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Pro-inflammatory cytokines and insulin resistance in postmenopausal women with central obesity: A cross-sectional analytical study

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The postmenopausal period is characterized by hormonal fluctuations that predispose women to increased visceral adiposity, chronic low-grade inflammation, and impaired glucose-insulin homeostasis. Pro-inflammatory cytokines, particularly TNF-α and IL-6, are believed to play integral roles in this interplay. This study aimed to investigate the association between circulating levels of TNF-α and IL-6 and insulin resistance (HOMA-IR) in postmenopausal women across different BMI categories, while accounting for relevant lifestyle and demographic factors. A total of 240 postmenopausal women aged 45–65 years were classified as normal weight, overweight, or obese. Anthropometric indices (BMI, WHR, WHtR), serum levels of TNF-α, IL-6, insulin, fasting glucose, and HbA1c were measured. Insulin resistance was calculated using the HOMA-IR formula. Lifestyle data were collected through structured questionnaires. Obese participants exhibited significantly higher levels of TNF-α (17.16 ± 3.22 ng/mL), IL-6 (9.03 ± 2.29 ng/mL), insulin, and HOMA-IR. TNF-α and IL-6 levels showed strong positive correlations with insulin resistance markers. Unhealthy lifestyle patterns, such as physical inactivity and increased meal frequency, were also more prevalent in the obese group. TNF-α and IL-6 appear to be early inflammatory biomarkers associated with metabolic dysregulation in postmenopausal women with central obesity. Screening for cytokine levels alongside HOMA-IR may enhance early identification of metabolic risk. Lifestyle modification should remain a cornerstone of preventive interventions in this population.

Keywords: menopause; TNF-α; IL-6; insulin resistance; obesity.

Introduction

Obesity is a multifactorial metabolic condition characterized by excessive accumulation of adipose tissue, which predisposes individuals to a wide range of chronic diseases, most notably type 2 diabetes mellitus and cardiovascular disorders. According to Gajewska et al. (2024), global projections indicate that by 2035, over 1.77 billion individuals will be classified as overweight and 1.53 billion as obese, accounting for more than half of the adult population worldwide (Lobstein et al., 2024). These figures reflect not only the growing public health burden of obesity but also the urgent need to understand its interaction with key physiological transitions, particularly in women.

Menopause represents a critical endocrinological turning point in women's lives, marked by a decline in circulating estrogen and progesterone levels. These hormonal alterations exert profound effects on energy homeostasis, including reductions in insulin sensitivity, dysregulation of glucose and lipid metabolism, and increased fat redistribution toward visceral compartments (Çelik et al., 2016). Importantly, the postmenopausal state is associated with a higher risk of central obesity, which in turn amplifies metabolic risk through inflammatory and hormonal pathways.

Among the biological mediators linking obesity to metabolic dysfunction are pro-inflammatory cytokines, notably tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). TNF-α is primarily secreted by macrophages infiltrating dysfunctional adipose tissue, but can also be produced by renal and hepatic cells under stress conditions. It plays a pivotal role in impairing insulin signaling pathways and promoting chronic low-grade inflammation in obese individuals (Jin et al., 2023). IL-6, encoded on chromosome 7p, is a glycosylated polypeptide with broad immunometabolic activity; it is secreted in response to adipocyte hypertrophy and contributes to hepatic gluconeogenesis and systemic inflammation (Grebenciucova & VanHaeren, 2023).

Adipose tissue in obese women is not merely a passive energy reservoir but an active endocrine organ that secretes a complex array of cytokines and adipokines. Elevated circulating levels of TNF-α and IL-6 have been consistently associated with increased fat mass and BMI, and have been shown to impair adipogenesis, disrupt insulin signaling, and aggravate the pro-inflammatory state (Varra et al., 2024). These cytokines are therefore not only markers of inflammation but potential mechanistic drivers of insulin resistance and metabolic deterioration in postmenopausal women.

While several studies have explored the role of inflammatory markers in obesity, limited research has focused on their specific interrelationships with insulin resistance in postmenopausal women, a population already predisposed to hormonal and metabolic instability. Moreover, few studies have concurrently assessed the influence of lifestyle patterns – such as physical activity, dietary habits, and education level – on this inflammatory–metabolic axis.

In light of these considerations, the present study aims to investigate the association between TNF-α, IL-6, and insulin resistance (measured via HOMA-IR) in postmenopausal women across different BMI categories. Additionally, it examines how sociodemographic and lifestyle factors interact with these biological markers, thereby offering an integrated perspective on metabolic vulnerability in this population.

