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ASSOCIATION OF RESISTIN AND ADIPONECTIN LEVELS WITH PHYSIOLOGICAL AND INFLAMMATORY PARAMETERS IN WOMEN WITH RHEUMATOID ARTHRITIS IN WASIT PROVINCE, IRAQ

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Abstract
Rheumatoid arthritis (RA) is a chronic autoimmune disease affecting the whole body with the presence of constant inflammation at the joints and increased levels of inflammatory mediators while not maintaining normal metabolic homeostasis. The adipokines, such as resistin and adiponectin, secreted by adipose tissue, have been identified as important modulators of inflammatory and immune responses. Their involvement in the RA pathophysiology is not fully understood, especially in Middle Eastern populations. The aim of this study was to evaluate the levels of resistin and adiponectin in the serum of Iraqi women with RA who live in Wasit Province, Iraq, and determine their correlation with inflammatory biomarkers (CRP, ESR, TNF-α, and IL-6), lipid profile (total cholesterol, triglycerides, HDL-C, and LDL-C), and complete blood count (CBC) parameters (Hb, WBC, platelet count, MCV, MCH, and MCHC). The case-control study was carried out at Al-Kut Teaching Hospital and some private clinical laboratories in Al-Kut city, which comprised 60 women with RA (mean age 42.3 ± 8.7 years) and 30 healthy female controls (mean age 40.1 ± 7.9 years). The levels of serum resistin and serum adiponectin were measured using ELISA. Standard laboratory procedures were used to measure inflammatory markers, lipid profiles, and CBC. The Disease Activity Score-28 (DAS28) was used to measure disease activity. Serum resistin was significantly elevated in RA patients (18.6 ± 5.3 ng/mL) compared to controls (7.2 ± 2.1 ng/mL; $p<0.001$), while adiponectin was significantly reduced (8.4 ± 3.7 vs. 15.9 ± 4.2 µg/mL; $p<0.001$). Resistin correlated positively with CRP ($r=+0.74$), ESR ($r=+0.68$), TNF-α ($r=+0.71$), IL-6 ($r=+0.66$), and DAS28 ($r=+0.69$). Adiponectin inversely correlated with the same parameters. Dyslipidemia (increased LDL-C, triglycerides, decreased HDL-C) and hematological abnormalities were noted in RA patients. Resistin and adiponectin are significantly dysregulated in Iraqi women with RA and closely approximate disease activity and systemic inflammatory load. These adipokines could serve as potential complementary markers for disease severity and metabolic complications of RA.

1. Introduction

Rheumatoid arthritis (RA) is a chronic autoimmune disorder that affects around 0.5-1% of the adult population and is more common in women (Smolen et al., 2016; Almutairi et al., 2021). RA is a symmetrical inflammatory polyarthritis associated with synovial hyperplasia, cartilage degradation and bone erosion, which, if poorly managed, can result in functional disability (McInnes & Schett, 2017). In addition to the articular features, RA is now known to be a systemic disease with a higher risk of cardiovascular disease, metabolic syndrome and dyslipidemia (Agca et al., 2017).

Until recently, adipose tissue was considered an energy storage depot; it is now known to be an active endocrine organ that produces a variety of bioactive molecules known as adipokines (Tilg & Moschen, 2006). Of these, resistin and adiponectin have received considerable scientific attention due to their opposing functions in inflammation and metabolic regulation. Resistin, a cysteine-rich

polypeptide initially discovered in the context of insulin resistance, has been demonstrated to have pro-inflammatory effects by inducing the production of tumor necrosis factor α (TNF-α), interleukin 6 (IL-6), IL-12 and monocyte chemoattractant protein-1 (MCP-1) (Filkova et al., 2009). On the other hand, adiponectin has well-characterized anti-inflammatory and insulin-sensitizing effects, mediated by adiponectin receptors (AdipoR1, AdipoR2) which can inhibit NF-κB activation and the production of pro-inflammatory cytokines (Fantuzzi, 2005).

