Impact of Weight Loss on Improvement of Insulin Resistance and Reduction of Fatty Hepatic Steatosis in Obese Patients with Type 2 Diabetes

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Abstract

Background: Obesity and type 2 diabetes mellitus (T2DM) are common metabolic disorders requiring effective non-pharmacological interventions. This study evaluated the effects of a regular dietary intervention program on anthropometric, body composition, and biochemical indicators using a randomized complete block design. Methods: Three groups were included: healthy control, infected (untreated) control, and experimental (dietary program). Anthropometric measures (BMI, BFM, VFA, TBW, protein, minerals, BMR), glycemic indicators (glucose, insulin, HbA1c, HOMA-IR, C-peptide), and liver enzymes (AST, ALT, ALP, GGT) were assessed using SPSS v26. Results: The infected group showed significant elevations in BMI (33.23), BFM (35.70), VFA (171.78 cm^2), glucose (176.01 mg/dL), HbA1c (8.52%), and C-peptide. The experimental group demonstrated marked improvement: reduced BMI, a ~34% decrease in VFA, a 16.2% decline in glucose (147.45 mg/dL), and an HbA1c reduction to 7.44% — clinically significant, as each 1% reduction in HbA1c lowers cardiovascular risk. C-peptide decreased, reflecting improved β-cell function. Liver enzymes remained within normal ranges, indicating reduced hepatic fat and inflammation. Conclusion: Regular dietary intervention significantly improved anthropometric, glycemic, and metabolic indicators in obese T2DM individuals, though values did not fully normalize. These findings support structured dietary programs as effective interventions for managing obesity-related metabolic dysfunction.

Keywords: Obesity, T2DM, Dietary intervention, Insulin resistance, HbA1c

1. Introduction

Obesity associated with type 2 diabetes is often accompanied by several health The coexistence of obesity with type 2 diabetes is accompanied by several health disorders, most notably nonalcoholic fatty liver disease (NAFLD), which can lead to impaired liver functional capacity [1]. The liver is one of the most vital organs in the body, responsible for maintaining homeostasis in metabolic processes. This function is influenced by numerous factors, disorders, and diseases, particularly autoimmune diseases, hypothyroidism, and other conditions related to energy production and consumption, especially fat metabolism [2]. Obesity is one such metabolic disorder and condition, often associated with other risk factors such as type 2 diabetes and its associated disorders, including insulin resistance and sensitivity [3]. Nonalcoholic fatty liver disease (NAFLD) is a clinical condition ranging from obesity, characterized by the accumulation of triglycerides (TG) in droplets in the cytoplasm of liver cells, to more severe cases with increasing accumulation of these fats in liver cells, leading to hepatocyte degeneration and varying degrees of liver fibrosis [4]. The metabolism and storage of triglycerides in liver cells are regulated by many genetic factors and enzymes [5]. Many previous studies have linked NAFLD with obesity and type 2 diabetes, which manifests as a disorder involving an increase in the secretion of certain liver factors associated with increased fat accumulation within liver cells, which increases glucose synthesis from non-carbohydrate materials through gluconeogenesis and decreases glycogen synthesis. This, in turn, promotes increased insulin secretion and then insulin resistance [3]. Adipose tissue is known as one of the body's endocrine organs, responsible for secreting Adipocytokines, immune factors involved in cellular and systemic regulation of various metabolic and inflammatory processes. Therefore, obesity leads to an excessive increase in adipose tissue mass due to an increase in the number and size of adipocytes, thus impairing its function, causing a loss of its functional and secretory properties, and increasing insulin resistance [6]. Insulin resistance is one of the primary metabolic disorders associated with both obesity and type 2 diabetes. Recent studies have indicated a significant correlation between obesity, type 2 diabetes, non-alcoholic fatty liver disease, and insulin resistance [7]. High blood insulin concentrations reflect either insulin sensitivity or resistance in cells. This increase in insulin levels is accompanied by a significant decrease in its effectiveness, which further contributes to elevated glucose levels [8]. Type 2 diabetes is a metabolic disorder significantly associated with obesity and generally manifests as liver disease, particularly non-alcoholic fatty liver disease (NAFLD). NAFLD is characterized by impaired fat metabolism and storage, as well as insulin resistance. Some studies have indicated that dietary management, following a regular diet, and exercising play a crucial role in mitigating these risks [9,10]. The initial stages of non-alcoholic fatty liver congestion include a marked increase in the levels of liver enzymes in the bloodstream (Alanine Aminotransferase ALT, Aspartate Transaminase AST, Alkaline Phosphatase ALP, and Gamma Glutamyl Transferase GGT). However, the level of these enzymes does not provide a sufficient indication to assess the state of liver congestion. The most accurate method is through taking a liver biopsy, through which the amount of triglycerides accumulated in liver cells is estimated. However, this method is characterized by high risk and high cost, in addition to the small size of the biopsy compared to the size of the liver [11].

Recently, numerous studies and research have focused on the importance of weight reduction and obesity prevention as procedural aspects of treating associated metabolic disorders and maintaining liver function. These studies have emphasized healthy diets, increased physical activity, and, in some cases, therapeutic or surgical interventions such as gastric sleeve surgery, gastric bypass, or liposuction [12,13]. This is why the current study is important: to highlight the effects of a high-protein, high-fat, low-carbohydrate diet combined with anti-obesity therapy and type 2 diabetes treatments on improving liver fat accumulation and insulin sensitivity and resistance as mechanisms for controlling and managing blood glucose levels.

2. Materials and Methods

2.1 Ethical statement

The study was conducted according to the guidelines of the Declaration of Helsinki and approved by the Research Ethics Committee at Al Iraqia University, College of Education; Approval No. CT23/286 IN 24/5/2026 Al-Iraqia University, Baghdad, Iraq. All participants provided written informed consent before participation in the study.

2.2 Study design and tests.

This case study was carried out on 60 adult Iraqi males from September 2023 to September 2025, participants (≥ 35 years old). The samples were selected through a diabetes clinic in Baghdad city, having obesity with a BMI ≥ 27. Otherwise, individuals were classified as normal (non-diabetic) if they were included as a healthy control group. Inclusion criteria were the age of 35– 55 years and consent to participate in the study. Exclusion criteria were no access to blood sample and/or laboratory results, suffering from diseases affecting weight, taking drugs affecting weight, and a lack of sufficient information to calculate BMI. Data related to demographic and social indices were collected via a general questionnaire. The height and weight of individuals were measured by an audiometer and an Esca scale, respectively, and BMI was calculated as weight (in kilograms) divided by height (in meters) squared. Analysis of fasting glucose levels in serum was measured using the enzymatic method with GLUC3 glucose hexokinase kit (Roche, Switzerland) on a Cobas instrument (Roche). Serum C-peptide levels were measured using the sandwich electrochemiluminescence (ECLIA/sandwich) immunoassay using Elecsys C-peptide kit (Roche), while serum insulin was measured using ECLIA/Sandwich immunoassay also on Cobas instrument (Roche). (IR) Indicators, detecting according to:

HOMA-IR = Fasting Insulin (μU/mL) x Fasting Glucose (mmol/L)/22.5 [14].

