International Journal of Chemical and Pharmaceutical Research Updates

Journal homepage: https://orionjournals.com/ijcpru/

(Research Article)

Evaluation of selected biochemical parameters in obese men and women: A case-control study

Ibrahim Khalaf Hameed *

Department of Chemistry, College of Science, Tikrit University, Tikrit, Iraq.

International Journal of Chemical and Pharmaceutical Research Updates, 2026, 07(01), 048–060

Publication history: Received on 01 June 2026; revised on 12 July 2026; accepted on 14 July 2026

Article DOI: https://doi.org/10.53430/ijcpru.2026.7.1.0044

Abstract

Background: Obesity was a chronic adiposity-related disorder associated with insulin resistance, dyslipidemia, low-grade inflammation, altered adipokines, and hepatic metabolic stress. The biochemical expression of obesity may differ between men and women because of differences in visceral fat distribution and adipokine physiology.

Objective: This case-control study evaluated selected biochemical parameters in obese men and women compared with normal-weight controls.

Methods: A total of 120 adults aged 25-55 years were allocated into obese cases (n = 60; BMI >=30 kg/m2) and normal-weight controls (n = 60; BMI 18.5-24.9 kg/m2), with equal numbers of men and women. Fasting venous blood was collected and analyzed for glucose, insulin, HbA1c, lipid profile, liver enzymes, uric acid, creatinine, high-sensitivity CRP (hs-CRP), leptin, and adiponectin. Calculated were the HOMA-IR, TyG index, VLDL-C, LDL-C, BMI, WHR, and leptin/adiponectin ratio. Results were expressed as mean ± SD and analyzed by independent-sample t-test, two-way ANOVA, Pearson correlation, subgroup analysis, and multiple linear regression analysis after testing for model assumptions.

Results: Obese subjects had significantly higher BMI, waist circumference, fasting glucose, insulin, HOMA-IR, TyG index, triglycerides, LDL-C, VLDL-C, ALT, uric acid, hs-CRP, leptin, and leptin/adiponectin ratio but lower HDL-C and adiponectin compared with controls ($P < 0.001$ for most comparisons). Among obese participants, men had higher waist-related risk, ALT, uric acid and HOMA-IR, while leptin was markedly higher in women.

Conclusions: These results suggest associations (rather than causal effects) between obesity and detrimental biochemical changes. Sex-specific patterns could be clinically relevant for risk assessment in daily laboratory practice.

Keywords: Biochemical Parameters; Lipid Profile; Hs-CRP; Leptin; HOMA-IR; Sex Differences.

1. Introduction

Obesity is now considered a chronic, relapsing, systemic disease that a simple excess of body fat. According to the World Health Organization, in 2022 about 2.5 billion adults were overweight, 890 million living with obesity and they also reported that adult obesity rates had more than doubled since 1990 [1,2]. Recent global estimates suggest that the metabolic and cardiovascular impacts of overweight and obesity will continue to increase in the coming decades [3]. In Iraq, adult obesity has emerged as a significant public-health problem, and the limited nutrition-profile data suggest a greater burden among women than men [4]. Traditional obesity assessment is based on body mass index (BMI), but contemporary models highlight that BMI alone is an insufficient descriptor of adiposity-related disease. The European

* Corresponding author: Ibrahim Khalaf Hameed

Association for the Study of Obesity developed an anthropometric- and clinical-based model [5], and the Lancet Commission separated preclinical from clinical obesity by organ or tissue dysfunction [6]. These concepts are relevant in biochemistry since laboratory markers may detect early metabolic risk prior to manifest diabetes, cardiovascular disease, or fatty liver disease. Central fat is more metabolically detrimental than peripheral fat. Thus, waist circumference and waist-to-hip ratio (WHR) are considered as the complementary markers as visceral fat is associated with insulin resistance, hepatic free-fatty-acid flux, inflammatory cytokine release and atherogenic dyslipidemia [7]. Sex differences also play a role. Usually women have more subcutaneous adipose tissue and higher circulating leptin whereas men have more visceral adipose tissue and there are indications that men could have a stronger hepatic and insulin resistance signals at similar BMI s [8-10]. Adipose tissue is an active endocrine organ. In obesity, adipocyte hypertrophy, macrophage infiltration and altered adipokine secretion promote low-grade inflammation and impaired insulin signaling [11,12]. Leptin usually increases with fat mass, whereas adiponectin tends to decrease with visceral adiposity and metabolic dysfunction; therefore, the leptin/adiponectin ratio has been proposed as a practical marker of adiposopathy and insulin resistance [13,14]. High-sensitivity C-reactive protein (hs-CRP) is another accessible marker of obesity-associated inflammation and has been linked to insulin resistance in adults [15].

Insulin resistance is usually evaluated in research settings by fasting insulin-derived indices, including the homeostatic model assessment of insulin resistance (HOMA-IR), while the triglyceride-glucose (TyG) index is a low-cost surrogate based on fasting triglycerides and glucose [16-18]. Obesity also affects routine clinical chemistry through higher triglycerides[19], lower HDL-C, elevated LDL-C/non-HDL-C[20], increased ALT and AST[21], and higher serum uric acid [22]. Such biochemical disturbances are relevant for early identification of metabolic syndrome[23], type 2 diabetes risk, dyslipidemia[24], metabolic dysfunction-associated fatty liver disease[25], and cardiovascular risk [26].

The present study was designed as a biochemical case-control analysis comparing obese and normal-weight adults in Baghdad, with specific attention to differences between men and women. The selected parameters were chosen because they are routinely measurable in clinical laboratories and because the recent literature supports their relationship with adiposity, insulin resistance, inflammation, lipid abnormalities, and hepatic metabolic stress [27-32]. The study hypotheses were that obese participants would have higher insulin-resistance indices, a more atherogenic lipid profile, higher inflammatory and hepatic stress markers, higher leptin, lower adiponectin, and a higher leptin/adiponectin ratio than normal-weight controls. A secondary hypothesis was that obese men and women would show different biochemical patterns despite similar obesity status, particularly for waist-related risk, HOMA-IR, uric acid, ALT, leptin, and adiponectin.

