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Nutritional and Metabolic Profile in Nonalcoholic Fatty Liver Disease after Lifestyle Management

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Author's contributions

This work was carried out in collaboration between all authors. Author FMF in the all of the study included the drafted of the manuscript. Authors GCBM and MSFM participated in the preparation of manuscript. Author SRLR participated in the adipokines analysis phase. Author AVC participated in the ultrassonografic evaluation. Author KAPLD participated in the data collection phase. Author MHGGS participated in the design, coordination of the study, and drafted the manuscript. All authors read and approved the final manuscript.

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ABSTRACT

Aims: The aim of this study was investigate whether a comprehensive lifestyle management is able to promote improvements in the nutritional and metabolic profiles of obese women with NAFLD.
Place and Duration of Study: This study was developed in the nutrition outpatient clinic of Julio Muller University Hospital from Mato Grosso Federal University at Cuiabá, Mato Grosso State,

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Brazil, during 6 months.
Methodology: We assessed 61 obese women who received instructional treatment to change their lifestyle, at baseline and after 6 months, by anthropometric, biochemical, clinical and ultrasound measurements including liver steatosis, visceral and subcutaneous adiposity. Food intake was assessed by a qualitative food frequency questionnaire and the women were placed in NAFLD group or Control group based on the presence of liver steatosis.
Results: No difference was found in food intake, but after treatment, both groups reduced their frequency of intake of fats, sugar and sweets and to increase their consumption of cereals, vegetables and fruits. The NAFLD group reduced body weight, waist circumference and liver steatosis. Both groups improved visceral and subcutaneous adiposity, the inflammatory cytokines (tumor necrosis factor-α, interleukin-6) whereas leptin were elevated and adiponectin were lowered during all the study. Strong positive correlations were found in the NAFLD group between visceral adiposity and body mass index, between subcutaneous adiposity and systolic and diastolic blood pressure, and between interleukin-6 and leptin. Although occurred in both groups during the study, high insulin resistance and low insulin sensitivity were more pronounced in the NAFLD group.
Conclusion: We observed that even a small management in lifestyle can play an important role in the improvement of nutritional and metabolic profiles of obese women with NAFLD.

Keywords: Liver steatosis; nutrition and metabolism; lifestyle management; women.

1. INTRODUCTION

The prevalence of nonalcoholic fatty liver disease (NAFLD) is greater in obesity and lifestyle management is the primary treatment [1]. Although obesity is closely associated with this disease, an excess of abdominal adiposity, particularly visceral fat storage, is acknowledged as the most important factor in NAFLD [2]. Excessive visceral fat accumulation plays a role in steatosis and fibrosis in the pathogenesis and prognosis of NAFLD [3]. Adipose tissue secretes several adipokines, including adiponectin, leptin, tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6) and resistin; visceral adipose tissue predominantly expresses TNF-α and IL-6, and subcutaneous adipose tissue predominantly expresses leptin and adiponectin [4].

Previous studies have shown that individuals with NAFLD have a greater intake of dairy products, meat, and foods with low nutritional value and high salt content, combined with low consumption of fruits [5]. Zelber-Sagi et al. [6] demonstrated that patients with NAFLD had a greater intake of soft drinks and meat and those women with this disease had a higher energy intake. In our laboratory, Magalhães et al. [7] showed in recent study, an association between sucrose and fatty foods with lower adiponectin levels in the liver disease group.

There are no established methods for intensive lifestyle modification in NAFLD because of the difficulties in achieving and maintaining weight reduction [8,9]. However, the usual management of NAFLD includes advising patients to improve diet quantity and quality and to increase physical activity, which is currently the first line of treatment [10].

Thus, the aim of this study was investigate whether a comprehensive lifestyle management is able to promote improvements in the nutritional and metabolic profiles of obese women with NAFLD.

2. MATERIALS AND METHODS

2.1 Study Design

This study was developed in the nutrition outpatient clinic of Julio Muller University Hospital from Mato Grosso Federal University at Cuiabá, Mato Grosso State, Brazil, in accordance with the principles of the Declaration of Helsinki and was formally approved by the Institutional Ethical Committee (CEP/UFMT Nº 346). Informed consent was obtained from each patient.

