesterol levels were significantly lower in the MetS group (38.6 ± 6.8 mg/dL) than in the Non-MetS group (49.2 ± 7.4 mg/dL) ($p < 0.01$). Independent Student's t-test was used for these comparisons.

The mean fasting serum insulin level was significantly higher in participants with MetS (18.4 ± 6.9 µIU/mL) compared to those without MetS (9.6 ± 3.8 µIU/mL). The median fasting insulin levels were 17.6 µIU/mL (IQR: 13.2–22.4) and 9.1 µIU/mL (IQR: 6.8–11.9), respectively. The difference was statistically significant ($p < 0.01$), as analyzed using the Mann–Whitney U test. The mean HOMA-IR value was significantly higher in the MetS group (5.3 ± 2.4) compared to the Non-MetS group (2.2 ± 1.1). The median HOMA-IR values were 4.9 (IQR: 3.6–6.8) and 2.0 (IQR: 1.4–2.8), respectively. This difference was statistically significant ($p < 0.01$), using the Mann–Whitney U test.

The mean serum adiponectin level in the MetS group was 4.8 ± 1.9 µg/mL, compared to 9.6 ± 3.1 µg/mL in the Non-MetS group. The median adiponectin levels were 4.5 µg/mL (IQR: 3.4–5.9) and 9.2 µg/mL (IQR: 7.4–11.6), respectively. The difference between the groups was statistically significant ($p < 0.01$), as assessed using the Mann–Whitney U test. Serum adiponectin levels showed a significant negative correlation with HOMA-IR ($r = −0.62$, $p < 0.01$), waist circumference ($r = −0.58$, $p < 0.01$), and fasting plasma glucose levels ($r = −0.46$, $p < 0.01$). A significant positive correlation was observed between serum adiponectin levels and HDL cholesterol ($r = 0.51$, $p < 0.01$). Pearson's correlation coefficient was used for normally distributed variables, while Spearman's correlation was applied where appropriate.

Table 1: Baseline Demographic and Anthropometric Characteristics of the Study Population (n = 80)

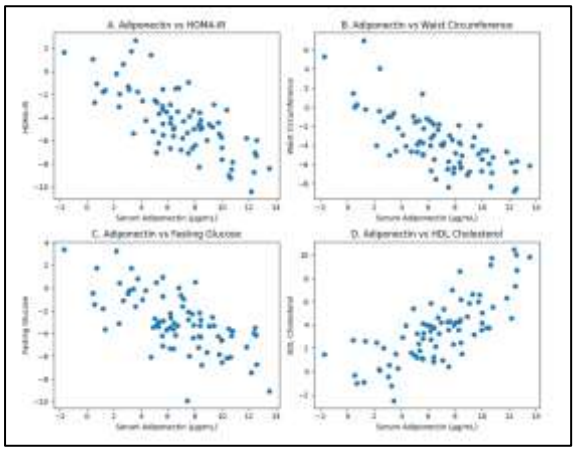
Parameter	MetS Group (n = 40)	Non-MetS Group (n = 40)	Statistical Test	p-value
Age (years), mean ± SD	52.4 ± 8.6	44.9 ± 7.9	Independent t-test	<0.01
Median age (IQR)	53 (47–58)	45 (39–50)	—	—
Male sex, n (%)	24 (60.0)	22 (55.0)	Chi-square test	0.65
Female sex, n (%)	16 (40.0)	18 (45.0)		
BMI (kg/m²), mean ± SD	28.6 ± 3.4	23.9 ± 2.8	Independent t-test	<0.01
Median BMI (IQR)	28.4 (26.1–31.2)	23.7 (22.1–25.4)	—	—
Waist circumference (cm), mean ± SD	102.8 ± 8.9	88.6 ± 7.4	Independent t-test	<0.01

Table 2: Comparison of Metabolic and Insulin Resistance Parameters Between Groups

Parameter	MetS Group (n = 40)	Non-MetS Group (n = 40)	Statistical Test	p-value
Fasting plasma glucose, mean ± SD	118.6 ± 26.4	92.8 ± 12.3	Independent t-test	<0.01
Median glucose (IQR)	114 (102–132)	90 (84–98)	—	—
Triglycerides, mean ± SD	186.2 ± 52.8	118.4 ± 36.1	Independent t-test	<0.01
HDL cholesterol, mean ± SD	38.6 ± 6.8	49.2 ± 7.4	Independent t-test	<0.01
Fasting insulin (µIU/mL), mean ± SD	18.4 ± 6.9	9.6 ± 3.8	Mann–Whitney U test	<0.01
Median insulin (IQR)	17.6 (13.2–22.4)	9.1 (6.8–11.9)	—	—
HOMA-IR, mean ± SD	5.3 ± 2.4	2.2 ± 1.1	Mann–Whitney U test	<0.01
Median HOMA-IR (IQR)	4.9 (3.6–6.8)	2.0 (1.4–2.8)	—	—

Table 3: Serum Adiponectin Levels and Correlation with Metabolic Parameters (n = 80)

Parameter	Value	Correlation Coefficient (r)	p-value	Statistical Test
Serum adiponectin (µg/mL), mean ± SD	7.2 ± 3.4	—	—	—
MetS group, mean ± SD	4.8 ± 1.9	—	<0.01	Mann–Whitney U test
Non-MetS group, mean ± SD	9.6 ± 3.1	—		
Correlation with HOMA-IR	—	−0.62	<0.01	Pearson correlation
Correlation with waist circumference	—	−0.58	<0.01	Pearson correlation
Correlation with fasting glucose	—	−0.46	<0.01	Pearson correlation
Correlation with HDL cholesterol	—	+0.51	<0.01	Pearson correlation