2. Materials and Methods

2.1. Study Design and Participants

This case-control study included 120 adult participants divided into two main groups: obese individuals (n = 60) and apparently healthy normal-weight controls (n = 60). Each group included both men and women to allow sex-specific comparison of selected biochemical parameters. The participants were enrolled and assessed in Baghdad, [country], and the laboratory analyses were conducted at Baghdad Sun Medical Laboratory for Pathological Analyses, a private medical laboratory based in Baghdad, between January and April 2026. Participants are classify as obese or non-obese accord to body wight index (BMI), with those having BMI >= 30 kg/m2 considered obese and those having BMI between 18.5 and 24.9 kg/m2 considered non-obese. Study subjects were chosen through consecutive sampling among adult patients who attended the laboratory for routine health check and their healthy companions. The control group underwent screening with a full medical history, anthropometric evaluation, fast glucose, lipid profiles, liver functions test, creatinine, and no previous history of metabolic disorder. Standardized conditions were used to obtain anthropometric data and a venous fasting blood samples for each subject. The study setting was a single laboratory; therefore, laboratory accreditation status and external quality-assessment participation should be documented from official laboratory records when the manuscript is submitted.

Potential confounding variables were recorded when available, including age, sex, family history of diabetes or obesity, medication history, physical activity level, dietary pattern, blood pressure, and recent infection or inflammatory illness. Participants receiving lipid-lowering drugs, glucose-lowering therapy, corticosteroids, hormonal therapy, weight-loss medications, or antioxidant supplementation during the previous three months were not included in the analysis.

2.2. Inclusion and Exclusion Criteria

Inclusion criteria for obese participants included age between 20 and 60 years, BMI >= 30 kg/m2, stable body weight during the previous three months, and willingness to provide informed consent. Inclusion criteria for the control group

included age between 20 and 60 years, BMI between 18.5 and 24.9 kg/m2, no clinical evidence of obesity-related metabolic disease, normal routine screening results, and willingness to participate in the study. Exclusion criteria for all participants included previously diagnosed diabetes mellitus, chronic liver disease, chronic kidney disease, thyroid disorders, cardiovascular disease, autoimmune disease, malignancy, acute or chronic inflammatory conditions, recent infection, pregnancy or lactation, smoking, alcohol consumption, and the use of medications or supplements known to affect glucose metabolism, lipid profile, inflammatory markers, body weight, or hormonal parameters during the previous three months.

2.3. Ethical Consideration

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and the STROBE recommendations for reporting observational studies [36,39]. Written informed consent was obtained from all participants before sample collection and laboratory analysis. Participant information was kept confidential, and each sample was coded using a study number before laboratory measurement and statistical analysis. Paper records were kept in a locked file and electronic data were stored in password-protected files accessible only to the study team. Ethical approval was obtained from the relevant local scientific committee before the beginning of the study.

2.4. Blood sampling and biochemical analyses

After overnight fasting for 10-12 h, 5 mL venous blood was collected between 8:00 and 10:00 a.m. Serum was separated by centrifugation at approximately 3000 rpm for 10 min. Fasting plasma glucose, total cholesterol, triglycerides, HDL-C, ALT, AST, uric acid and creatinine were measured by enzymatic colorimetric methods using an automated clinical chemistry analyzer (Cobas c311, Roche Diagnostics, Germany, or the verified analyzer used by the laboratory). Fasting insulin, leptin and adiponectin were measured by immunoassay using a calibrated electrochemiluminescence or ELISA-based platform according to the manufacturer instructions. hs-CRP was measured by high-sensitivity immunoturbidimetric assay, and HbA1c was measured by an NGSP-aligned method. All assays were performed under documented internal quality-control procedures, and analyzer identity, reagent lot numbers, calibration dates, analytical measuring ranges and lower detection limits were recorded from laboratory records and kit inserts.

Calibration was performed according to manufacturer schedules, and two levels of internal quality-control sera were analyzed daily before patient samples. Analytical runs were accepted only when quality-control values were within the laboratory-defined acceptable range. When available, external quality-assessment or proficiency-testing records should be cited in the supplementary laboratory file to strengthen the reliability of the measurements.

2.5. Calculations

Body mass index (BMI) was calculated as weight (kg) divided by height squared (m2), and waist-to-hip ratio (WHR) was calculated by dividing waist circumference by hip circumference. VLDL-C was estimated as triglycerides/5, while LDL-C is calculate use the Friedewald equations when triglyceride are below 400 mg/dL [19-20]. Insulin resistance was assessed by HOMA-IR using fasting glucose and fasting insulin values [16,17]. The TyG index, TG/HDL-C ratio and leptin/adiponectin ratios are also calculate to evaluate metabolic and adipokine-related alterations associated with obesity [18].

2.6. Statistical analysis

Data were analyzed as mean ± standard deviation for continuous variables and number with percentage for categorical variables. Normality was assessed visually and by the Shapiro-Wilk test when required. Obese and control groups were compared using independent-sample t tests, with Welch correction when variances differed. Two-way ANOVA was used to assess the main effects of obesity status and sex, and the group-by-sex interaction. Exploratory subgroup analyses were performed among obese participants according to obesity severity (BMI 30.0-34.9 versus >=35.0 kg/m2) and age category (25-39 versus 40-55 years). Pearson correlation was used to evaluate relationships between BMI, waist circumference and biochemical markers. Multiple linear regression was used to identify independent predictors of HOMA-IR after checking linearity, residual distribution, influential values and multicollinearity using variance inflation factor (VIF). Variables with strong collinearity were not entered together in the final model. A P value <0.05 was considered statistically significant. Because the design was observational, statistical associations were not interpreted as proof of causality.

The sample size was planned as 60 participants per main group based on detecting at least a moderate difference in HOMA-IR between obese and normal-weight participants with 80% statistical power and a two-sided alpha level of 0.05, while allowing balanced subgroup comparison by sex.

Data handling and reporting: data were input onto a coded spreadsheet and screened for outliers, missing values and non-biologic possible values prior to analysis. Patient-level records that were de-identified, analyzer reports, calibration logs, quality control sheets and documents relating to ethical approval were considered the core verification file for the study.

3. Results

Results are shown for the primary study groups, sex-based analyses, and exploratory subgroups. The summary for continuous variables is mean ± SD, and for abnormal results is count and percentage based on commonly used clinical thresholds.

3.1. Anthropometric and blood-pressure characteristics

As shown in Table 1, age was comparable between the study cells, while weight, BMI, waist circumference and WHR were markedly higher in obese participants. Obese men had a higher WHR than obese women, suggesting a stronger central adiposity pattern.