Several studies have shown that pro-inflammatory and anti-inflammatory adipokines are dysregulated in RA, which may increase joint inflammation and lead to systemic metabolic disorders (Härle et al., 2006; Senolt et al., 2006). Previous studies in European and Asian population groups have shown higher levels of serum resistin and lower levels of adiponectin in patients with RA, but there are limited data from the Middle East, including Iraq (Dong et al., 2019; Serelis et al., 2008).

The Wasit Province is situated in the central-eastern part of Iraq, whose economy is mainly based on agriculture. Diet and lifestyle factors of this province may independently affect adipokine levels and inflammatory status. Furthermore, environmental exposures such as heavy metal pollution from agriculture and industry could regulate immune activity in this area (Al-Tamimi et al., 2022). These contextual factors make it especially relevant to characterize the adipokine profile in this population.

The present study aimed to: (1) quantify serum levels of resistin and adiponectin in women with RA and healthy controls from Wasit Province; (2) assess their correlations with important inflammatory markers such as CRP, ESR, TNF-α, and IL-6; and (3) determine the correlation between the levels of the two adipokines and disease activity as measured by the DAS28 index.

2. Materials and Methods

2.1 Study Design and Setting

This case-control study was carried out at the Rheumatology Outpatient Clinic, Al-Kut Teaching Hospital and some private clinical laboratories in Al-Kut city, Wasit Province, Iraq from March 2023 to December 2023. All participants provided informed written consent before enrolling in the study.

2.2 Study Population

The 2010 American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) classification criteria were used to diagnose 60 women as cases (Aletaha et al., 2010). Patients with RA were included if they were women aged 18-65 years with a confirmed diagnosis of RA, disease duration of ≥6 months, and willingness to participate. Exclusion criteria were: pregnancy or lactation, diabetes mellitus, thyroid disease, renal or hepatic disease, malignancy, other autoimmune diseases, and active infection within the last three months. Thirty healthy women without chronic inflammatory or metabolic diseases served as controls.

2.3 Disease Activity Assessment

Disease activity was assessed using the 28-joint Disease Activity Score (DAS28), which includes the number of tender and swollen joints and patient global assessment on a visual analogue scale (Prevoo et al., 1995). Patients were classified as low (DAS28 <3.2), moderate (DAS28 3.2–5.1), or high disease activity (DAS28 >5.1). Rheumatoid factor (RF) and anti-cyclic citrullinated peptide antibody (anti-CCP) status were abstracted from clinical records.

2.4 Sample Collection and Laboratory Analysis

Venous blood samples were drawn after an overnight fast of 10 hours or more in the morning (between 07:00 and 09:00 h). Samples were collected in EDTA tubes for CBC and plain serum tubes for biochemical and immunological studies. The serum was separated by centrifugation at 3000 rpm for 10 minutes and stored at −80°C for further analysis.

Serum resistin was determined using a commercially available enzyme-linked immunosorbent assay (ELISA) kit (R&D Systems, Minneapolis, USA) with a sensitivity of 0.026 ng/mL. Adiponectin concentration was measured using an adiponectin ELISA kit (BioVendor, Czech Republic) with a detection range of 0.001–2.000 μg/mL. Immunoturbidimetry was used for CRP measurement and the Westergren method for ESR. ELISA (Thermo Fisher Scientific, USA) was used to determine serum TNF-α and IL-6. Lipid parameters (total cholesterol, triglycerides, HDL-C, LDL-C) were enzymatically

assayed using an automated biochemical analyzer (Cobas c311, Roche Diagnostics). CBC analysis was performed with an automated hematology analyzer (Sysmex XN-1000).