Assessment of HbA1C levels was performed for all participants in the total study group. Participants were classified as having type 2 diabetes mellitus (T2DM) if they reported having diabetes or were receiving diabetes treatment, with HbA1C levels between 5.7% and 9.4%, or fasting glucose levels (≥ 8 hours) between 5.6 and 6.9 mmol/L Clinical and biochemical data collection procedures for the study participants have been described previously (22). Laboratory assessment: Five milliliters of blood samples were collected from the participants in the fasted state. Serum levels of GGT, ALP, ALT, and AST were determined using an auto- analyzer (BT1500; Biotecnica Instrument) and Pars Azmun standard kits.

2.3 Dietary program

Design the experimental group's dietary program to be a system low in carbohydrates and rich in proteins and animal fats.

2.4 Statistical Analysis

The data collected from the study samples were analyzed to interpret the results and reach accurate scientific conclusions. This was carried out using all statistical procedures available in SPSS (Statistical Package for the Social Sciences) version 26, which is one of the most widely used programs in medical and biological research due to its advanced tests for descriptive and inferential analysis. Microsoft Excel 2019 was also used to organize databases, perform some preliminary calculations, and create charts that helped present the results in a simplified and visual manner. The study followed a Randomized Complete Block Design (RCBD). (REFERENCE)

3. Results

3.1: Comparative Analysis of Biochemical Parameters among Study Groups

All indicators in Table 4-1 showed significant differences among groups ($p < 0.001$). The negative control group recorded the highest glucose concentration (176.0 ± 22.8 mg/dl^{a}), highest HbA1c ($8.52 \pm 0.38\%^{a}$), and highest C-peptide (7.40 ± 0.29^{a}). The experimental group showed intermediate values (glucose: 147.5 ± 33.5^{b}, HbA1c: 7.44 ± 0.34^{b}, C-peptide: 2.47 ± 0.16^{c}). The positive control group recorded the lowest glucose (95.7 ± 10.5^{c}) and HbA1c (5.32 ± 0.51^{c}). All differences were significant according to the LSD test.

Table 3-1: Glucose concentration, HbA1c, and C-peptide levels in Study groups.

Parameters / Groups	Glucose concentration (mg/dl)	HbA1c (%)	C-Peptide (mg/dl)
Positive control	95.665 ± 10.535^{c}	5.324 ± 0.514^{c}	2.950 ± 0.204^{b}
Negative control	176.011 ± 22.768^{a}	8.520 ± 0.381^{a}	7.404 ± 0.288^{a}
Experimental	147.450 ± 33.505^{b}	7.435 ± 0.339^{b}	2.465 ± 0.157^{c}

LSD (α=0.05)	15.3	0.265	0.141
P-value	<0.001*	<0.001*	<0.001*

*Significant difference between groups (p value < 0.05)
Values are expressed as Mean ± SD (n = 20). Different superscript letters within the same column indicate significant differences between groups at p < 0.05 according to the LSD test

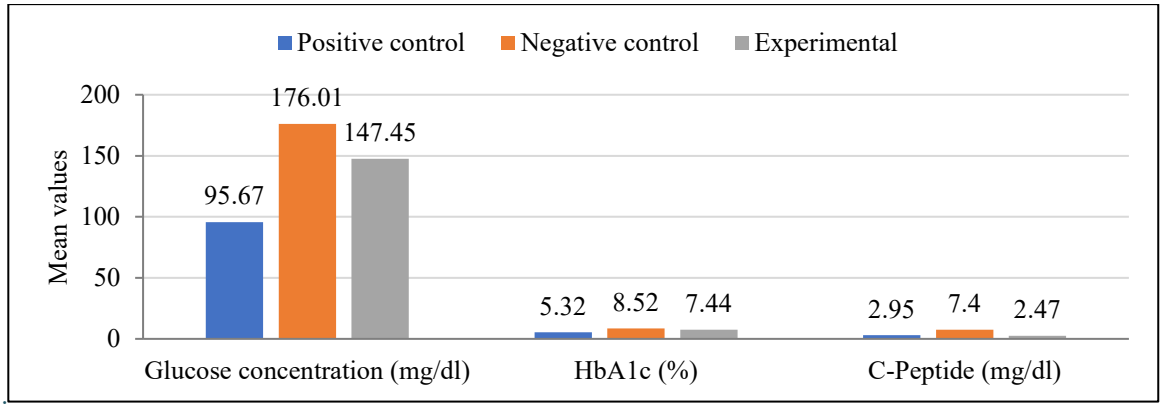


Figure 3-1 **Glucose concentration, HbA1c, and C-peptide levels in study groups.**

3.2: Results in Table (3.2) showed significant differences (p < 0.001). The negative control had the highest insulin (32.18 ± 2.04 µU/ml[a]) and HOMA-IR (13.98 ± 0.63[a]), but intermediate QUICKI (0.351 ± 0.03[b]). The positive control showed intermediate insulin (5.78 ± 0.08[b]), lowest HOMA-IR (0.99 ± 0.21[c]), and highest QUICKI (0.536 ± 0.05[a]). The experimental group had the lowest insulin (4.24 ± 0.19[c]) and QUICKI (0.305 ± 0.06[c]), with improved HOMA-IR (1.99 ± 1.68[b]) vs negative control. All LSD tests confirmed significant pairwise differences.

Table 3- 2: Insulin, HOMA-IR, and QUICKI index levels in Study groups.

Parameters / Groups	Insulin (µU/ml)	HOMA-IR (index)	QUICKI Index (index)
Positive control	5.779 ± 0.081[b]	0.990 ± 0.212[c]	0.536 ± 0.045[a]
Negative control	32.175 ± 2.044[a]	13.982 ± 0.631[a]	0.351 ± 0.031[b]
Experimental	4.240 ± 0.188[c]	1.988 ± 1.683[b]	0.305 ± 0.056[c]
LSD (α=0.05)	0.751	0.661	0.029
P-value	<0.001*	<0.001*	<0.001*

*Significant difference between groups (p value < 0.05)
Values are expressed as Mean ± SD (n = 20). Different superscript letters within the same column indicate significant differences between groups at p < 0.05 according to the LSD test

