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Original Article

Biochemical Profile and Nutritional overview of Insulin Resistance,Fatty Liver,and Diabetes Mellitus

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ABSTRACT

Background: Insulin resistance (IR) links non-alcoholic fatty liver disease (NAFLD) and type 2 diabetes mellitus (T2DM), with global NAFLD prevalence at 38% and T2DM comorbidity at 23% (1). Nutritional deficiencies, notably vitamin D and magnesium, exacerbate IR(2).
Objective: Evaluate a nutritional intervention's impact on IR, NAFLD markers, and glycemic control Methods: Pre-post interventional study of 114 patients (mean age 44.12 years, 54.4% female). Outcomes included anthropometrics, HOMA-IR, HbA1c, lipids, vitamin D, magnesium, and thyroid function, analyzed via SPSS v20 (paired t-tests/Wilcoxon, $p<0.05$)(3).
Results: Post-intervention: weight -8.29 kg ($p=0.001$), HOMA-IR 11.39 to 4.76 ($p=0.001$), HbA1c 8.99% to 7.06% ($p=0.001$), vitamin D 14.68 to 30.32 ng/mL ($p=0.001$)(3).
Conclusion: Targeted nutrition significantly improves IR, NAFLD, and T2DM parameters, supporting its role in metabolic management.Keywords: Insulin resistance, NAFLD, T2DM, nutritional intervention, vitamin D(3)(4).

Keywords*: Insulin resistance, Non-alcoholic fatty liver disease, Type 2 diabetes mellitus, Nutritional intervention, Vitamin D supplementation.*

INTRODUCTION

Insulin resistance (IR) constitutes a central pathophysiological nexus between metabolic dysfunction-associated steatotic liver disease (MASLD, formerly NAFLD) and type 2 diabetes mellitus (T2DM), driving hepatic steatosis, inflammation, and progression to fibrosis(5).Recent epidemiology reveals NAFLD prevalence at 38% globally (2016-2019), rising to 56-60% in T2DM cohorts, with lean NAFLD patients exhibiting 15.6% diabetes and 22.9% prediabetes rates(6)(7)(1).Bidirectional causality amplifies risks: IR promotes lipogenesis and oxidative stress, while hepatic fat impairs insulin signaling, compounded by obesity epidemics(5)(8).Nutritional deficiencies intensify this triad. Vitamin D insufficiency, prevalent in 70-90% of NAFLD/T2DM cases, elevates HOMA-IR by impairing beta-cell function and inflammation; meta-analyses confirm supplementation reduces HOMA-IR by 1.06 (95% CI -1.66 to -0.45)(2)(9).Magnesium deficit, linked to hypomagnesemia in 20-30% of diabetics, correlates with higher fasting glucose and IR via ATP-dependent pathways(3).Thyroid dysregulation (elevated TSH) further hinders lipid metabolism in NAFLD(10).Lifestyle interventions, emphasizing fiber-rich diets and micronutrient repletion, yield superior outcomes over pharmacotherapy alone, mirroring Mediterranean diet trials that cut liver fat by 30-40%(11)(4). This pre-post study assesses a comprehensive nutritional protocol's efficacy on IR (HOMA-IR), anthropometrics, glycemia (FBS, HbA1c), lipids, vitamins/minerals, and thyroid function in a high-risk cohort, addressing gaps in regional data from Libya/Egypt where metabolic burdens
Escalate(3)(5)..

Statistical analysis of the data

The statistical analysis of the data was performed using IBM SPSS software version 20.0 (Armonk, NY: IBM Corp, released 2011).Categorical data were summarized as numbers and percentages. For continuous data, normality was assessed using the Shapiro-Wilk test. Quantitative data were presented as range, mean, standard deviation, and median.

Paired t-test was used to compare two periods for normally distributed quantitative variables, while the Wilcoxon signed ranks test was used for non-normally distributed quantitative variables. The significance level for all statistical tests was set at 5%.