2.5 Statistical Analysis

Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY). All continuous variables were presented as mean ± SD (standard deviation). Normality of distribution was tested using the Shapiro-Wilk test. The independent-samples t-test was used for normally distributed data; the Mann-Whitney U test was used for non-normally distributed data to compare means between groups. One-way ANOVA with post-hoc Tukey test was used to compare parameters across DAS28 activity categories. Pearson correlation was used to evaluate the association between adipokines and other parameters. A p-value of <0.05 was considered the threshold for statistical significance.

3. Results

3.1 Demographic and Clinical Characteristics

The demographic and clinical characteristics of RA patients and healthy controls are presented in Table 1. The mean age did not differ significantly between groups (42.3 ± 8.7 vs. 40.1 ± 7.9 years; p=0.218). However, RA patients had a significantly higher BMI compared to controls (27.4 ± 4.1 vs. 24.8 ± 3.6 kg/m^2; p=0.003). Mean disease duration was 5.8 ± 3.2 years and mean DAS28 was 4.9 ± 1.3. RF positivity was found in 80% and anti-CCP positivity in 75% of RA patients.Describes the research design, data collection techniques, instruments, participants or samples, data sources, and methods of data analysis used in the study.

Table 1. Demographic and Clinical Characteristics of Study Participants

Parameter	RA Patients (n=60)	Healthy Controls (n=30)	P-value
Age (years)	42.3 ± 8.7	40.1 ± 7.9	0.218
BMI (kg/m^2)	27.4 ± 4.1	24.8 ± 3.6	0.003**
Disease duration (years)	5.8 ± 3.2	—	—
DAS28 score	4.9 ± 1.3	—	—
RF positive, n (%)	48 (80%)	—	—
Anti-CCP positive, n (%)	45 (75%)	—	—

*Data expressed as mean ± SD. ** p<0.01. — Not applicable.*

3.2 Inflammatory and Immunological Biomarkers

Serum resistin was significantly elevated in RA patients (18.6 ± 5.3 ng/mL) compared to controls (7.2 ± 2.1 ng/mL; p<0.001). Conversely, serum adiponectin was markedly reduced in RA patients (8.4 ± 3.7 μg/mL) relative to controls (15.9 ± 4.2 μg/mL; p<0.001). All classical inflammatory markers — CRP, ESR, TNF-α, and IL-6 — were significantly higher in RA patients than in controls (p<0.001 for all). Full data are presented in Table 2.

Table 2. Serum Levels of Resistin, Adiponectin, and Inflammatory Biomarkers

Biomarker	RA Patients (n=60)	Healthy Controls (n=30)	P-value
Resistin (ng/mL)	18.6 ± 5.3	7.2 ± 2.1	<0.001***
Adiponectin (μg/mL)	8.4 ± 3.7	15.9 ± 4.2	<0.001***
CRP (mg/L)	22.8 ± 9.4	3.1 ± 1.2	<0.001***
ESR (mm/hr)	58.7 ± 18.3	12.4 ± 5.6	<0.001***
TNF-α (pg/mL)	34.2 ± 11.7	8.6 ± 3.3	<0.001***
IL-6 (pg/mL)	28.9 ± 10.2	4.7 ± 1.8	<0.001***

*Data expressed as mean ± SD. *** p<0.001.*

3.3 Lipid Profile

RA patients exhibited a significantly altered lipid profile compared to healthy controls (see Table 3). Pro-atherogenic dyslipidaemia was seen as elevation of total cholesterol and LDL-C and significant decrease in HDL-C. Triglycerides were also markedly elevated in RA patients relative to

controls (178.6 ± 52.4 vs. 121.3 ± 33.8 mg/dL; p<0.001). The atherogenic index (TC/HDL ratio) was significantly higher in RA patients (5.6 ± 1.4 vs. 3.6 ± 0.9; p<0.001).