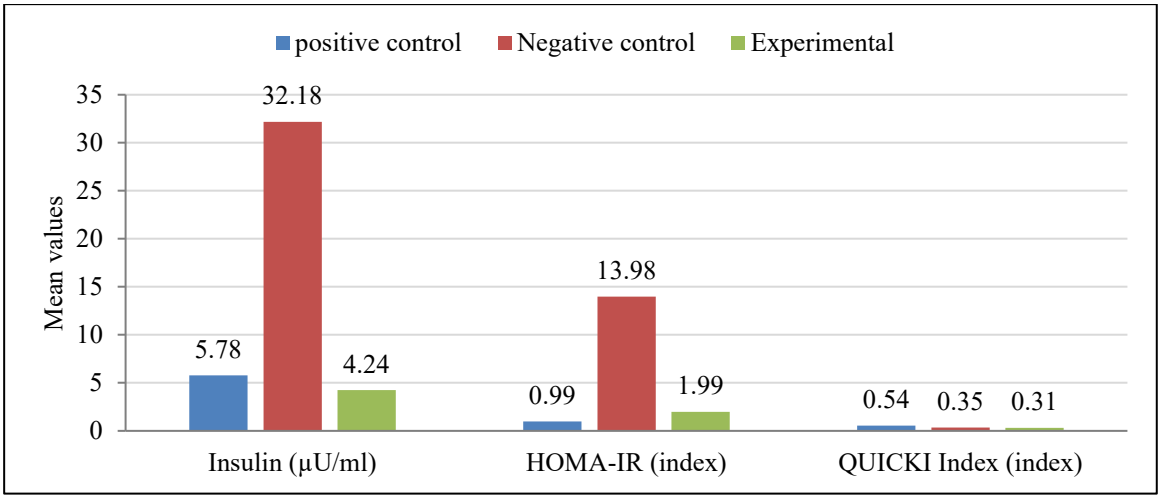


Figure3-2 Insulin, HOMA-IR, and QUICKI index levels in study groups.

3.3: The results in Table (4-3) showed significant group differences (p < 0.001). The negative control had the highest urea (54.66 ± 3.13 mg/dl[a]), uric acid (6.88 ± 0.18[a]), and creatinine (1.13 ± 0.15[a]). The positive control recorded the lowest values (urea: 27.85 ± 1.70[c], uric acid: 5.26 ± 0.20[c], creatinine: 0.91 ± 0.10[c]). The experimental group showed intermediate levels (urea: 45.41 ± 4.61[b], uric acid: 6.57 ± 0.26[b], creatinine: 1.00 ± 0.11[b]). indicating partial improvement in renal function.

Table 3-3. Kidney function parameters (urea, uric acid, and creatinine) in Study groups.

Parameters / Groups	Urea (mg/dl)	Uric acid (mg/dl)	Creatinine (mg/dl)
Positive control	27.850 ± 1.698^{c}	5.263 ± 0.201^{c}	0.909 ± 0.104^{c}
Negative control	54.660 ± 3.131^{a}	6.880 ± 0.184^{a}	1.133 ± 0.151^{a}
Experimental	45.410 ± 4.606^{b}	6.569 ± 0.260^{b}	1.002 ± 0.109^{b}
LSD (α=0.05)	2.128	0.138	0.078
P-value	<0.001*	<0.001*	<0.001*

*Significant difference between groups (p value < 0.05)
Values are expressed as Mean ± SD (n = 20). Different superscript letters within the same column indicate significant differences between groups at p < 0.05 according to the LSD test

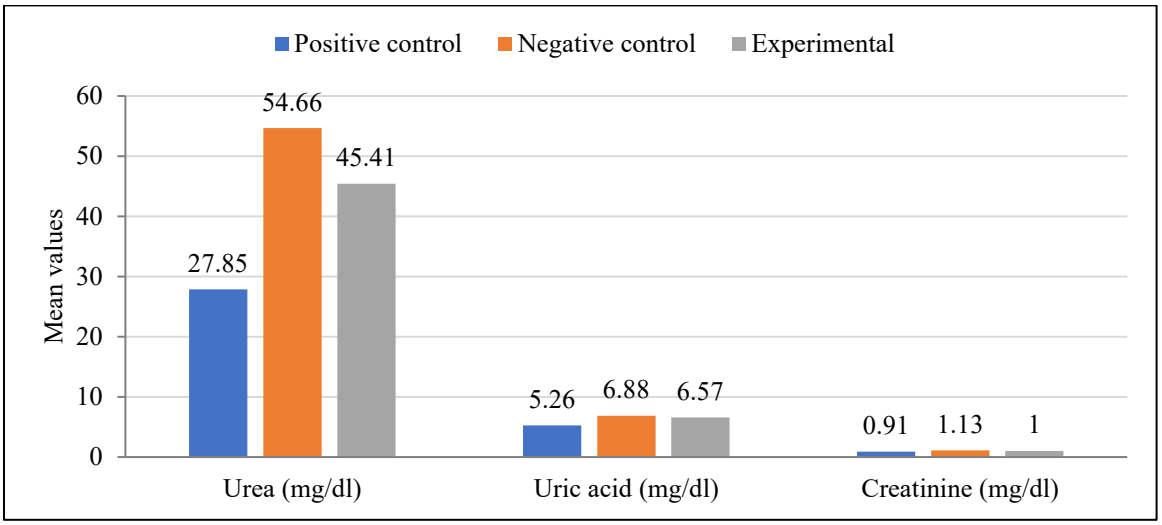


Figure3-3. Kidney function parameters (urea, uric acid, and creatinine) in study groups.

3-4: The results showed in Table 3-4 that all liver enzymes differed significantly among groups (p < 0.001). The positive control had the highest ALT (40.35 ± 1.12 U/L^{a}), AST (39.75 ± 0.57^{a}), and GGT (28.30 ± 0.35^{a}), with intermediate ALP (98.67 ± 0.39^{b}). The negative control showed lowest AST (16.30 ± 0.25^{c}) and highest ALP (123.45 ± 0.48^{a}), with intermediate ALT (22.05 ± 1.70^{b}). The experimental group recorded the lowest ALP (93.44 ± 0.48^{c}) and GGT (11.26 ± 0.69^{c}), with near-normal ALT (19.01 ± 0.94^{c}) and AST (17.73 ± 0.31^{b}). All LSD tests confirmed significant differences.

Table3- 4. Liver function parameters (ALT, AST, ALP, and GGT) in Study g\oups.

Parameters / Groups	ALT (U/L)	AST (U/L)	ALP (U/L)	GGT (U/L)
Positive control	40.349 ± 1.120^{a}	39.745 ± 0.574^{a}	98.672 ± 0.393^{b}	28.300 ± 0.346^{a}
Negative control	22.047 ± 1.703^{b}	16.300 ± 0.252^{c}	123.448 ± 0.477^{a}	19.895 ± 2.526^{b}
Experimental	19.012 ± 0.941^{c}	17.725 ± 0.305^{b}	93.442 ± 0.482^{c}	11.261 ± 0.691^{c}
LSD (α=0.05)	0.82	0.255	0.286	0.965
P-value	<0.001*	<0.001*	<0.001*	<0.001*

*Significant difference between groups (p value < 0.05)
Values are expressed as Mean ± SD (n = 20). Different superscript letters within the same column indicate significant differences between groups at p < 0.05 according to the LSD test.