Materials and methods

Study design and ethical approval. This study employed a cross-sectional analytical design to evaluate the relationship between pro-inflammatory cytokines (TNF-α and IL-6), insulin resistance indices (HOMA-IR), and lifestyle parameters among postmenopausal women across different adiposity profiles. Ethical approval was granted by the Research Ethics Committee of the Department of Chemistry, College of Education for Pure Sciences, Tikrit University, Iraq (Approval No. CHM-TU/2023/44; Date: 25 October 2023). All study procedures adhered to the Declaration of Helsinki (2013 revision). Written informed consent was obtained from all participants after providing detailed information about the objectives, procedures, risks, and benefits of participation.

Participant recruitment and group classification. A total of 240 postmenopausal women, aged 45 to 65 years, were recruited from the National Obesity Clinic in Mosul, Iraq, between December 2023 and July 2024. Menopausal status was confirmed based on clinical criteria (≥12 months of amenorrhea without alternative pathological or physiological causes). Exclusion criteria included known systemic diseases (e.g., autoimmune, hepatic, renal, or endocrine disorders), history of malignancy, hormone replacement therapy, recent infection, or ongoing use of corticosteroids or immunosuppressive agents. Participants were categorized into three BMI-based groups according to WHO criteria: normal weight: BMI < 25 kg/m² (n = 80); overweight: BMI 25–29.9 kg/m² (n = 80); obese: BMI ≥ 30 kg/m² (n = 80).

Anthropometric measurements were taken by trained personnel using calibrated equipment under standardized conditions. Height was measured to the nearest 0.1 cm using a wall-mounted stadiometer, and weight was recorded to the nearest 0.1 kg using a digital scale (Seca 769, Germany). Waist and hip circumferences were measured using non-stretchable measuring tapes. Body mass index (BMI) was calculated as weight (kg) divided by the square of height (m²). Waist-to-Hip Ratio (WHR) and Waist-to-Height Ratio (WHtR) were derived from standard formulas.

Lifestyle and sociodemographic data. Sociodemographic and lifestyle data were collected through structured, pre-validated interviewer-administered questionnaires. Variables included: family history of obesity (Yes/No); physical activity status (defined as ≥150 minutes/week of moderate activity); smoking status (current or past use); daily meal frequency (categorized as ≤2 meals, 2–3 meals, >3 meals); educational attainment (none, primary, secondary, tertiary).

Questionnaire reliability was pre-tested on a pilot sample (n = 15) and yielded a Cronbach's alpha of 0.84.

Sample collection and processing. After an overnight fast of 10–12 hours, 5 mL of venous blood were drawn into sterile plain vacutainer tubes. Samples were allowed to clot at room temperature for 15 minutes and then centrifuged at 3000 rpm for 10 minutes. Serum aliquots were stored at –40 °C until analysis. All samples were processed within 2 hours of collection to minimize pre-analytical variability.

Biochemical and hormonal assays. Cytokine quantification:

Serum TNF-α was quantified using a sandwich ELISA kit (Cat. No: MBS355573; MyBioSource, USA), and IL-6 was measured using a high-sensitivity ELISA kit (Cat. No: E2363h; Bioassay Laboratory Technology, China). Serum insulin was assessed using a competitive ELISA (Cat. No: 88286; Monobind Inc., USA). Fasting blood glucose (FBS) was measured via enzymatic colorimetric method (BioSystems, Spain), while HbA1c levels were determined by ion-exchange high-performance liquid chromatography (HPLC) using NGSP-certified systems. All assays were performed in duplicate, and intra-assay and inter-assay coefficients of variation (CVs) were below 8% and 10%, respectively.

IL-6 concentrations were determined using an ELISA kit provided by Bioassay Laboratory Technology (China).

1. Insulin and glycemic indices: serum insulin was assayed using an ELISA kit (Monobind Inc., USA); fasting blood glucose (FBS) was determined via enzymatic colorimetric method using a kit from BioSystems (Spain); glycated hemoglobin (HbA1c) was assessed using high-performance liquid chromatography (HPLC)-based protocols standardized to NGSP/DCCT criteria.