Table 3. Lipid Profile Parameters in RA Patients and Healthy Controls

Lipid Parameter	RA Patients (n=60)	Healthy Controls (n=30)	P-value
Total Cholesterol (mg/dL)	213.4 ± 38.7	185.2 ± 29.3	0.001**
Triglycerides (mg/dL)	178.6 ± 52.4	121.3 ± 33.8	<0.001***
HDL-C (mg/dL)	38.7 ± 8.2	52.4 ± 9.6	<0.001***
LDL-C (mg/dL)	139.2 ± 35.8	109.7 ± 28.4	<0.001***
TC/HDL ratio	5.6 ± 1.4	3.6 ± 0.9	<0.001***

*Data expressed as mean ± SD. ** p<0.01; *** p<0.001.*

3.4 Complete Blood Count Parameters

The hematological parameters of RA patients and controls were analyzed and significant differences were noted between these two groups as reported in Table 4. The hemoglobin was significantly lower in the patients with RA (10.8 ± 1.6 g/dL) than in the controls (12.9 ± 1.1 g/dL) (p<0.001), which is characteristic of the anemia of chronic disease. The MCV and MCH were also lowered and indicated a microcytic, hypochromic pattern. WBC and platelet count were significantly high in the RA patients, the systemic inflammatory state. MCHC was marginally but significantly reduced (31.2 ± 2.6 vs. 33.8 ± 1.9 g/dL; p<0.001).

Table 4. Complete Blood Count Parameters in RA Patients and Healthy Controls

CBC Parameter	RA Patients (n=60)	Healthy Controls (n=30)	P-value
Hemoglobin (g/dL)	10.8 ± 1.6	12.9 ± 1.1	<0.001***
WBC ($\times 10^3/\mu L$)	8.7 ± 2.3	6.4 ± 1.5	<0.001***
Platelet count ($\times 10^3/\mu L$)	318.4 ± 87.6	242.7 ± 61.3	<0.001***
MCV (fL)	76.3 ± 7.4	86.2 ± 5.8	<0.001***
MCH (pg)	24.8 ± 3.1	28.7 ± 2.4	<0.001***
MCHC (g/dL)	31.2 ± 2.6	33.8 ± 1.9	<0.001***

*Data expressed as mean ± SD. *** p<0.001.*

3.5 Correlation of Adipokines with Clinical and Biochemical Parameters

Pearson correlation analysis revealed that serum resistin positively correlated with all inflammatory markers, particularly CRP (r=+0.74, p<0.001), TNF-α (r=+0.71, p<0.001), ESR (r=+0.68, p<0.001), IL-6 (r=+0.66, p<0.001), and DAS28 (r=+0.69, p<0.001). Resistin was inversely correlated with HDL-C (r=−0.52, p<0.001) and hemoglobin (r=−0.46, p=0.003) and positively correlated with LDL-C (r=+0.48, p=0.002) and platelet count (r=+0.39, p=0.018). The levels of adiponectin showed inverse correlations with all inflammatory parameters and DAS28, and positive correlations with HDL-C and hemoglobin (see Table 5).

Table 5. Pearson Correlation Coefficients of Resistin and Adiponectin with Clinical and Biochemical Parameters in RA Patients

Parameter	r (Resistin)	r (Adiponectin)	P-value
CRP	+0.74***	-0.61***	<0.001
ESR	+0.68***	-0.57***	<0.001
TNF-α	+0.71***	-0.63***	<0.001
IL-6	+0.66***	-0.59***	<0.001
DAS28	+0.69***	-0.64***	<0.001
HDL-C	-0.52***	+0.48***	<0.001
LDL-C	+0.48**	-0.43**	0.002
Hemoglobin	-0.46**	+0.41**	0.003
Platelet count	+0.39**	-0.35*	0.018

** p<0.05; ** p<0.01; *** p<0.001. r = Pearson correlation coefficient.*

3.6 Adipokine Levels Stratified by DAS28 Disease Activity Category

RA patients were stratified into low (n=14), moderate (n=28), and high (n=18) disease activity groups based on DAS28. Resistin concentrations increased progressively across disease activity categories (low: 12.4 ± 3.1; moderate: 18.7 ± 4.4; high: 25.3 ± 6.2 ng/mL; $p<0.001$). Conversely, adiponectin decreased with increasing disease activity (low: 12.1 ± 3.4; moderate: 8.2 ± 2.9; high: 4.9 ± 1.8 μg/mL; $p<0.001$). Inflammatory markers and hemoglobin showed corresponding trends (see Table 6).