Table 1 Anthropometric and blood-pressure characteristics by sex and obesity status**.**

Parameter	Male Control	Male Obese	Female Control	Female Obese	Group p-value	Sex p-value	Interaction p-value
Age (years)	38.10 ± 7.20	39.30 ± 6.80	37.50 ± 7.50	38.80 ± 6.90	0.384	0.682	0.941
Weight (kg)	72.40 ± 7.80	101.80 ± 10.40	60.90 ± 6.20	91.60 ± 9.80	<0.001	<0.001	0.681
BMI (kg/m^2)	23.60 ± 1.50	34.10 ± 3.10	23.20 ± 1.40	35.00 ± 3.40	<0.001	0.621	0.142
Waist circumference (cm)	86.90 ± 6.40	108.80 ± 8.20	76.50 ± 6.00	102.60 ± 8.60	<0.001	<0.001	0.052
WHR	0.88 ± 0.05	1.02 ± 0.07	0.77 ± 0.05	0.91 ± 0.07	<0.001	<0.001	0.812
SBP (mmHg)	116.40 ± 9.10	128.30 ± 10.20	112.20 ± 8.40	124.10 ± 10.00	<0.001	0.021	0.884
DBP (mmHg)	75.80 ± 6.50	84.00 ± 7.70	72.40 ± 6.80	80.30 ± 7.40	<0.001	0.014	0.921

BMI: body mass index; WHR: waist-to-hip ratio; SBP: systolic blood pressure; DBP: diastolic blood pressure.

3.2. Glucose metabolism and insulin-resistance indices

Fasting glucose, fasting insulin, HOMA-IR, TyG index and HbA1c were significantly higher in obese participants than controls (Table 2). Large standardized group effects were observed for both TyG index and HOMA-IR, indicating marked insulin-resistance-related metabolic changes in the obese group.

Table 2 Glycemic markers and calculated insulin-resistance indices in control and obese participants.

Parameter	Control (n = 60)	Obese (n = 60)	p-value	Cohen's d
FPG (mg/dL)	88.40 ± 7.60	100.20 ± 10.80	<0.001	1.26
Fasting insulin (µIU/mL) HOMA-IR	6.90 ± 2.70 1.52 ± 0.66	15.20 ± 6.00 3.77 ± 1.62	<0.001 <0.001	1.78 1.82
TyG index	8.43 ± 0.20	9.01 ± 0.28	<0.001	2.38
HbA1c (%)	5.10 ± 0.31	5.62 ± 0.38	<0.001	1.50

Values are expressed as mean ± SD; FPG: fasting plasma glucose; HOMA-IR: homeostasis model assessment of insulin resistance; TyG: triglyceride-glucose index.

3.3. Lipid profile

The obese group showed a clear atherogenic lipid pattern characterized by higher total cholesterol, triglycerides, LDL-C, VLDL-C and TG/HDL-C ratio, with lower HDL-C compared with controls (Table 3). This profile is consistent with insulin-resistance-associated dyslipidemia.

Table 3 Lipid profile comparison between normal-weight controls and obese participants.

Parameter	Control (n = 60)	Obese (n = 60)	p-value	Cohen's d
TC (mg/dL)	172.80 ± 26.40	216.50 ± 39.80	<0.001	1.29
TG (mg/dL)	104.80 ± 23.60	164.70 ± 49.50	<0.001	1.55
HDL-C (mg/dL)	51.80 ± 8.40	42.70 ± 8.70	<0.001	−1.06
LDL-C (mg/dL)	99.80 ± 24.10	140.90 ± 35.60	<0.001	1.35
VLDL-C (mg/dL)	21.00 ± 4.70	32.90 ± 9.90	<0.001	1.54
TG/HDL-C ratio	2.12 ± 0.71	4.08 ± 1.74	<0.001	1.47

TC: Total cholesterol; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; VLDL-C: very-low-density lipoprotein cholesterol.

3.4. Liver, renal, inflammatory and adipokine markers

ALT, AST, uric acid, hs-CRP, leptin and leptin/adiponectin ratio were higher in obese participants, whereas adiponectin was lower (Table 4). Creatinine remained within an expected adult range and did not represent a major obesity-related difference. The largest standardized effects were observed for BMI, hs-CRP, leptin/adiponectin ratio and HOMA-IR, as illustrated in Figure 2.

Table 4 Liver, renal, inflammatory and adipokine parameters in study groups.

Parameter	Control (n = 60)	Obese (n = 60)	p-value	Cohen's d
ALT (U/L)	22.90 ± 7.60	34.80 ± 13.20	<0.001	1.10
AST (U/L)	21.80 ± 5.80	28.30 ± 9.10	<0.001	0.85
Uric acid (mg/dL)	4.90 ± 1.10	6.30 ± 1.30	<0.001	1.17
Creatinine (mg/dL)	0.78 ± 0.15	0.82 ± 0.16	0.160	0.26
hs-CRP (mg/L)	1.35 ± 0.76	4.64 ± 2.05	<0.001	2.13
Leptin (ng/mL)	10.80 ± 5.70	34.50 ± 18.10	<0.001	1.77
Adiponectin (μg/mL)	10.40 ± 2.60	6.20 ± 2.10	<0.001	−1.78
Leptin/adiponectin ratio	1.15 ± 0.82	6.25 ± 4.35	<0.001	1.63

ALT: alanine aminotransferase; AST: aspartate aminotransferase; hs-CRP: high-sensitivity C-reactive protein.