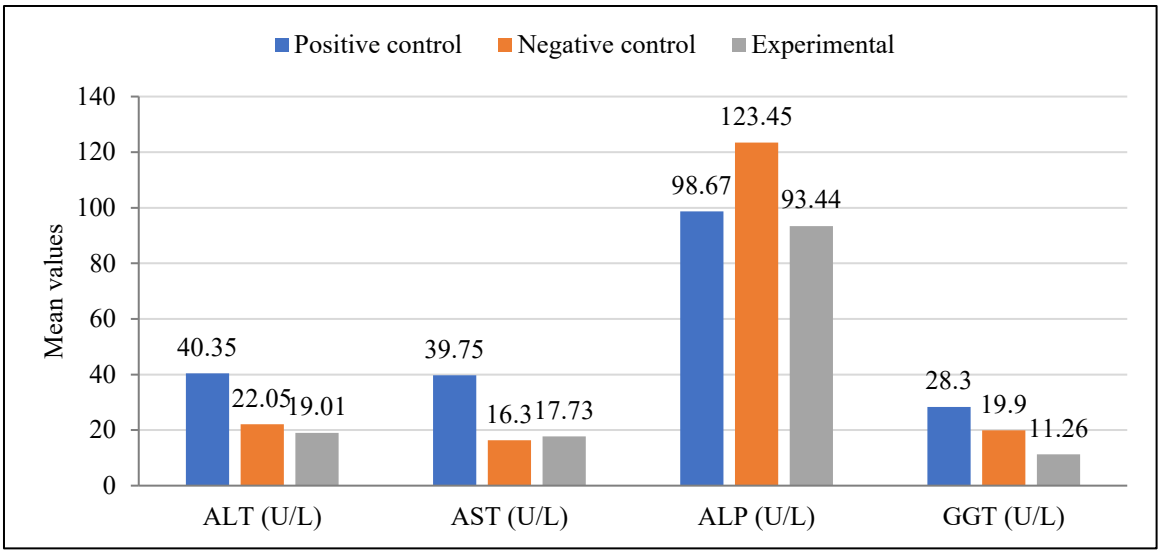


Figure 4- 4. Liver function parameters (ALT, AST, ALP, and GGT) in study groups.

3-5: The results in able (3-5) showed that TBW and intracellular water differed significantly ($p < 0.001$): negative control had the highest (TBW: 52.71 ± 5.08 L^a; ICW: 31.58 ± 3.39^a), positive control the lowest (33.64 ± 5.19^c; 25.14 ± 3.26^c), and experimental group intermediate (46.60 ± 4.10^b; 27.88 ± 3.23^b). BMR was highest in positive control (1925.25 ± 52.43 kcal^a), lowest in experimental group (1699.55 ± 76.74^c). Proteins showed limited significance (p=0.049). No significant differences were found for extracellular water, minerals, or WHR.

Table 3- 5. Body composition parameters (TBW, proteins, intracellular water, extracellular water, minerals, BMR, and WHR) in Study group.

Parameters / Groups	TBW (L)	Proteins (kg)	Intracellular water (L)	Extracellular water (L)	Minerals (kg)	BMR (kcal)	WHR (%)
Positive control	33.643 ± 5.192^c	11.650 ± 2.286^b	25.136 ± 3.257^c	14.646 ± 2.480^a	3.162 ± 1.148^a	1925.250 ± 52.427^a	0.824 ± 0.349^a
Negative control	52.705 ± 5.077^a	13.430 ± 2.297^a	31.575 ± 3.389^a	16.500 ± 2.996^a	3.691 ± 1.466^a	1855.500 ± 50.364^b	1.063 ± 0.509^a
Experimental	46.595 ± 4.097^b	12.060 ± 2.437^a	27.875 ± 3.232^b	15.745 ± 3.374^a	3.611 ± 1.520^a	1699.550 ± 76.736^c	0.830 ± 0.350^a
LSD (α=0.05)	3.048	1.482	2.085	1.882	0.879	38.64	0.259
P-value	<0.001*	0.049*	<0.001*	0.149	0.435	<0.001*	0.118

*Significant difference between groups (p value < 0.05)

Values are expressed as Mean ± SD (n = 20). Different superscript letters within the same column indicate significant differences between groups at $p < 0.05$ according to Duncan's multiple range test.

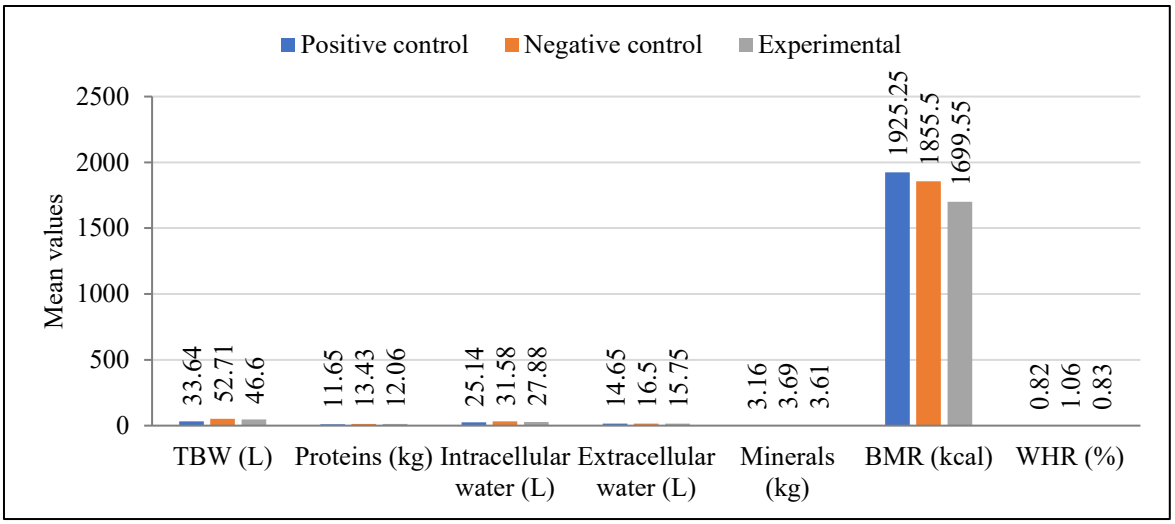


Figure 3- 5. Body composition parameters (TBW, proteins, intracellular water, extracellular water, minerals, BMR, and WHR) in study groups.

3-6 lipid profile results:

All lipid parameters showed significant differences ($p < 0.001$). The negative control had the highest cholesterol (178.75 ± 5.47^{a}), triglycerides (181.94 ± 5.94^{a}), and LDL (130.56 ± 3.18^{a}), with the lowest HDL (34.39 ± 2.02^{c}). The positive control recorded the lowest cholesterol (137.71 ± 8.21^{c}), triglycerides (127.54 ± 5.12^{c}), and VLDL (26.49 ± 2.12^{c}), with the highest HDL (41.16 ± 3.64^{a}). The experimental group showed intermediate values except for the highest VLDL (46.41 ± 4.49^{a}) and the lowest LDL (99.36 ± 1.43^{c}). All LSD tests confirmed significant differences.

Table 3-6. Lipid profile parameters (cholesterol, triglycerides, HDL, LDL, and VLDL) in Study groups.