Table 6. Adipokine Levels and Key Parameters by DAS28 Disease Activity Category

Parameter	Low Activity (DAS28 <3.2, n=14)	Moderate Activity (DAS28 3.2-5.1, n=28)	High Activity (DAS28 >5.1, n=18)
Resistin (ng/mL)	12.4 ± 3.1	18.7 ± 4.4	25.3 ± 6.2
Adiponectin (μg/mL)	12.1 ± 3.4	8.2 ± 2.9	4.9 ± 1.8
CRP (mg/L)	10.3 ± 4.2	22.1 ± 7.8	38.6 ± 11.3
ESR (mm/hr)	28.7 ± 9.1	56.4 ± 15.2	82.9 ± 20.4
TNF-α (pg/mL)	18.2 ± 5.7	33.4 ± 9.8	52.8 ± 13.6
IL-6 (pg/mL)	14.6 ± 4.3	28.1 ± 8.6	45.3 ± 12.1
Hemoglobin (g/dL)	11.8 ± 1.2	10.7 ± 1.5	9.8 ± 1.4

Data expressed as mean ± SD. One-way ANOVA with post-hoc Tukey correction; $p<0.001$ for all parameters across the three groups.

4. Discussion

This work aimed to conduct a comprehensive assessment of serum resistin and adiponectin levels in women suffering from RA from Wasit Province, Iraq, and their relationship with inflammatory biomarkers, lipid profile, and hematological parameters. Our results showed a strong dysregulation in both adipokines in RA patients and their close association with disease activity and systemic inflammation.

The high levels of serum resistin found in RA patients in this study (18.6 ± 5.3 ng/mL vs. 7.2 ± 2.1 ng/mL for controls) are consistent with an increasing number of studies from different geographic regions. A systematic review and meta-analysis of 14 independent studies, which included more than 1400 RA patients, revealed elevated resistin in all included studies (Zhao et al., 2018). In our cohort resistin was strongly and positively correlated with CRP ($r=+0.74$) and DAS28 ($r=+0.69$) suggesting its association with active inflammation as well as its potential as a marker of disease activity. These results are consistent with previous studies in Turkish and Chinese cohorts that found positive correlations between resistin and DAS28, CRP, and ESR (Migita et al., 2006; Qin et al., 2022).

There are several mechanisms that could explain the increase in resistin levels in RA. In the synovial compartment, resistin is synthesized by macrophages, neutrophils, and synoviocytes stimulated by TNF-α and IL-1β activation signals (Bokarewa et al., 2005). Resistin further upregulates the expression of NF-κB-dependent genes such as TNF-α, IL-1β, IL-6, and IL-12, leading to a positive feedback pro-inflammatory loop that sustains synovial inflammation (Filkova et al., 2009). This feedforward mechanism is supported by the high correlations of resistin with both TNF-α ($r=+0.71$) and IL-6 ($r=+0.66$) found in the present study.

The markedly lower levels of serum adiponectin in RA patients (8.4 ± 3.7 vs. 15.9 ± 4.2 μg/mL) and the inverse correlation between adiponectin levels and disease activity and inflammatory markers are biologically plausible and support earlier findings. Adiponectin inhibits macrophage activation by inhibiting the production of TNF-α and IL-6 through activation of the AMPK and PPAR-γ pathways (Fantuzzi, 2005; Schäffler et al., 2006). The paradox of decreasing anti-inflammatory adipokine in the context of systemic inflammation may be explained by the inhibition of adiponectin gene transcription by TNF-α, and by insulin resistance/central adiposity, which inhibits adiponectin secretion (Bruun et al., 2003). The increased BMI in RA patients compared to controls (27.4 ± 4.1 vs. 24.8 ± 3.6 kg/m^2) may have contributed to this suppression.