2. Insulin resistance estimation: homeostatic model assessment for insulin resistance (HOMA-IR) was calculated using the formula: HOMA-IR = Fasting Insulin (μIU/mL) / times × Fasting Glucose (mg/dL)/405. The threshold for insulin resistance was defined as HOMA-IR > 2.5, as previously validated in similar postmenopausal populations (Kurtoglu et al., 2010).

Statistical analysis. Data were analyzed using SAS software (version 19.0, SAS Institute Inc., Cary, NC, USA). Normality of distribution was assessed using the Shapiro-Wilk test. Continuous variables were expressed as mean ± standard deviation (SD), and intergroup differences were evaluated using one-way ANOVA followed by Tukey's post hoc test. Homogeneity of variances was verified by Levene's test. Categorical data were analyzed using Pearson's Chi-square test (χ^2). Correlations between cytokine levels, HOMA-IR, and anthropometric parameters were assessed using Spearman's rank correlation (ρ). A P-value < 0.05 was considered statistically significant.

Results

As shown in Table 1, the prevalence of sedentary lifestyle, high meal frequency, and positive family history of obesity was significantly higher in obese women compared to controls (P < 0.001). A significantly higher proportion of obese women reported a positive family history of obesity (20.7%) compared to overweight (14.6%) and control (5.4%) groups (P < 0.001). Furthermore, physical inactivity was markedly more prevalent among the obese (25.6%) and overweight (24.3%) groups than the control group (17.4%) (P = 0.005).

Notably, the frequency of meal intake exceeding three meals per day was considerably higher in the overweight (16.0%) followed by obese group (12.7%) and control group (5.7%) (P < 0.001) indicating a positive trend between excessive caloric intake and BMI. In contrast, the control group exhibited a healthier behavioral profile with higher rates of regular exercise, lower smoking prevalence, and a greater proportion of college-educated women.

Table 1
Distribution of sociodemographic and lifestyle variables among postmenopausal women classified by BMI category

Questionnaire	Control	Overweight	Obese	P-value
Family history (Yes), %	5.4	14.6	20.7	<0.001
Exercise (Yes), %	15.0	8.0	6.7	0.005
Meals >3/day, %	5.7	16.0	12.7	<0.001
Smoking (Yes), %	4.0	7.3	6.7	0.39
College education, %	12.7	11.2	11.0	0.75

Note: data presented as percentages (%); P-values based on Chi-square test.

Anthropometric indices increased significantly with higher BMI categories (Table 2), with WHR and WHtR showing marked elevation in the obese group (P < 0.001). There were no statistically significant differences in mean age across the groups (P = 0.289). However, BMI increased progressively from the control group (21.27 ± 1.82 kg/m²) to the overweight (26.89 ± 1.47 kg/m²) and obese group (33.29 ± 2.22 kg/m²), reaching high statistical significance (P < 0.001). Similarly, both Waist-to-Hip Ratio (WHR) and Waist-to-Height Ratio (WHtR) were significantly elevated in the obese group compared to the other groups (P < 0.001), with the highest values observed among obese women (WHR = 0.95 ± 0.07; WHtR = 0.63 ± 0.04 (Table 1). Figure 1 illustrates the progressive increase in WHR and WHtR, indicating central fat accumulation in obese women.

Table 2
Comparison of anthropometric indices (BMI, WHR, WHtR) across BMI-based groups in postmenopausal women

Parameter	Control	Overweight	Obese	P-value
Age, years	53.00 ± 6.23	53.32 ± 6.43	55.68 ± 6.30	0.289
BMI, kg/m²	21.27 ± 1.82	26.89 ± 1.47	33.29 ± 2.22	<0.001
WHR	0.73 ± 0.07	0.79 ± 0.08	0.95 ± 0.07	<0.001
WHtR	0.47 ± 0.04	0.58 ± 0.05	0.63 ± 0.04	<0.001

Note: data are expressed as mean ± SD; P-values calculated using one-way ANOVA.

In Figure 1, boxes represent the interquartile range (Q_1–Q_3); the line indicates the median, and whiskers show minimum and maximum values. Different letters denote statistically significant differences between groups (ANOVA with Tukey's post hoc test, P < 0.05).