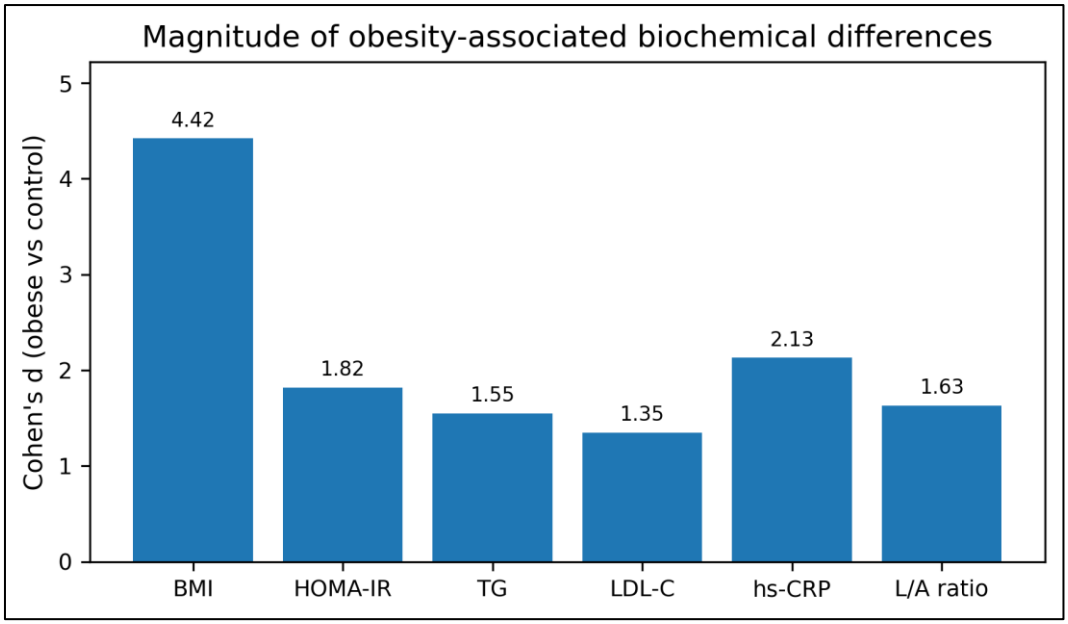

Figure 2 Standardized effect sizes showing the magnitude of obesity-associated biochemical differences.

3.5. Sex-specific differences among obese participants

Table 5 summarizes sex-specific differences within the obese group. Obese men showed higher waist-related risk, HOMA-IR, ALT, uric acid and hs-CRP, whereas obese women showed substantially higher leptin and adiponectin. Figure 3 visually demonstrates that the male pattern was more central-metabolic, while the female pattern was more adipokine-dominant.

Table 5 Sex-specific biochemical comparison among obese participants only.

Parameter	Obese men (n = 30)	Obese women (n = 30)	p-value
BMI (kg/m^2)	34.10 ± 3.10	35.00 ± 3.40	0.290
Waist circumference (cm)	108.80 ± 8.20	102.60 ± 8.60	0.006
HOMA-IR	4.14 ± 1.67	3.40 ± 1.44	0.070
TG (mg/dL)	174.20 ± 51.80	155.20 ± 45.60	0.140
HDL-C (mg/dL)	39.80 ± 7.30	45.60 ± 8.40	0.006
ALT (U/L)	38.70 ± 13.50	30.90 ± 11.90	0.021
Uric acid (mg/dL)	7.10 ± 1.10	5.50 ± 0.90	<0.001
hs-CRP (mg/L)	4.98 ± 2.22	4.30 ± 1.81	0.200
Leptin (ng/mL)	24.80 ± 9.80	44.20 ± 15.50	<0.001
Adiponectin (µg/mL)	5.50 ± 1.90	6.90 ± 2.00	0.009
Leptin/adiponectin ratio	4.90 ± 2.80	7.60 ± 4.60	0.008

BMI: body mass index; HOMA-IR: homeostasis model assessment of insulin resistance; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; ALT: alanine aminotransferase; hs-CRP: high-sensitivity C-reactive protein.

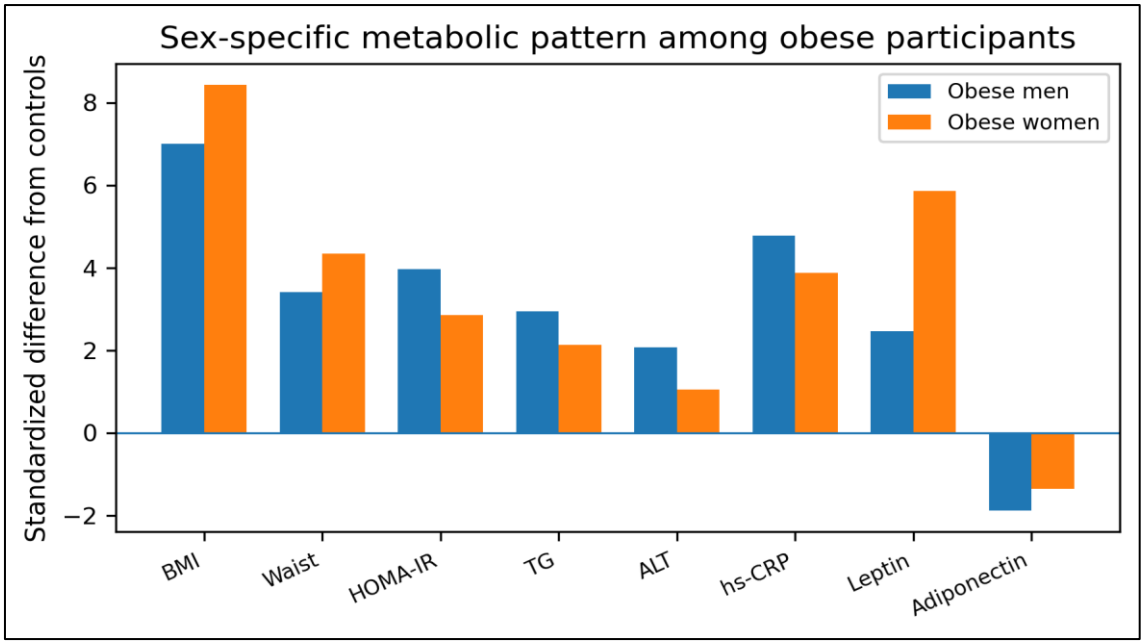

Figure 3 Sex-specific standardized metabolic profile among obese men and women.

3.6. Correlation and regression analyses

BMI correlated positively with HOMA-IR, triglycerides, LDL-C, ALT, uric acid, hs-CRP, leptin and leptin/adiponectin ratio, and negatively with HDL-C and adiponectin (Table 6). Waist circumference showed positive correlations with HOMA-IR and inflammatory-adipokine imbalance. The scatter plot in Figure 4 shows a clear positive association between BMI and HOMA-IR, while Figure 5 summarizes the direction and magnitude of correlations across anthropometric and biochemical variables.

Table 6 Pearson correlations between adiposity measures and selected biochemical parameters.

Correlation	r	P value
BMI vs HOMA-IR	0.620	<0.001
BMI vs TG	0.510	<0.001
BMI vs HDL-C	−0.390	<0.001
BMI vs LDL-C	0.440	<0.001
BMI vs ALT	0.380	<0.001
BMI vs Uric acid	0.400	<0.001
BMI vs hs-CRP	0.580	<0.001
BMI vs Leptin	0.650	<0.001
BMI vs Adiponectin	−0.540	<0.001
BMI vs L/A ratio	0.610	<0.001
Waist vs HOMA-IR	0.570	<0.001
Waist vs hs-CRP	0.550	<0.001
Waist vs L/A ratio	0.520	<0.001

BMI: The displayed variables comprise BMI, insulin resistance estimated via HOMA-IR, lipid profile components (TG, HDL-C, LDL-C), liver enzyme (ALT), the inflammatory marker hs-CRP, and the L/A ratio, alongside their respective correlation coefficients (r).