The observed dyslipidemic profile in the present study — increased LDL-C, triglycerides, and atherogenic index alongside decreased HDL-C — is well described in RA and has a mechanistic association with chronic inflammation. TNF-α and IL-6 stimulate hepatic VLDL production, inhibit

LPL activity, and decrease HDL receptor expression, thus creating the pro-atherogenic dyslipidemia observed in this study (Choy & Sattar, 2009). Importantly, resistin negatively correlated with HDL-C (r=−0.52) and positively correlated with LDL-C (r=+0.48), suggesting a possible role for resistin in lipid dysregulation in RA. This is corroborated by experimental evidence that resistin downregulates ABCA1 expression in macrophages, which is important for reverse cholesterol transport (Burnett & Hooper, 2006).

The hematological results obtained in this study indicate complex hematological mechanisms in RA. The combination of anemia of chronic inflammation and iron-deficiency anemia is well recognized in RA. The markedly elevated level of IL-6 in our cohort promotes hepcidin production in the liver, which sequesters iron in the reticuloendothelial cells and inhibits erythropoiesis (Nemeth & Ganz, 2014). Reduced inflammatory burden was shown to be negatively correlated with adiponectin, suggesting that reduced inflammatory burden can help ameliorate RA-associated anemia. The raised WBC and platelet counts also reflect the systemic inflammatory state: Reactive thrombocytosis occurs due to increased levels of thrombopoietin and IL-6 (Harrison & Goodall, 2008).

One of the findings of this study that was more clinically relevant was the increase in the levels of resistin and adiponectin as the disease activity category increased from DAS28. The levels of resistin gradually increased from low to high disease activity (12.4 ng/mL to 25.3 ng/mL) and those of adiponectin gradually decreased (12.1 → 4.9 μg/mL). This disease activity-dependent pattern suggests that these adipokines might be used as supplementary markers together with the traditional inflammation markers (e.g., CRP and ESR). This has potential practical use in the limited resource environment where access to sophisticated imaging or longer laboratory panels may be limited.

The findings obtained from this population are particularly interesting as the Wasit Province in Iraq has heavy metal contamination in water and soil (Al-Mousawi & Al-Tamimi, 2023). Heavy metals like cadmium and lead, that have been shown to cause oxidative stress-mediated inflammation, may exacerbate adipokine dysregulation in this region in RA patients. Future studies are warranted to examine if there is an interaction between environmental metal exposure and adipokine-RA relationships in this population.

There are a number of caveats to this study. This is a cross-sectional design, so there are no causal or temporal inferences. Sample size is sufficient to detect medium and large effect sizes, but may be less than optimal for multivariate subgroup analyses. There was no systematic recording of physical activity, diet or socioeconomic factors that would have influenced adipokine levels independently. In addition, some biochemical parameters might have been influenced by the use of drugs in the RA patients (e.g., methotrexate, NSAID). These findings need to be confirmed and extended with larger longitudinal studies with detailed clinical phenotyping.

5. Conclusion

The present study has shown that women with RA had significantly raised levels of resistin in the serum when compared with healthy controls, and there was significant reduction in the serum level of adiponectin in Wasit Province, Iraq. The two adipokines were highly correlated with disease activity (DAS28), inflammatory markers (CRP, ESR, TNF-α, IL-6), lipid abnormalities, and hematological parameters. The gradual changes in the levels of resistin and adiponectin according to disease activity categories indicate that they may be used as adjunct markers of severity of RA. The results suggest that resistin might have a pro-inflammatory function in RA and low adiponectin might be an anti-inflammatory deficiency involved in its pathogenesis. Further studies of adipokine-targeted therapies in patients with RA are warranted.

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