The serum concentrations of TNF-α and IL-6 across groups are presented in Table 3. TNF-α levels were significantly elevated in the obese group (17.16 ± 3.22 ng/mL) compared to both overweight (5.31 ± 2.20 ng/mL) and control (5.00 ± 2.88 ng/mL) groups (P < 0.001). Similarly, IL-6 concentrations followed a comparable pattern, being highest in the obese cohort (9.03 ± 2.29 ng/mL), relative to

overweight (2.96 ± 1.23 ng/mL) and control (3.12 ± 1.50 ng/mL) groups (P < 0.001) As detailed in Table 3, serum TNF-α and IL-6 levels were significantly higher in obese women compared to normal weight and overweight groups (P < 0.001), As depicted in Figure 2, both cytokines exhibited a sharp elevation in the obese group, reinforcing the inflammation-obesity linkage.

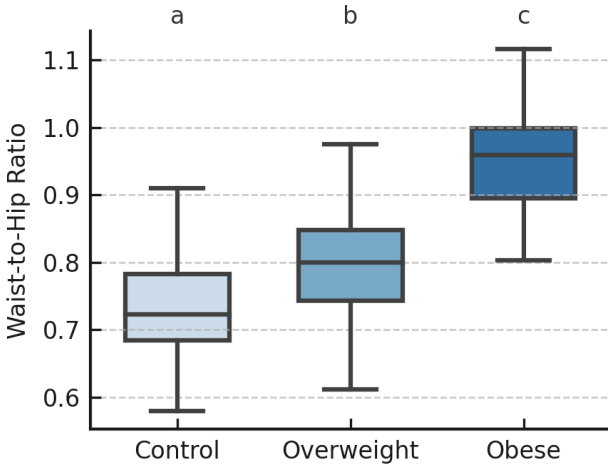

Fig. 1. Distribution of waist-to-hip ratio among normal weight, overweight, and obese postmenopausal women

Table 3
Serum levels of pro-inflammatory cytokines among postmenopausal women stratified by body weight category

Group	Tumor necrosis factor-alpha, ng/mL	Interleukin-6, ng/mL
Control	5.00 ± 2.88[a]	3.12 ± 1.50[a]
Overweight	5.31 ± 2.20[a]	2.96 ± 1.23[a]
Obese	17.16 ± 3.22[b]	9.03 ± 2.29[b]

Note: values are presented as mean ± standard deviation; different letters in superscript indicate statistically significant differences based on Tukey's post hoc test (P < 0.05).

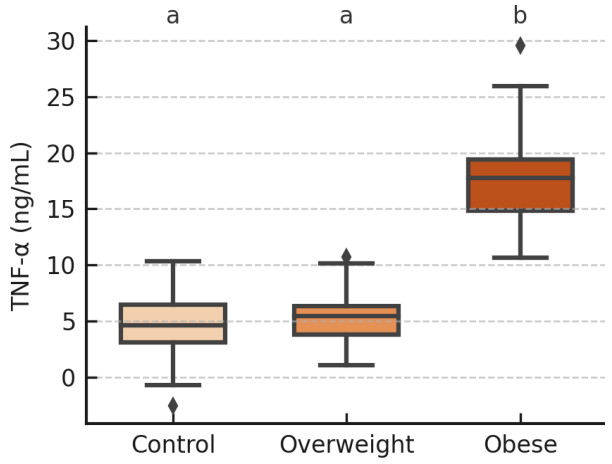

Fig. 2. Serum concentrations of tumor necrosis factor-alpha across normal weight, overweight, and obese postmenopausal women

Table 4 details the insulin-related biomarkers. Serum insulin concentrations were significantly higher in obese participants (13.44 ± 2.46 μIU/mL), followed by overweight (9.90 ± 2.75 μIU/mL), and control groups (6.59 ± 1.23 μIU/mL), with a high level of statistical significance (P < 0.001).

In parallel, the HOMA-IR index, an indicator of insulin resistance, demonstrated a significant upward trend from control (1.49 ± 0.38), to overweight (2.28 ± 0.35), to obese individuals (3.87 ± 0.58) (P < 0.001).

There was no statistically significant difference in fasting blood sugar (FBS) among the three groups (P = 0.024), though HbA1c levels remained statistically non-significant (P = 0.979), with minimal variation Obese participants showed significantly elevated insulin and HOMA-IR levels, while HbA1c remained statistically unchanged.

Obese participants showed significantly elevated insulin and HOMA-IR levels, while HbA1c remained statistically unchanged (Table 4). Figures 3 and 4 demonstrate the progressive elevation in serum insulin and insulin resistance indices across body weight categories.