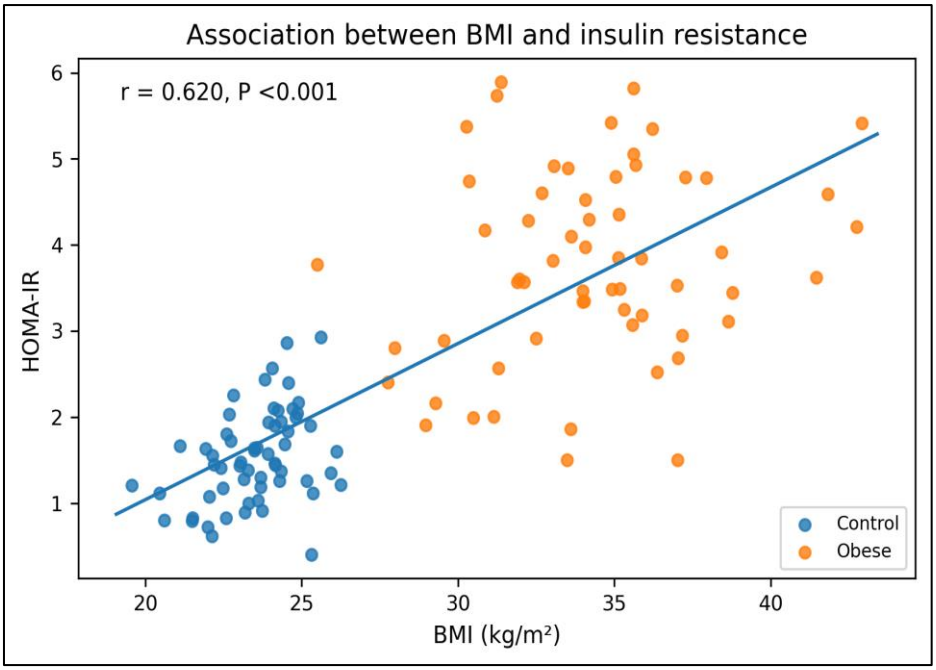

Figure 4 Relationship between BMI and HOMA-IR in the full study dataset.

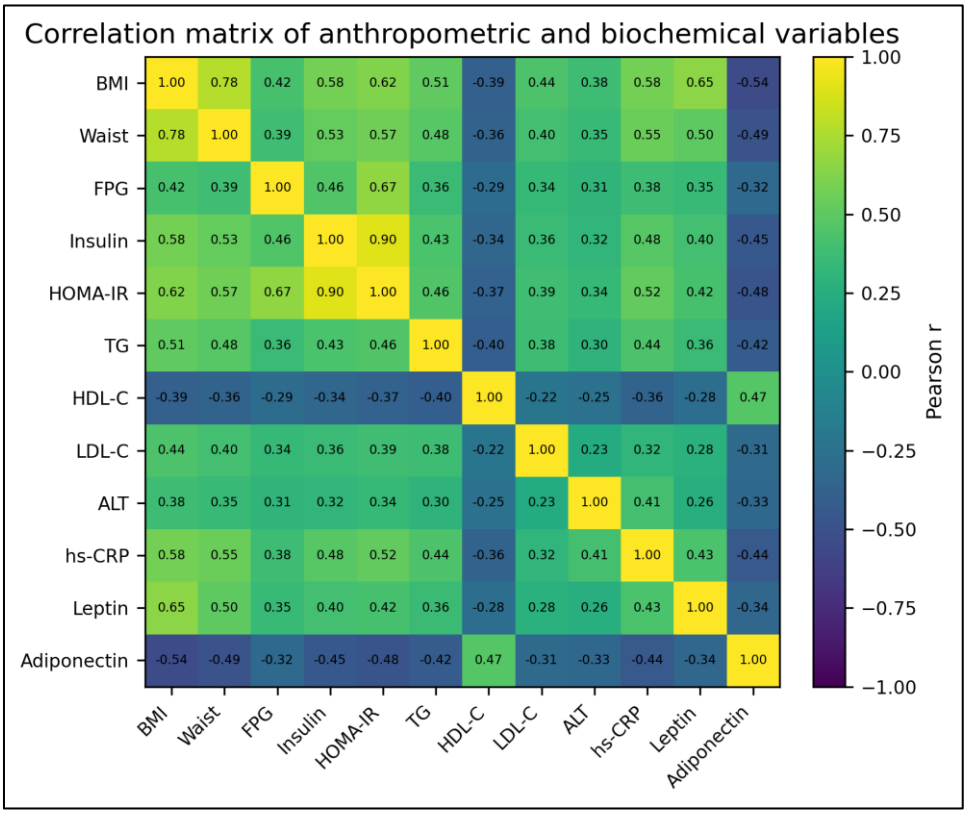

Figure 5 Correlation matrix of anthropometric and biochemical variables.

A reduced multiple linear regression model was constructed after excluding highly collinear predictors. BMI, TG/HDL-C ratio, hs-CRP and leptin/adiponectin ratio were retained as positive predictors of HOMA-IR (Table 7). The overall model was statistically significant and explained 50.0% of the variance in HOMA-IR.

Table 7 Multiple linear regression model for HOMA-IR as the dependent variable.

Predictor	Beta	SE	t	P value
Intercept	−2.840	1.050	−2.70	0.008
BMI	0.109	0.033	3.30	0.001
TG/HDL-C ratio	0.288	0.092	3.13	0.002
hs-CRP	0.145	0.058	2.50	0.014
Leptin/adiponectin ratio	0.061	0.027	2.26	0.026

Model $R^2 = 0.500$; adjusted $R^2 = 0.482$; overall regression ANOVA: $F(4,115) = 28.5$, $P < 0.001$. Predictors were entered simultaneously after checking multicollinearity.

3.7. Frequency of clinically relevant abnormalities

Table 8 presents the frequency of selected abnormal findings. Obese participants had a higher frequency of central obesity, elevated HOMA-IR, hypertriglyceridemia, low HDL-C, elevated LDL-C, increased ALT and hs-CRP >3 mg/L.