Parameters / Groups	Cholesterol (mg/dl)	Triglycerides (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)
Positive control	137.710 ± 8.210^{c}	127.539 ± 5.122^{c}	41.156 ± 3.642^{a}	102.031 ± 2.416^{b}	26.489 ± 2.119^{c}
Negative control	178.749 ± 5.468^{a}	181.941 ± 5.941^{a}	34.386 ± 2.015^{c}	130.555 ± 3.178^{a}	31.124 ± 1.565^{b}
Experimental	168.701 ± 5.482^{b}	160.380 ± 6.968^{b}	37.800 ± 2.018^{b}	99.360 ± 1.428^{c}	46.414 ± 4.491^{a}
LSD (α=0.05)	4.125	3.835	1.691	1.550	1.903
P-value	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*

*Significant difference between groups (p value < 0.05)

-Values are expressed as Mean ± SD (n = 20).

-Different superscript letters within the same column indicate significant differences between groups at $p < 0.05$ according to the LSD test.

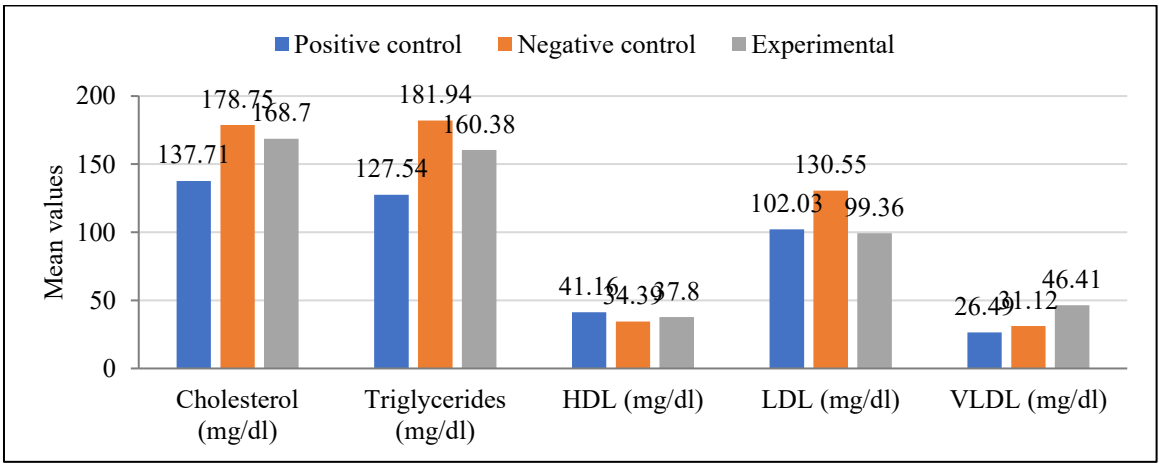


Figure4-6. Lipid profile parameters (cholesterol, triglycerides, HDL, LDL, and VLDL) in study groups.

3-7: All body composition parameters differed significantly ($p < 0.001$). The negative control had the highest BMI (33.23 ± 0.84^{a}), BCM (44.30 ± 1.15^{a}), BFM (35.70 ± 1.21^{a}), VFA (171.78 ± 1.84^{a}), and PBF (34.65 ± 1.68^{a}). The positive control recorded the lowest values (BMI: 23.33 ± 1.12^{c}, BCM: 36.38 ± 0.94^{c}, BFM: 10.50 ± 0.92^{c}, VFA: 77.43 ± 1.53^{c}, PBF: 13.32 ± 0.85^{c}). The experimental group showed intermediate values across all parameters. All LSD tests confirmed significant pairwise differences.

Table 3- 7. Body mass index (BMI), body cell mass (BCM), body fat mass (BFM), visceral fat area (VFA), and percent body fat (PBF) in Study groups.

Parameters / Groups	BMI (kg/m²)	BCM (kg)	BFM (kg)	VFA (cm²)	PBF (%)
Positive control	23.331 ± 1.122[c]	36.380 ± 0.940[c]	10.496 ± 0.915[c]	77.425 ± 1.532[c]	13.315 ± 0.847[c]
Negative control	33.232 ± 0.836[a]	44.300 ± 1.149[a]	35.697 ± 1.212[a]	171.775 ± 1.844[a]	34.645 ± 1.683[a]
Experimental	24.714 ± 1.067[b]	39.555 ± 1.305[b]	26.446 ± 1.072[b]	113.815 ± 1.817[b]	29.735 ± 1.277[b]
LSD (α=0.05)	0.643	0.723	0.680	1.099	0.832
P-value	<0.001*	<0.001*	<0.001*	<0.001*	<0.001*

*Significant difference between groups (p value < 0.05)
Values are expressed as Mean ± SD (n = 20). Different superscript letters within the same column indicate significant differences between groups at p < 0.05 according to the LSD test.

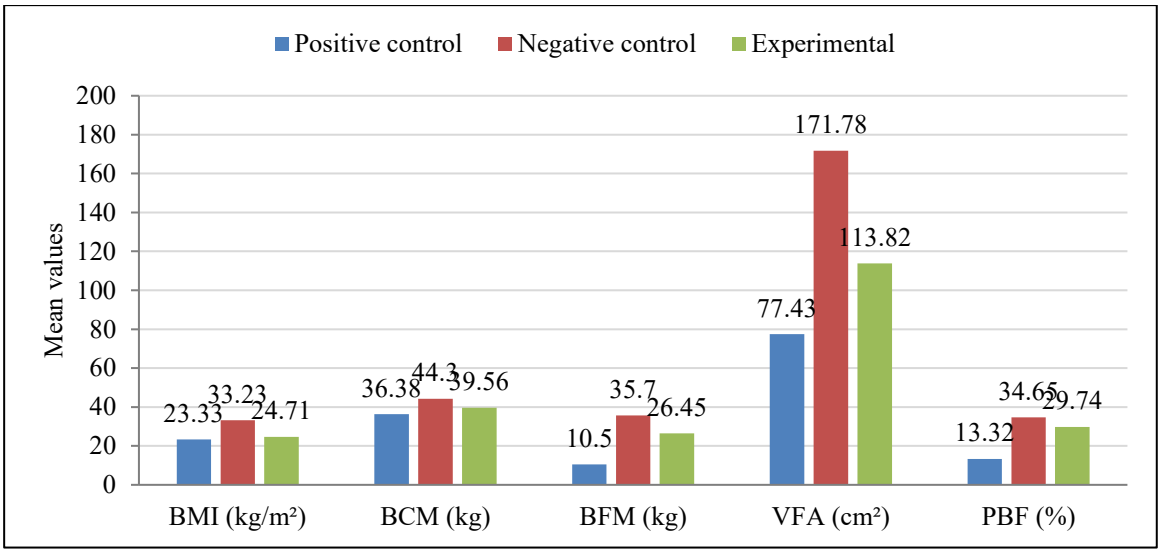
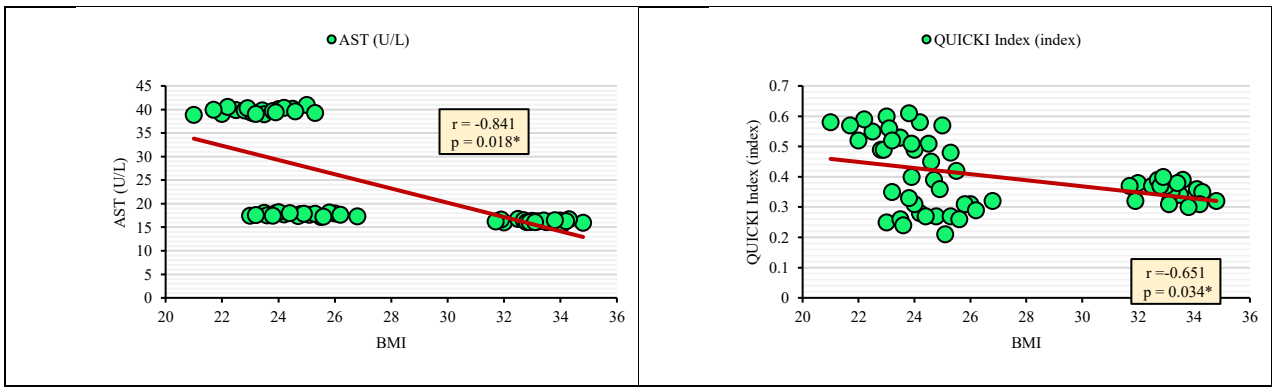