Table 4
Glycemic and insulin-related indices across postmenopausal women classified by body weight

Group	Insulin, μIU/mL	Fasting blood sugar, mg/dL	Hemoglobin A1c, %	Homeostatic model assessment of insulin resistance
Control	6.59 ± 1.23[a]	80.10 ± 11.61	5.17 ± 0.34	1.49 ± 0.38[a]
Overweight	9.90 ± 2.75[b]	95.47 ± 15.94	5.19 ± 0.33	2.28 ± 0.35[b]
Obese	13.44 ± 2.46[c]	85.89 ± 24.84	5.18 ± 0.32	3.87 ± 0.58[c]

Note: see Table 3.

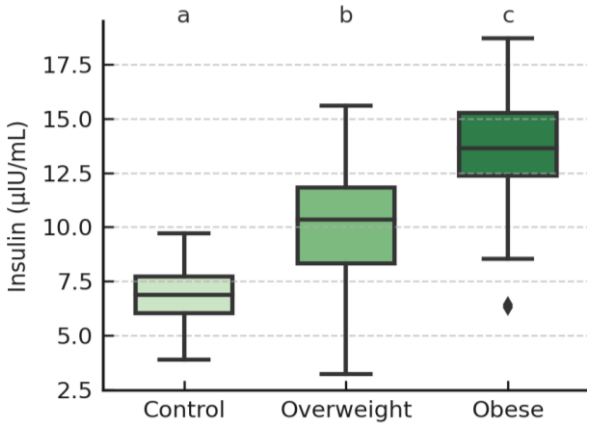

Fig. 3. Serum levels of immunoreactive insulin across normal weight, overweight, and obese postmenopausal women

Positive and statistically significant correlations were observed between TNF-α and IL-6, as well as between insulin and HOMA-IR (Spearman ρ > 0.70, P < 0.001). These correlations underscore the biological interplay between systemic inflammation and metabolic dysregulation in the obese postmenopausal population. The correlation matrix revealed strong positive associations between TNF-α and IL-6, as well as between insulin and HOMA-IR (ρ > 0.70, P < 0.001).

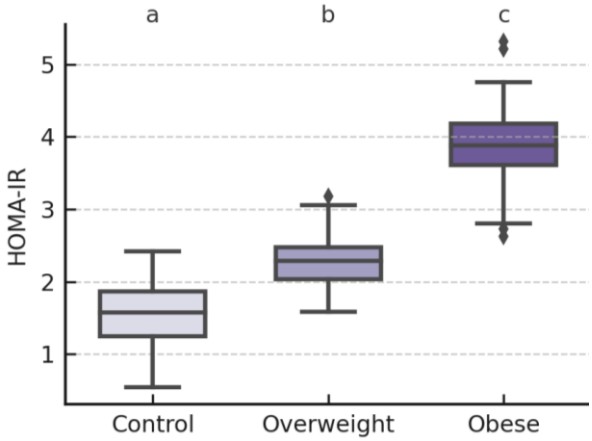

Fig. 4. Homeostatic model assessment of insulin resistance (HOMA-IR) values in postmenopausal women across normal weight, overweight, and obese categories

Discussion

The present cross-sectional investigation elucidates a robust association between central adiposity, elevated pro-inflammatory cytoki-

nes (TNF-α, IL-6), and insulin resistance in postmenopausal women. These findings support the hypothesis that adipose tissue dysfunction in this demographic is not merely a passive consequence of hormonal withdrawal but rather an active pro-inflammatory milieu that accelerates metabolic deterioration. The concurrent elevation of WHR and WHtR highlights a visceral fat phenotype that is increasingly recognized as a more potent predictor of cardiometabolic risk than BMI alone (Kozakowski et al., 2017; Ntikoudi et al., 2024). This is particularly salient in postmenopausal women, in whom estrogen deficiency promotes a shift toward android fat distribution and systemic inflammation.

Our data revealed a significant progressive increase in body mass index (BMI), waist-to-hip ratio (WHR), and waist-to-height ratio (WHtR) among overweight and obese women compared to controls. Notably, lifestyle factors – such as physical inactivity, positive family history of obesity, and increased meal frequency – were more prevalent among the obese subgroup. These findings are consistent with prior evidence suggesting that sedentary behavior and poor nutritional regulation are primary contributors to central adiposity during the menopausal transition (Çelik et al., 2016; Opoku et al., 2023).