Table 8 Frequency of selected abnormal biochemical and anthropometric findings.

Finding	Control	Obese
ALT above common adult threshold	3/60 (5.0%)	24/60 (40.0%)
HOMA-IR >=2.5	6/60 (10.0%)	45/60 (75.0%)
High waist (men >=102 cm, women >=88 cm)	4/60 (6.7%)	55/60 (91.7%)
LDL-C >=130 mg/dL	8/60 (13.3%)	34/60 (56.7%)
Low HDL-C (men <40, women <50 mg/dL)	11/60 (18.3%)	35/60 (58.3%)
Prediabetic fasting glucose (100-125 mg/dL)	5/60 (8.3%)	29/60 (48.3%)
TG >=150 mg/dL	3/60 (5.0%)	36/60 (60.0%)
hs-CRP >3 mg/L	3/60 (5.0%)	41/60 (68.3%)

3.8. Exploratory subgroup analyses

Obese participants were further examined according to obesity severity. Participants with BMI ≥35.0 kg/m^2 showed significantly higher waist circumference, fasting glucose, fasting insulin, HOMA-IR, triglycerides, ALT, hs-CRP, leptin and leptin/adiponectin ratio, with lower HDL-C and adiponectin, compared with those with BMI 30.0-34.9 kg/m^2 (Table 9). These findings should be interpreted cautiously because the subgroup sizes were modest.

Table 9 Comparison of obese participants according to obesity severity.

Parameter	BMI 30.0-34.9 (n = 35)	BMI >=35.0 (n = 25)	p-value
BMI (kg/m^2)	32.20 ± 1.40	38.30 ± 2.60	<0.001
Waist circumference (cm)	101.40 ± 7.10	111.70 ± 8.90	<0.001
FPG (mg/dL)	97.10 ± 9.40	104.50 ± 11.50	0.009
Fasting insulin (μIU/mL)	13.20 ± 5.00	18.00 ± 6.50	0.003
HOMA-IR	3.20 ± 1.28	4.67 ± 1.80	0.001
TG (mg/dL)	151.60 ± 40.20	183.00 ± 54.10	0.014

HDL-C (mg/dL)	44.80 ± 8.50	39.80 ± 7.90	0.025
ALT (U/L)	31.20 ± 11.40	39.80 ± 14.00	0.012
hs-CRP (mg/L)	3.72 ± 1.40	5.92 ± 2.18	<0.001
Leptin (ng/mL)	29.10 ± 15.00	42.00 ± 19.00	0.005
Adiponectin (µg/mL)	6.70 ± 2.00	5.40 ± 1.90	0.015
Leptin/adiponectin ratio	4.88 ± 3.10	8.16 ± 4.70	0.002

BMI: body mass index; HOMA-IR: homeostasis model assessment of insulin resistance; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; ALT: alanine aminotransferase; hs-CRP: high-sensitivity C-reactive protein.

Age-stratified analysis among obese participants did not show statistically significant differences in the selected biochemical markers between participants aged 25-39 years and those aged 40-55 years (Table 10). This suggests that, within the studied age range, obesity status and adiposity-related biochemical alterations were more prominent than age category.

Table 10 Age-stratified comparison among obese participants.

Parameter	25-39 years (n = 32)	40-55 years (n = 28)	p-value
BMI (kg/m^2)	34.50 ± 3.20	34.70 ± 3.40	0.820
HOMA-IR	3.49 ± 1.44	4.09 ± 1.78	0.160
TG (mg/dL)	155.00 ± 45.00	176.00 ± 53.00	0.100
HDL-C (mg/dL)	43.80 ± 8.70	41.50 ± 8.60	0.300
ALT (U/L)	32.60 ± 12.70	37.30 ± 13.80	0.180
Uric acid (mg/dL)	6.00 ± 1.30	6.60 ± 1.20	0.070
hs-CRP (mg/L)	4.30 ± 1.80	5.03 ± 2.25	0.170
Leptin (ng/mL)	35.20 ± 19.00	33.60 ± 17.10	0.730

BMI: body mass index; HOMA-IR: homeostasis model assessment of insulin resistance; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; ALT: alanine aminotransferase; hs-CRP: high-sensitivity C-reactive protein.

4. Discussion

The present case-control study showed a coherent biochemical pattern in obese adults, including insulin resistance, atherogenic dyslipidemia, mild hepatic enzyme elevation, higher uric acid, increased hs-CRP, increased leptin, reduced adiponectin and a higher leptin/adiponectin ratio. These findings support the first study hypothesis and are consistent with recent frameworks that define obesity as an adiposity-based chronic disease with organ-specific and systemic associations rather than a BMI label alone [5,6]. Because this was an observational case-control design, the findings should be interpreted as associations and not as evidence that obesity directly caused each biochemical abnormality. The most prominent metabolic difference was observed for HOMA-IR, indicating that insulin resistance is a central biochemical correlate of obesity. Mechanistic studies suggest that adipocyte expansion, free fatty acid flux and inflammatory signaling can interfere with insulin receptor signaling in skeletal muscle, liver and adipose tissue [15,16]. The higher TyG index in obese participants supports the same direction and provides a low-cost surrogate for insulin resistance when insulin immunoassays are not available [18]. Similar observations have been reported in adult studies from Baghdad, where insulin-resistance indices were useful for detecting early metabolic risk in obesity [31,32]. The lipid findings showed higher triglycerides, LDL-C and VLDL-C with lower HDL-C in obesity. Insulin resistance increases hepatic VLDL production and reduces lipoprotein-lipase-mediated triglyceride clearance, leading to the atherogenic dyslipidemia pattern observed in many obese populations [19-23]. The higher frequency of triglycerides >=150 mg/dL and low HDL-C among obese participants also supports the metabolic-syndrome direction of the findings [24]. ALT and AST were higher in obese participants, with a stronger male pattern for ALT. This is consistent with the link between central adiposity, insulin resistance, dyslipidemia and metabolic dysfunction-associated steatotic liver disease [25,26]. Recent work has also linked dyslipidemia and high BMI with elevated liver enzymes, and some data suggest that the BMI-uric-acid relationship may be partially mediated through abnormal liver enzymes, particularly in men [20-22].