Figure 4- 7. Body mass index (BMI), body cell mass (BCM), body fat mass (BFM), visceral fat area (VFA), and percent body fat (PBF) in study groups.

4-8 : Correlation Analysis between Body Mass Index and Biochemical Parameters

Significant correlations ($p < 0.05$) were observed between BMI and all tested biochemical parameters. BMI showed strong positive correlations with glucose ($r = 0.831$), HbA1c ($r = 0.621$), insulin ($r = 0.827$), urea ($r = 0.861$), and ALP ($r = 0.561$). Negative correlations were found with AST ($r = -0.841$), ALT ($r = -0.652$), and QUICKI ($r = -0.651$). These findings indicate that higher BMI is associated with impaired glycemic regulation, reduced insulin sensitivity, elevated renal markers, and altered liver enzyme profiles, reflecting underlying metabolic stress.



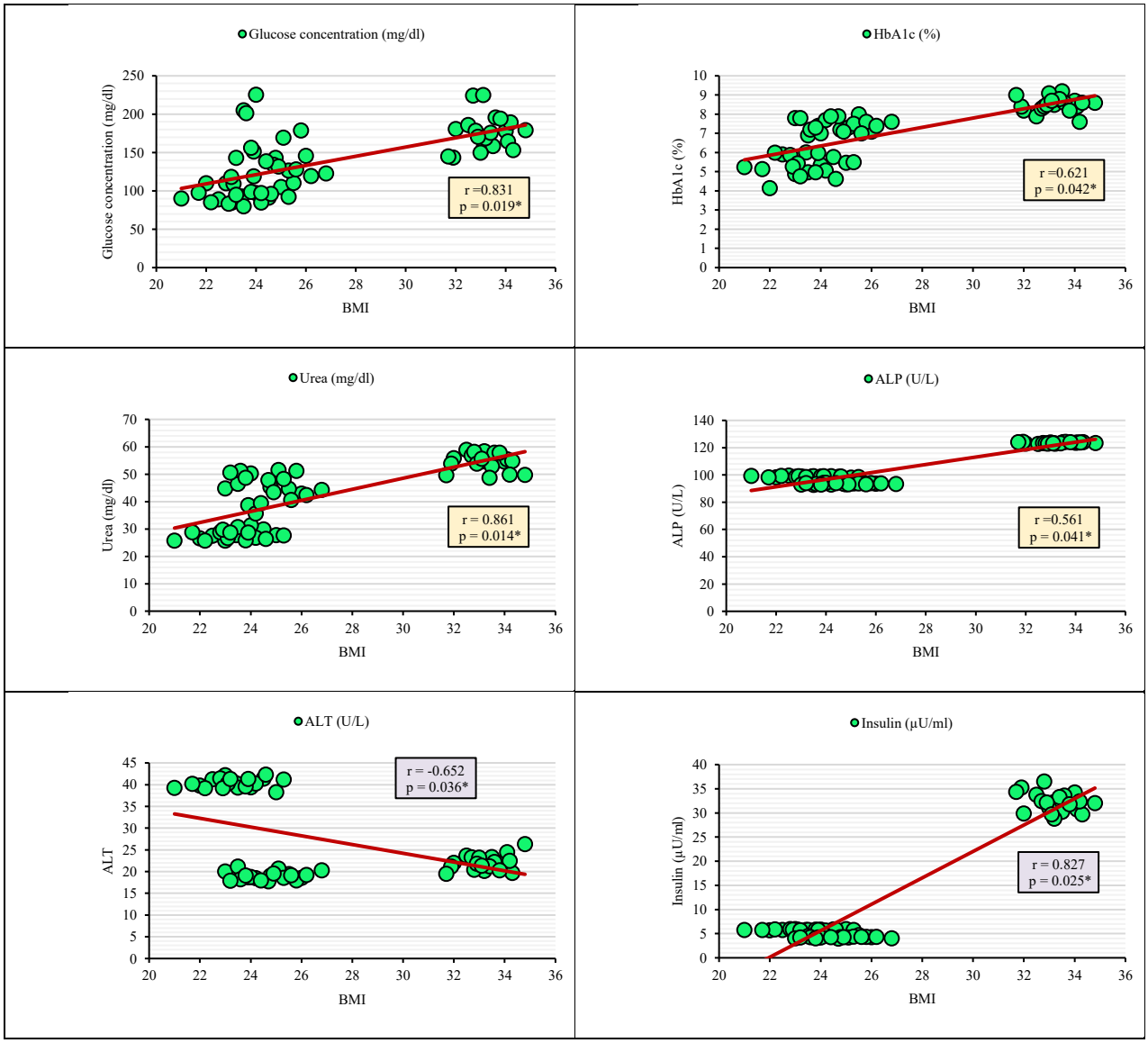
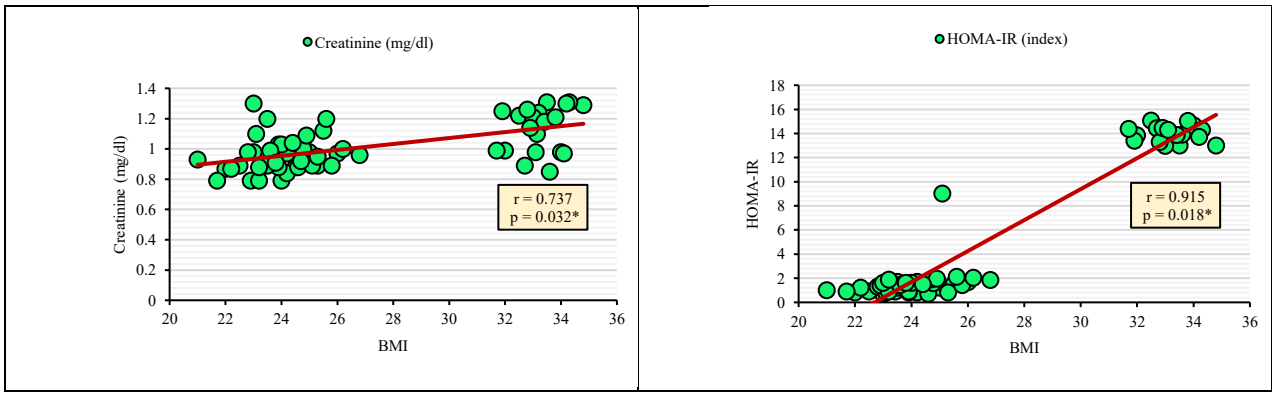