The elevation in WHR and WHtR among obese women further reinforces the utility of these indices as sensitive anthropometric markers of visceral fat accumulation. This is particularly important in postmenopausal women, where estrogen decline fosters a shift toward android fat distribution (Kozakowski et al., 2017). Studies such as those by Arthur et al. (2013) and Uyanık & Yıldız (2024) have also identified WHR > 0.80 as a reliable predictor of metabolic risk, aligning with our present observations.

The markedly elevated levels of TNF-α and IL-6 among obese participants underscore the critical role of adipose-derived inflammation in mediating insulin resistance. These cytokines, predominantly secreted by hypertrophied adipocytes and infiltrating macrophages, impair insulin receptor substrate (IRS-1/2) signaling through serine phosphorylation, thereby disrupting glucose homeostasis (Obradovic et al., 2021). Furthermore, IL-6 activation of the JAK/STAT3 axis promotes hepatic gluconeogenesis, aggravating hyperinsulinemia and increasing β-cell stress. Our data align with prior mechanistic studies demonstrating that even in the absence of overt hyperglycemia, inflammatory signals can precede metabolic syndrome (Chandrasekaran & Weiskirchen, 2024).

Our findings are corroborated by studies showing that increased adiposity promotes a pro-inflammatory state that underpins the pathophysiology of obesity-related metabolic syndrome (Azeez, 2023; Mohammad et al., 2024). Furthermore, IL-6 levels were particularly elevated among obese women, suggesting its potential role as a biomarker of systemic inflammation in this demographic (Abd & Majeed, 2024).

Despite the pronounced elevation in insulin and HOMA-IR values among obese women, the lack of a significant increase in HbA1c suggests an early-phase insulin compensatory response aimed at preserving normoglycemia. This dissociation underscores the importance of incorporating insulin and HOMA-IR assessments into screening protocols, particularly in high-risk groups. While FBS showed mild fluctuation, its variability may be confounded by diurnal patterns and short-term dietary influences, making HOMA-IR a more stable and pathophysiologically relevant marker (Vigil et al., 2022; Kumar et al., 2023).

The correlation matrix further delineated significant positive associations between TNF-α and IL-6, and between insulin and HOMA-IR ($\rho > 0.70$, $P < 0.001$). These findings highlight a shared pathological axis wherein chronic inflammation and metabolic dysregulation mutually reinforce one another. Previous investigations by Noh et al. (2022) and Valaei-Barhagh et al. (2025) support this bidirectional model, wherein pro-inflammatory cytokines impair insulin signaling, and metabolic overload perpetuates inflammatory signaling loops.

Although the correlation matrix revealed a strong interdependence between TNF-α, IL-6, and HOMA-IR, it is important to recognize that the cross-sectional nature of the study limits the ability to infer causality. Future longitudinal studies incorporating cytokine gene polymorphisms, sex hormone profiling, and dynamic insulin clamp techniques are warranted to delineate causal pathways. Moreover, interventions targeting IL-6 inhibition, such as tocilizumab therapy, may serve as translational tools to validate the pro-inflammatory hypothesis in metabolic dysfunction among postmenopausal populations.

These findings underscore the necessity of early intervention in postmenopausal women, particularly those with central obesity. Incorporating cytokine profiling alongside routine glycemic monitoring could serve as a valuable clinical approach for identifying those at heightened risk for insulin resistance and its sequelae. Furthermore, public health strategies targeting modifiable risk factors—such as exercise promotion and dietary restructuring—should be prioritized in midlife women.

Despite the clinical relevance of our findings, this study has several limitations. Its cross-sectional design limits causal inferences. Additionally, important modulators such as estradiol, SHBG, lipid profiles, and genetic polymorphisms were not assessed. The sample was restricted to a single center in Iraq, which may limit generalizability. Future studies should include longitudinal tracking, hormonal profiling, and genotyping to clarify causality and enhance translational impact.

Conclusions

This study demonstrates a clear link between central obesity and systemic inflammation, mediated by elevated TNF-α and IL-6 levels, which strongly correlate with insulin resistance in postmenopausal women. These cytokines may serve not merely as correlational biomarkers but as potential mechanistic contributors to early metabolic dysfunction. The findings support the integration of inflammatory and metabolic profiling into clinical screening for postmenopausal women at risk of metabolic syndrome – even before overt glycemic alterations appear. Public health strategies targeting lifestyle optimization remain critical. Nevertheless, future longitudinal and mechanistic studies incorporating hormonal and genetic data are warranted to refine causality and intervention frameworks.

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