Table (1): Distribution of the studied cases according to demographic data (n = 114)

	No. (%)
Gender	
Male	52 (45.6%)
Female	62 (54.4%)
Age (years)	
Min. – Max.	26.0 – 65.0
Mean ± SD.	44.12 ± 10.53
Median (IQR)	44.0 (35.0 – 53.0)
Height (cm)	
Min. – Max.	150.0 – 185.0
Mean ± SD.	167.0 ± 9.79
Median (IQR)	167.0 (160.0 – 173.0)

IQR: Inter quartile range SD: Standard deviation

Table (2): Comparison between before and after according to anthropometric measurement and laboratory data

	Before	After	Test of Sig.	p
Weight (kg)				
Min. – Max.	68.0 – 179.0	62.0 – 112.0	Z= 9.158	<0.001*
Mean ± SD.	93.70 ± 16.65	85.41 ± 13.81		
Median (IQR)	90.50 (80.0 – 106.0)	85.50 (74.0 – 97.0)		
Waist circumference (cm)				
Min. – Max.	60.0 – 150.0	55.0 – 139.0	Z= 9.113	<0.001*
Mean ± SD.	99.36 ± 17.31	91.82 ± 15.66		
Median (IQR)	96.0 (87.0 – 110.0)	89.50 (81.0 – 100.0)		
BMI (kg/m2)				
Min. – Max.	20.20 – 46.50	18.40 – 41.90	t= 16.843	<0.001*
Mean ± SD.	33.33 ± 5.73	30.57 ± 5.20		
Median (IQR)	32.52(29.62 – 36.90)	30.16(27.30 – 33.75)		
FBS				
Min. – Max.	95.0 – 310.0	70.0 – 164.0	Z= 9.263	<0.001*
Mean ± SD.	171.7 ± 37.10	114.4 ± 24.70		
Median (IQR)	170.0(152.0 – 193.0)	113.0 (98.0 – 131.0)		
HBAIC				
Min. – Max.	5.0 – 14.0	5.10 – 9.40	Z= 9.193	<0.001*
Mean ± SD.	8.99 ± 1.54	7.06 ± 1.0		
Median (IQR)	9.0 (8.40 – 9.80)	7.10 (6.30 – 7.80)		
Insulin				
Min. – Max.	13.0 – 84.0	3.50 – 98.0	Z= 8.905	<0.001*
Mean ± SD.	28.50 ± 7.41	17.80 ± 9.64		
Median (IQR)	28.90(24.30 – 31.70)	17.0 (12.90 – 20.60)		
HOMA–IR				
Min. – Max.	4.38 – 20.90	1.25 – 11.50	t= 26.435*	<0.001*
Mean ± SD.	11.39 ± 3.30	4.76 ± 2.04		
Median (IQR)	11.60 (8.64 – 13.81)	4.49 (3.41 – 6.11)		
Vit D				
Min. – Max.	4.10 – 29.0	2.30 – 86.70	Z= 9.179	<0.001*
Mean ± SD.	14.68 ± 4.76	30.32 ± 8.56		
Median (IQR)	15.0 (10.90 – 18.0)	30.30(26.50 – 34.40)		

Magnesium				
Min. – Max.	1.08 – 2.80	1.40 – 2.50	t= 18.925*	<0.001*
Mean ± SD.	1.63 ± 0.25	2.02 ± 0.23		
Median (IQR)	1.64 (1.50 – 1.80)	2.0 (1.90 – 2.17)		

IQR: Inter quartile range SD: Standard deviation t: Paired t-test
Z: Wilcoxon signed ranks test
p: p value for comparing between before and after
*: Statistically significant at $p \leq 0.05$.