Figure 3-8: Correlation between Body Mass Index (BMI) and Selected Biochemical Parameters among the Studied Group

Significant positive correlations were found between BMI and creatinine (r = 0.737, p = 0.032), C-peptide (r = 0.902, p = 0.004), HOMA-IR (r = 0.915, p = 0.018), uric acid (r = 0.561, p = 0.049), and TBW (r = 0.528, p = 0.046). These associations indicate that higher BMI is linked to increased insulin resistance, compensatory hyperinsulinemia, and elevated renal markers. In contrast, GGT (r = 0.361, p = 0.096) and total body protein (r = 0.361, p = 0.087) showed non-significant correlations. Overall, elevated BMI correlates with impaired glucose-insulin homeostasis and early renal metabolic stress.



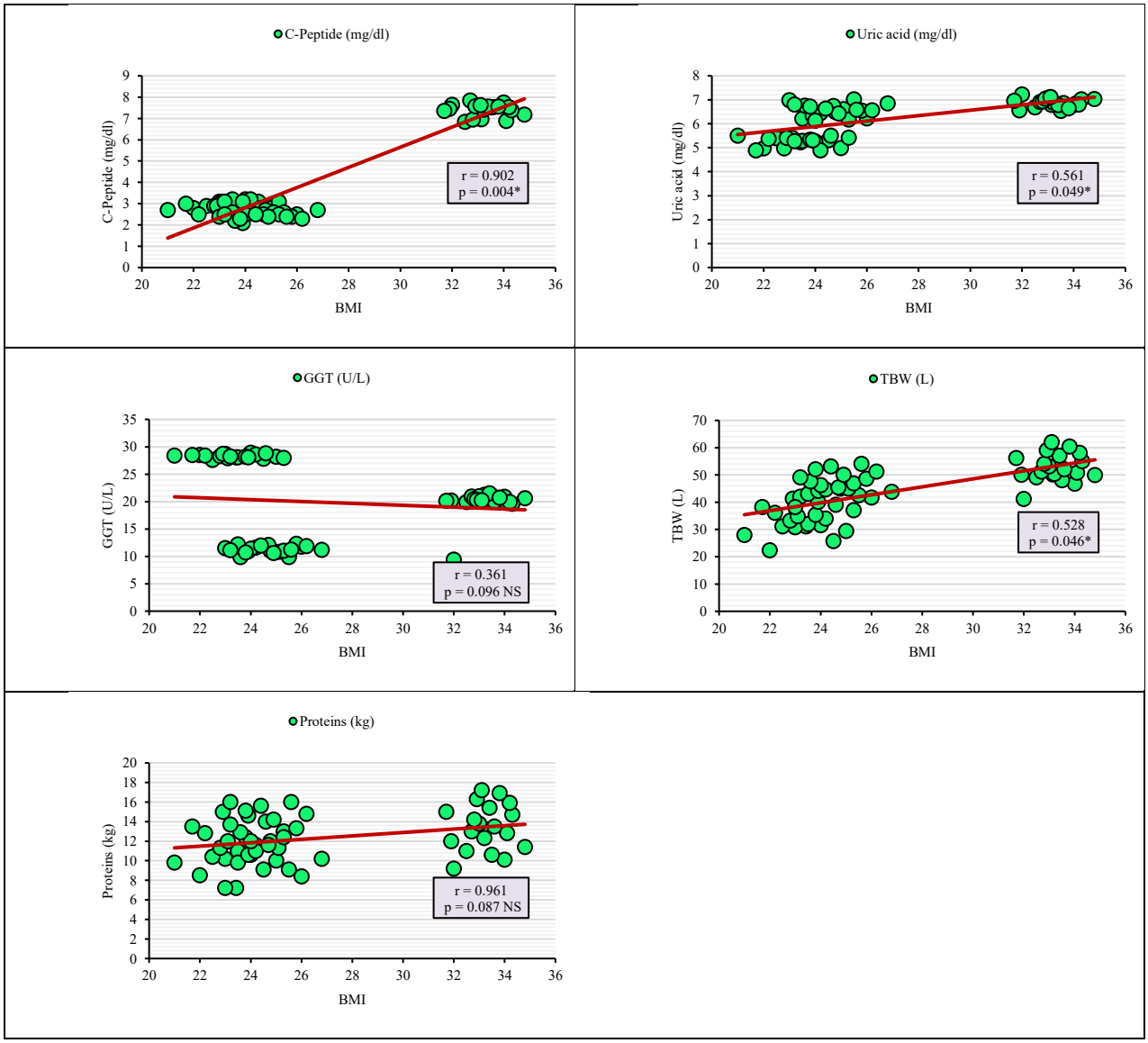


Figure 4-9. Correlation between Body Mass Index (BMI) and Renal, Metabolic, and Body Composition Parameters among the Studied Individuals.

4. Discussion

These results clearly reflect the effect of regular dietary intervention in improving health indicators associated with obesity and type 2 diabetes mellitus. The variable values for the healthy control group represented healthy individuals free from any disease, with recorded BMI values ranging between 18.5 and 24.9, which fall within the normal range [15. The BFM value was low, reflecting a normal fat percentage [16], while a VFA of less than 100 cm^2 is considered normal and not associated with metabolic risks [17]. The significant differences recorded in the values of the studied variables in the negative control group (infected) confirmed the highest significant elevations and indicators of metabolic disturbances associated with obesity and type 2 diabetes mellitus, as the variable values showed highly significant increases compared to the healthy control group. An increase in BMI > 30 is classified as obesity according to the World Health Organization and is associated with an increased risk of developing type 2 diabetes mellitus [15] The elevated values of BMI = 33.23, BFM = 35.70, and VFA = 171.78 in the infected control group can be explained based on insulin resistance, which leads to increased fat storage in adipose tissue, particularly visceral fat. Elevated insulin also contributes to the stimulation of lipoprotein lipase and inhibits lipolysis [18]. Furthermore, elevated visceral fat (VFA = 171.78) is closely associated with increased secretion of inflammatory cytokines (TNF-α, IL-6), which exacerbate insulin resistance [19].