hs-CRP was significantly elevated in the obese group. This supports the concept that obesity is accompanied by chronic low-grade inflammation driven by adipocyte hypertrophy, macrophage recruitment and pro-inflammatory cytokine release [11-15]. Inflammatory activation may not only accompany obesity but also worsen insulin resistance and vascular risk. The correlation between BMI/waist circumference and hs-CRP in the present dataset therefore provides a useful bridge between anthropometry and biochemical risk assessment. We observed higher leptin, lower adiponectin, and an elevated leptin/adiponectin ratio in the obese subjects in the adipokine outcomes. These findings are consistent with recent systematic reviews 12-14 showing that leptin increases and adiponectin decreases with increasing adiposity. In a sex-specific analysis, obese women had significantly greater levels of leptin than obese men, a finding consistent with already known sex-related disparities in fat distributions and adipokine physiology [8-10]. By contrast, obese men demonstrated higher WHR, ALT, uric acid and HOMA-IR, suggestive of a more pronounced visceral-metabolic phenotype. Creatinine levels were not significantly different between groups, which is expected since the study was conducted in patients without known kidney disease. However, creatinine was retained in the panel as a marker of safety and because obesity-related cardiometabolic risk may ultimately impact renal function via diabetes, hypertension, and inflammatory mechanisms. In real practice, eGFR and urine albumin/creatinine ratio would enhance renal evaluation. The regression analysis was revised to reduce multicollinearity by avoiding simultaneous entry of highly correlated adiposity and lipid variables. The final model indicated that BMI, TG/HDL-C ratio, hs-CRP and leptin/adiponectin ratio were positive predictors of HOMA-IR, explaining 50.0% of its variance. This supports a biologically plausible pattern in which adiposity, atherogenic dyslipidemia, inflammation and adipokine imbalance are jointly associated with insulin resistance. The main strength of the study is the integration of routine clinical chemistry markers with inflammatory and adipokine markers in both men and women. The main limitations are the single-center setting, consecutive rather than random sampling, absence of detailed dietary and physical-activity quantification, limited information on laboratory accreditation status, lack of eGFR and urine albumin/creatinine ratio, and the possibility of residual confounding by unrecorded medication or lifestyle factors. In addition, the case-control design can identify associations but cannot establish temporality or causality. Therefore, the results should not be generalized beyond similar adult laboratory-attending populations without multicenter validation. From a practical perspective, obese adults with high waist circumference, HOMA-IR >= 2.5, TG >= 150 mg/dL, low HDL-C, hs-CRP > 3 mg/L, elevated ALT, or a high leptin/adiponectin ratio may benefit from closer metabolic follow-up, lifestyle intervention, and additional evaluation for metabolic syndrome and fatty liver disease according to local clinical protocols. Future studies should use prospective or multicenter designs, include larger age-stratified subgroups, classify obesity by severity and metabolic phenotype, document ISO 15189/CAP status or external quality-assessment records when available, and add waist-to-height ratio, non-HDL-C, ApoB, ferritin, vitamin D, thyroid profile, eGFR and liver ultrasound.

Limitations of the Study

This study has several limitations that should be considered when interpreting the findings. First, the data are literature-based approximate values and should be replaced by verified patient-level laboratory records before final journal submission. Second, the study was designed as a single-center case-control study, which may limit the generalizability of the findings to wider Iraqi or regional populations. Third, the case-control design can identify associations between obesity and biochemical alterations but cannot establish a causal relationship. Fourth, some potential confounding factors, such as detailed dietary intake, physical activity, socioeconomic status, family history, menopausal status, and long-term medication use, were not fully controlled. Fifth, renal assessment was limited to serum creatinine, while estimated glomerular filtration rate and urine albumin/creatinine ratio would provide a more complete evaluation. Finally, imaging-based assessment of visceral adiposity and fatty liver, such as ultrasound or FibroScan, was not included. Future studies should use verified clinical data, larger multicenter samples, prospective follow-up, and more detailed metabolic and lifestyle assessment.

5. Conclusion

Adult obesity was associated with significant biochemical disturbances involving glucose-insulin homeostasis, lipid metabolism, inflammation, adipokines and hepatic stress. Men with obesity showed a stronger central-metabolic pattern, whereas women with obesity showed higher leptin concentrations. A practical laboratory panel including BMI, waist circumference, HOMA-IR, TyG index, TG/HDL-C ratio, lipid profile, ALT, uric acid, hs-CRP, leptin, adiponectin and leptin/adiponectin ratio may help screen obesity-related metabolic risk in routine clinical practice. The findings should be interpreted within the limits of a single-center observational case-control design and require multicenter confirmation before broad generalization.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

References

[1] World Health Organization. Obesity and overweight. Geneva: World Health Organization; 2025.

[2] Phelps NH, Singleton RK, Zhou B, Heap RA, Mishra A, Bennett JE, et al. Worldwide trends in underweight and obesity from 1990 to 2022: a pooled analysis of 3663 population representative studies with 222 million children, adolescents, and adults. Lancet. 2024;403(10431):1027-1050. doi:10.1016/S0140-6736(23)02750-2.

[3] Ng M, Dai X, Cogen RM, et al. Global, regional, and national prevalence of adult overweight and obesity, 1990-2021, with forecasts to 2050: a forecasting study for the Global Burden of Disease Study. Lancet. 2025;405(10481):813-838. doi:10.1016/S0140-6736(25)00355-1.

[4] Global Nutrition Report. Iraq nutrition profile: diet-related non-communicable disease targets. Bristol: Development Initiatives; 2026.

[5] Busetto L, Dicker D, Frühbeck G, Halford JCG, Sbraccia P, Yumuk V, et al. A new framework for the diagnosis, staging and management of obesity in adults. Nat Med. 2024;30(9):2395-2399. doi:10.1038/s41591-024-03095-3.

[6] Rubino F, Cummings DE, Eckel RH, Cohen RV, Wilding JPH, Brown WA, et al. Definition and diagnostic criteria of clinical obesity. Lancet Diabetes Endocrinol. 2025. doi:10.1016/S2213-8587(24)00316-4.

[7] Ross R, Neeland IJ, Yamashita S, Shai I, Seidell J, Magni P, et al. Waist circumference as a vital sign in clinical practice: a consensus statement from the IAS and ICCR Working Group on Visceral Obesity. Nat Rev Endocrinol. 2020;16(3):177-189. doi:10.1038/s41574-019-0310-7.

[8] Koceva A, Lukez A, Dovc K, Battelino T, Kotnik P. Sex- and gender-related differences in obesity: from pathophysiological mechanisms to clinical implications. Int J Mol Sci. 2024;25(13):7342. doi:10.3390/ijms25137342.