Table (3): Comparison between before and after according to laboratory data

	Before	After	Test of Sig.	p
Cholesterol				
Min. – Max.	179.0 – 354.0	140.0 – 256.0	t= 22.452*	<0.001*
Mean ± SD.	240.6 ± 32.18	195.1 ± 32.73		
Median (IQR)	237.0 (220.0 – 264.0)	196.0 (169.0 – 221.0)		
TG				
Min. – Max.	110.0 – 392.0	60.0 – 315.0	t= 24.118*	<0.001*
Mean ± SD.	254.8 ± 53.03	160.4 ± 49.69		
Median (IQR)	256.5 (215.0 – 289.0)	160.0 (132.0 – 193.0)		
TSH				
Min. – Max.	0.81 – 8.80	0.58 – 4.83	Z= 8.677	<0.001*
Mean ± SD.	3.05 ± 1.10	2.57 ± 1.0		
Median (IQR)	2.83 (2.22 – 3.63)	2.40 (1.80 – 3.24)		
T3				
Min. – Max.	0.42 – 1.47	0.60 – 8.35	t= 5.339*	<0.001*
Mean ± SD.	1.05 ± 0.19	1.20 ± 0.72		
Median (IQR)	1.06 (0.93 – 1.18)	1.13 (0.96 – 1.32)		
T4				
Min. – Max.	6.62 – 10.71	6.33 – 11.07	t= 2.707*	0.008*
Mean ± SD.	8.46 ± 0.82	8.62 ± 0.96		
Median (IQR)	8.37 (7.86 – 9.03)	8.66 (7.90 – 9.18)		

IQR: Inter quartile range SD: Standard deviation t: Paired t-test
Z: Wilcoxon signed ranks test
p: p value for comparing between before and after
*: Statistically significant at $p \leq 0.05$

RESULTS

The study enrolled 114 participants, predominantly female (54.4%), with a mean age of 44.12 ± 10.53 years.Significant improvements were observed post-intervention in anthropometric measures, including weight (from 93.70 ± 16.65 kg to 85.41 ± 13.81 kg,
p=0.001), waist circumference (99.36 ± 17.31 cm to 91.82 ± 15.66 cm, p=0.001), and BMI (33.33 ± 5.73 kg/m² to 30.57 ± 5.20 kg/m², p=0.001).Glycemic control markedly enhanced, with fasting blood sugar decreasing from 171.7 ± 37.10 mg/dL to 114.4 ± 24.70 mg/dL (p=0.001), HbA1c from 8.99 ± 1.54% to 7.06 ± 1.0% (p=0.001), insulin from 28.50 ± 7.41 µU/mL to 17.80 ± 9.64 µU/mL (p=0.001), and HOMA-IR from 11.39 ± 3.30 to 4.76 ± 2.04 (p=0.001).

DISCUSSION

These findings demonstrate that the nutritional intervention effectively ameliorated insulin resistance, as evidenced by substantial reductions in HOMA-IR, alongside improvements in glycemic parameters and anthropometric indices, consistent with prior studies on dietary management of NAFLD and type 2 diabetes. The observed enhancements in vitamin D (14.68 ± 4.76 ng/mL to 30.32 ± 8.56 ng/mL, p=0.001) and magnesium levels (1.63 ± 0.25 mg/dL to 2.02 ± 0.23 mg/dL, p=0.001) likely contributed to better insulin sensitivity, aligning with meta-analyses showing vitamin D supplementation reduces HOMA-IR by -0.39 in prediabetic and diabetic populations. Lipid profiles improved significantly (cholesterol 240.6 ± 32.18 mg/dL to 195.1 ± 32.73 mg/dL, p=0.001; triglycerides 254.8 ± 53.03 mg/dL to 160.4 ± 49.69 mg/dL, p=0.001), and thyroid function normalized (TSH 3.05 ± 1.10 µIU/mL to 2.57 ± 1.0 µIU/mL, p=0.001), supporting the role of nutrition in reversing metabolic dysfunction associated with fatty liver and diabetes.The paired improvements, assessed via Wilcoxon signed ranks and t-tests (all p<0.01), underscore the intervention's efficacy in this Middle Eastern cohort, potentially due to addressing deficiencies common in the region. Limitations include the absence of a control group and reliance on non-invasive measures; future randomized trials with liver histology could validate these changes.

CONCLUSION

This nutritional intervention significantly improved insulin resistance, fatty liver markers, and diabetes control in 114 participants, highlighting its potential as a first-line therapy. Early implementation of targeted nutrition, emphasizing vitamin D and magnesium repletion alongside calorie reduction, offers a scalable strategy for metabolic syndrome management, warranting broader clinical adoption and further research in diverse populations.

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