DISCUSSION

Baseline Demographic Characteristics

In the present cross-sectional study of 80 participants, individuals with metabolic syndrome (MetS) were significantly older than those without MetS, with a mean age of 52.4 ± 8.6 years versus 44.9 ± 7.9 years, respectively ($p < 0.01$). This finding indicated that advancing age was an important determinant of MetS, likely reflecting cumulative exposure to metabolic stressors, progressive insulin resistance, and increased visceral adiposity. Similar age-related increases in MetS prevalence have been reported by Eckel et al. and Grundy, who attributed this trend to age-associated declines in insulin sensitivity and metabolic flexibility.[1,2] Gronner et al. also observed a higher prevalence of MetS among older adults in population-based analyses.[3]

Sex distribution did not differ significantly between the MetS and non-MetS groups (60% vs. 55% males; $p = 0.65$), suggesting that sex alone did not independently influence MetS occurrence in this cohort. Comparable observations were reported by Singhal et al. in an Indian population, emphasizing that metabolic and lifestyle factors may outweigh sex-specific influences.[17] Participants with MetS demonstrated significantly higher measures of general and central obesity. The mean BMI was 28.6 ± 3.4 kg/m² in the MetS group compared to 23.9 ± 2.8 kg/m² in the non-MetS group, while waist circumference was 102.8 ± 8.9 cm versus 88.6 ± 7.4 cm, respectively ($p < 0.01$ for both). These findings underscored the pivotal role of abdominal obesity in the pathogenesis of MetS. Ibrahim highlighted the metabolic activity of visceral adipose tissue and its strong association with insulin resistance.[6] Matsuzawa further emphasized waist circumference as a key marker of visceral fat syndrome and metabolic risk.[11] Similar findings were reported by Cho et al., who identified visceral fat as a strong predictor of MetS development.[16] Blood pressure levels were significantly elevated in the MetS group, with mean systolic and diastolic pressures of 138.6 ± 14.2 mmHg and 88.4 ± 9.8 mmHg, respectively, compared to 118.9 ± 11.6 mmHg and 76.2 ± 8.4 mmHg in the non-MetS group ($p < 0.01$). These results were consistent with the diagnostic framework of MetS and previous reports by Grundy and Eckel et al., who identified hypertension as a core component contributing to cardiovascular risk in MetS.[1,2]

Participants with MetS also exhibited significant metabolic derangements, including higher fasting plasma glucose (118.6 ± 26.4 mg/dL vs. 92.8 ± 12.3 mg/dL), elevated triglycerides (186.2 ± 52.8 mg/dL vs. 118.4 ± 36.1 mg/dL), and reduced HDL cholesterol levels (38.6 ± 6.8 mg/dL vs. 49.2 ± 7.4 mg/dL), all with $p < 0.01$. These findings reflected the classical dyslipidemic and glycemic profile of MetS, consistent with reports by Eckel et al. and Moller et al,[1,5] as well as Balsan et al., who linked these abnormalities to adipose tissue dysfunction and insulin resistance.[12] Insulin resistance was markedly higher among MetS participants, with significantly elevated fasting insulin levels (18.4 ± 6.9 µIU/mL vs. 9.6 ± 3.8 µIU/mL) and HOMA-IR values (5.3 ± 2.4 vs. 2.2 ± 1.1; $p < 0.01$). These findings reinforced the central role of insulin resistance in MetS pathophysiology, as described by Kahn et al., Rabe et al., and Kadowaki et al.[4,7,9]

A major finding of this study was the significantly reduced serum adiponectin levels in the MetS group (4.8 ± 1.9 µg/mL) compared to the non-MetS group (9.6 ± 3.1 µg/mL; $p < 0.01$). Furthermore, adiponectin showed strong negative correlations with HOMA-IR ($r = -0.62$), waist circumference ($r = -0.58$), and fasting glucose ($r = -0.46$), and a positive correlation with HDL cholesterol ($r = 0.51$), all statistically significant. These findings were consistent with the seminal observations of Arita et al,[10] and subsequent studies by Matsuzawa, Yamauchi, Matsushita et al., and Cho et al., which demonstrated inverse relationships between adiponectin, visceral adiposity, and metabolic risk.[8,11,15,16] Mechanistic insights from Kim et al. further suggested that obesity-induced epigenetic suppression of adiponectin expression contributes to insulin resistance.[13]

CONCLUSION

The present cross-sectional study demonstrated that metabolic syndrome was associated with advancing age, increased adiposity, adverse metabolic parameters, and marked insulin resistance. Participants with metabolic syndrome had higher body mass index, waist circumference, blood pressure, fasting plasma glucose, triglycerides, fasting insulin, and HOMA-IR, along with lower HDL cholesterol, compared to those without metabolic syndrome. Serum adiponectin levels were significantly reduced in the metabolic syndrome group and showed an inverse correlation with insulin resistance, waist circumference, and fasting plasma glucose, and a positive correlation with HDL cholesterol.

Limitations

- The study was conducted at a single tertiary care centre with a modest sample size of 80 participants, which may limit generalizability and introduce selection bias.
- Insulin resistance was estimated using HOMA-IR rather than direct methods such as the hyperinsulinemic–euglycemic clamp.
- Only total serum adiponectin was measured; high-molecular-weight adiponectin, the most biologically active isoform, was not separately assessed.
- Lifestyle and socioeconomic factors, including diet, physical activity, smoking, and income, were not evaluated in detail and could have influenced adiponectin levels and insulin resistance.

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