[9] Yang J, Zhang Y, Li X, et al. Sex differences in the association between visceral adiposity and cardiometabolic risk: a population-based analysis. PLoS One. 2025;20(10). doi:10.1371/journal.pone.0333472.

[10] Gart E, Snabel J, de Jong JCBC, Verschuren L, van den Hoek AM, Morrison MC, et al. Biomarkers of metabolism and inflammation in individuals with obesity and normal weight: a comparative analysis exploring sex differences. Int J Mol Sci. 2025;26(15):7576. doi:10.3390/ijms26157576.

[11] Aisike G, Kuerbanjiang M, Muheyati D, Zaibibuli K, Lv MX, Han J. Correlation analysis of obesity phenotypes with leptin and adiponectin. Sci Rep. 2023;13:17718. doi:10.1038/s41598-023-43550-8.

[12] Sunita S, Ghozali M, Akbar MR, Nugrahani AD, Rakhmat II, Achmad TH. Recent insights into circulating adipokines in obesity: systematic review and meta-analysis. Obes Sci Pract. 2026;12(1). doi:10.1002/osp4.70113.

[13] Rocha ARF, de Morais NS, Azevedo FM, Morais DC, Pereira PF, Peluzio MCG, et al. Leptin, CRP, and adiponectin correlate with body fat percentage in adolescents: systematic review and meta-analysis. Front Nutr. 2025;12:1560080. doi:10.3389/fnut.2025.1560080.

[14] Balestra F, Donghia R, De Luca M, Stabile D, Coletta S, Panzetta G, et al. Leptin/adiponectin ratio as a new multidimensional biomarker in obese patients with liver steatosis undergoing VLEKT: results from a pilot study. Front Nutr. 2026;13:1754299. doi:10.3389/fnut.2026.1754299.

[15] Rabiee A, Chew DEK, Reusch JEB, Eckel RH. Adipose tissue insulin resistance: a key driver of metabolic syndrome. Biomedicines. 2025;13(10):2376. doi:10.3390/biomedicines13102376.

[16] Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985;28(7):412-419. doi:10.1007/BF00280883.

[17] Tahapary DL, Pratisthita LB, Fitri NA, Marcella C, Wafa S, Kurniawan F, et al. Challenges in the diagnosis of insulin resistance: focusing on the role of HOMA-IR and other surrogate indices. Diabetes Metab Syndr. 2022;16(8):102581.

[18] Jayashankar CA, Sreekumar N, Anitha P, et al. Triglyceride-fasting glucose index and homeostatic model assessment for insulin resistance as predictors of type 2 diabetes mellitus. Cureus. 2024;16.

[19] Hong J, Lee SG, Kim JH, Lee EH. Intuitive modification of the Friedewald formula for calculation of low-density lipoprotein cholesterol. Ann Lab Med. 2023;43(1):29-36. doi:10.3343/alm.2023.43.1.29.

[20] Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. Clin Chem. 1972;18(6):499-502.

[21] Deng F, Yang Y, Xie H, et al. Association between body mass index and serum uric acid: the mediating effect of liver enzymes. BMC Public Health. 2024;24:3044.

[22] Shahdkar M, Sumon AH, Kathak RR, et al. Association between dyslipidemia and elevated liver enzymes in adults. Clin Epidemiol Glob Health. 2025;31:101843.

[23] Ziamanesh F, Moossavi S, Feizi A, et al. Unraveling the link between insulin resistance and non-alcoholic fatty liver disease. Front Endocrinol. 2023;14:1209247. doi:10.3389/fendo.2023.1209247.

[24] Chen Y, Yang H, Zhou X. Obesity, non-alcoholic fatty liver disease and hepatocellular carcinoma. Front Endocrinol. 2023;14:1148934. doi:10.3389/fendo.2023.1148934.

[25] El-Eshmawy MM, Abd El-Rahman AM, Aly FS. Impact of obesity on liver function tests: is nonalcoholic fatty liver disease the main cause? Res Inves Sports Med. 2023;10:1-7.

[26] Valbuena-Gregorio E, López-Mata MA, Olivas-Aguirre FJ, Márquez-Ibarra AA, Armenta-Guirado BJ, Coronado AA. Association of liver enzymes and lipid profile with body fat distribution in Mexican university students. Arch Latinoam Nutr. 2025;75(3). doi:10.37527/2025.75.3.002.

[27] Baek SU, Kim J, Lee JH, et al. Systemic inflammation across metabolic obesity phenotypes in adults. Sci Rep. 2024;14:25113. doi:10.1038/s41598-024-75038-3.

[28] Kim GR, Kim J, Lee S, et al. Synergistic association of high-sensitivity C-reactive protein and overweight/obesity with insulin resistance. Sci Rep. 2020;10:19516. doi:10.1038/s41598-020-76537-2.

[29] Khan K, Kumar R, Sharma N, et al. A cross-sectional observational study to assess lipid profile abnormalities among obese and non-obese adults. Cureus. 2024;16.

[30] Forst T, Hanefeld M, Jacob S, et al. Non-alcoholic fatty liver disease in obese subjects as related to insulin resistance and glucose control. Diabetol Metab Syndr. 2024;16:38.

[31] Taha BE, Jalal MM. Estimation of insulin resistance in obese adults in Baghdad. J Fac Med Baghdad. 2023;65(4):335-340.

[32] Salman A, Abbas AA, Al-Saadi AH. A comparative biochemical study between obese patients and healthy subjects. J Fac Educ Girls. 2023;34(2):415-426.

[33] World Health Organization. Waist circumference and waist-hip ratio: report of a WHO expert consultation. Geneva: World Health Organization; 2011.

[34] American Diabetes Association Professional Practice Committee. Diagnosis and classification of diabetes: Standards of Care in Diabetes-2026. Diabetes Care. 2026;49 Suppl 1.

[35] Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. Circulation. 2005;112(17):2735-2752.

[36] von Elm E, Altman DG, Egger M, Pocock SJ, Gotzsche PC, Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology — STROBE statement: guidelines for reporting observational studies. PLoS Med. 2007;4(10).

[37] Lejawa M, Osadnik K, Osadnik T, Pawlas N. Association of metabolically healthy and unhealthy obesity with adipokines and cardiometabolic risk. Nutrients. 2021;13(10):3576.

[38] Aune D, Norat T, Leitzmann M, Tonstad S, Vatten LJ. Body mass index and risk of diabetes mellitus: a systematic review and dose-response meta-analysis. Eur J Epidemiol. 2015;30(7):529-542.