The experimental group underwent the regular dietary program, which was reflected in a marked improvement compared to the infected control group: all studied variables showed improvement in BMI, which is attributed to calorie reduction and improved balance between energy production and consumption, thereby promoting weight loss [20]. The reduction in BFM occurs as a result of stimulating lipolysis due to caloric deficit and meal regulation [21]. Similarly, the reduction in VFA by approximately 34% is clinically significant because visceral fat is the most metabolically active and most strongly associated with cardio-metabolic risks. Regular dietary intervention improves insulin sensitivity and reduces systemic inflammation [22].

Among the most prominent reasons that may explain the elevated TBW in the infected control group (52.70 L) are several physiological factors associated with obesity and type 2 diabetes mellitus, which may include increased total body mass water constitutes approximately 50–60% of body weight, so the greater the weight, the greater the

total body fluids. Individuals with obesity have greater body mass and therefore a larger total body water volume [23,24]. It could also be due to fluid retention related to insulin resistance, as a recent study found that insulin resistance leads to increased sodium reabsorption in the renal tubules through stimulation of ENaC (epithelial sodium channels), resulting in fluid retention. Mild edema associated with chronic systemic inflammation, or the fact that obesity elevates antidiuretic hormone (ADH) levels through inflammatory pathways [25]. As for what explains the lower TBW in the experimental group (46.60 L) compared to the infected control group, it reflects the success of the dietary program in reducing weight and fat, along with the associated fluid retention [26].

The elevation in protein mass in both the infected and experimental groups (13.43 and 12.06 kg, respectively), with no significant difference between them, may be explained by the fact that fat-free mass (FFM) increases with total body weight. Individuals with obesity have greater muscle mass because muscles bear a larger body weight [27]. This is supported by recent studies showing that obese patients with type 2 diabetes retain higher muscle mass than healthy normal-weight individuals in the early stages of the disease [28].

The results for the blood glucose variable in the healthy control group showed a value of (95.67), which falls within the normal range (<100 mg/dL) according to the American Diabetes Association [29]. In contrast, its value in the infected control group was (176.01), which exceeds the diagnostic threshold for diabetes (≥126 mg/dL), attributed to insulin resistance that impairs the ability of the liver and muscles to uptake glucose from the blood [30]. The experimental group recorded a blood glucose value of 147.45, indicating a significant reduction of 16.2% compared to the infected control group, although it remained within the diabetic range. This improvement is attributed to the regular dietary program, which enhances insulin sensitivity by reducing intramyocellular lipid accumulation and improving GLUT4 regulation [31,32].

The results of the present study regarding the glycated hemoglobin (HbA1c) variable showed a highly significant elevation in the infected control group (8.52%), indicating poor chronic glycemic control over the preceding 2–3 months. This elevation reflects chronic exposure to high glucose levels, leading to increased non-enzymatic binding to hemoglobin (glycation) [33]. The lower HbA1c in the experimental group (7.44%) compared to the infected control group is of significant clinical relevance, as the UKPDS demonstrated that each 1% reduction in HbA1c decreases the risk of cardiovascular complications [33,34].

C-Peptide is produced in equimolar amounts to insulin (1:1) and is used as an indicator of β-cell function; therefore, its elevation indicates stress on these cells [35]. The marked increase in C-Peptide levels can be interpreted as a compensatory hyperinsulinemia state resulting from insulin resistance in pancreatic β-cells. In contrast, the decrease in the experimental group is notably significant. This can be attributed to the regular dietary program substantially reducing insulin resistance, thereby decreasing the need for compensatory insulin secretion, and consequently reducing both insulin and C-Peptide levels. This state is considered a key therapeutic goal for prolonging the lifespan of insulin-secreting cells [33,36].

The results of liver enzyme activity in the study samples showed variation in enzyme levels, where a slight elevation in AST, ALT, ALP, and GGT was observed in the healthy control group, and this difference falls within the normal range for adults. Their levels were decreased in the experimental group; this decrease is atypical compared to what is expected in obesity and diabetes, where enzyme levels would normally be elevated due to metabolic dysfunction-associated Steatotic liver disease (MASLD). This may be explained by the fact that the liver health status in the experimental group could be affected by medications for diabetes or obesity, such as metformin, which may lower ALT [37,38]. We differ in our opinion from studies that assumed this decrease is due to advanced MASLD with fibrosis, where liver enzymes decline as fibrosis progresses and functional hepatocytes are replaced by fibrous tissue, thereby reducing ALT and AST secretion [40,41]. The decrease could also be attributed to protein malnutrition associated with obesity [42]. Accordingly, this decrease within the normal range of liver enzymes reflects an additional improvement induced by the dietary program in reducing hepatic fat and inflammation [43, 44].

5. Conclusion

The regular dietary program markedly improved all standard biochemical indicators in the experimental group versus the untreated infected controls, though values did not fully normalize to healthy control levels. The healthy group's lower TBW is attributable to lower body mass and not a negative finding. Significant associations were found between BMI and creatinine, C-peptide, HOMA-IR, and TBW. Strong effects of C-peptide and HOMA-IR on BMI confirm obesity as a key driver of insulin resistance and compensatory hyperinsulinemia. Non-significant relationships for GGT and total protein suggest certain hepatic and structural parameters remain less affected in early BMI elevation. Overall, the results highlight a metabolic imbalance linked to increased adiposity, reflecting the interconnected effects of obesity on glucose metabolism, renal function, and systemic homeostasis. Finally, these results have contributed to improved liver function, detoxification, and a relative return to normalcy in non-alcoholic fatty liver disease.

Abbreviations: Table 3-8 Abbreviations

Abbreviation	Full Words
AASLD	American Association for the Study of Liver Diseases
ALP	Alkaline phosphatase
ALT	Alanine Aminotransferase,

AST	aspartate aminotransferase
BCM	body cell mass
BFM	Body Fat Mass
BMI	Body Nass Index
BMR	Basal Metabolic Rate
C-peptide)	connecting peptide
ECLIA	electrochemiluminescence immunoassay
GGT	gamma-Glutamyl transferase
GLUC3	glucose hexokinase kit
HbA1c	hemoglobin A1C
HDL	High Density Lipoprotein
HOMA-IR	Insulin Resistance
LDL	Low-Density Lipoprotein
LSD	stands for Lysergic acid diethylamide
NAFLD	Nonalcoholic fatty liver disease
PBF	percent body fat
p-value	probability value
QUICKI	Quantitative Insulin Sensitivity Check Index
RCBD	Randomized Complete Block Design
SD	Standard Deviation
SPSS	Statistical Package for the Social Sciences
T2DM	Type 2 diabetes mellitus
TBW	Total Body Water
TG	triglycerides
VFA	Visceral Fat Area
VLDL	Very Low Density Lipoprotein
WHR	Waist-to-Hip Ratio

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