Sixty-one obese women, aged 20-65 years, were evaluated at baseline and after 6 months of treatment. Body mass index (BMI) ≥ 30 Kg/m^2 was used as the initial criterion to identify the presence of obesity according to the World Health Organization classification standards. The exclusion criteria included a history of excessive alcohol intake (> 20 g/day), the use of drugs

associated with secondary NAFLD and other causes of chronic hepatic disease [11].

2.2 Study Measurements

Participant evaluation included an interview, anthropometric measurements, body composition determination, biochemical and clinical assessments and ultrasound (US) examination.

2.3 Interview

A face-to-face interview was carried out in all cases by the same interviewer. The first part of the questionnaire included demographic data, health status, medication use, physical exercise, current alcohol intake and smoking status. The second part was a detailed qualitative food frequency questionnaire (FFQ) that included 8 food groups based on the Adapted Food Pyramid for the Brazilian population: 1- cereals, bread, and tubercles; 2- vegetables (lettuce, watercress, broccoli, onion, cabbage, cauliflower, gherkins, cucumbers, tomato, squash, zucchini, eggplant, beets, carrots, chayote, green pepper, okra, green beans); 3- fruits; 4- legumes (beans, soy, peas, chick peas, broad beans, peanut); 5- meat and eggs; 6- milk and dairy products; 7- sugar and sweets; and 8- oils and fats [12]. For each food group, participants indicated their average frequency of consumption (daily, once a week, twice a week, three or more times per week, occasionally or never). “Frequent” food intake was defined as daily, once a week, twice a week or three or more times per week. “Infrequent” food intake was defined as occasionally or never.

2.4 Anthropometric Measurements and Body Composition

Body weight was measured in light clothing and without shoes to the nearest half-kilogram. Height was measured to the nearest half-centimeter. Body mass index (BMI) was calculated as weight (kilograms) divided by height (meters) squared. Waist circumference (WC) was measured to the nearest half-centimeter at the narrowest point below the lower rib margin and the iliac crest, and hip circumference was measured at the widest point between the hips and buttocks. Body fat distribution was evaluated by waist-to-hip ratio (WHR).

Body fat, water, muscle and bone were analyzed using the Body Composition Monitor, Tanita Ironman InnerScan Model: BC-558. Thickness measurements of visceral and subcutaneous fat (cm) were obtained by ultrasound examination, and all measurements were performed by the same examiner and service according to Ribeiro-Filho et al. [13]. Subcutaneous adiposity (SA) was defined as the distance between the skin and external face of the rectus abdominal muscle; visceral adiposity (VA) was defined as the distance between the internal face of the rectus abdominal muscle and the anterior wall of the aorta 1 cm below the navel [13].

2.5 Biochemical Tests

Each participant underwent biochemical testing following a 12-hour fast to measure the following: the liver enzymes alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyltranferase (GGT); a serum lipid profile including total cholesterol (TC), very low density lipoprotein (VLDL), low density lipoprotein (LDL), high density lipoprotein (HDL) and triacylglycerol (TAG); glucose and insulin levels; and C-reactive protein (CRP); serological tests were performed for hepatitis. All biochemical assessments were performed in the Laboratory of Julio Muller University Hospital/UFMT, Cuiabá, MT, Brazil following standard methods.

The degree of insulin resistance was determined by the homeostatic model assessment-insulin resistance (HOMA-IR: fasting insulin (μIU/mL) x fasting glucose (mmol/L)/22.5) [14] and the quantitative insulin sensitivity check index (QUICKI: 1/[log fasting insulin (μIU/mL) + log fasting glucose (mg/dL)]) [15].

Cytokine and adipokine levels were measured in blood drawn obese women (NAFLD group and Control group) in the morning after an overnight fast. The blood samples were immediately centrifuged, and the serum samples were stored at -80ºC until analysis at the Laboratory of Biological Food Evaluation (UFMT) with an enzyme-linked immunosorbent assay (ELISA) kit according to the manufacturer’s instructions. The serum samples were quantified after dilution, and each measurement was performed in duplicate. Tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) levels were quantified using the ELISA MAX™ Deluxe Set from Biolegend (San Diego, USA). Leptin and adiponectin (adipo) levels were measured with an ELISA kit from Abcam (Cambridge, USA). The absorbance was read at 450 nm by Spectra Max 190. The calibration curves were constructed by plotting the net average absorbance of the standards on the Y-

axis and the concentrations on the X-axis and drawing the best-fitting curve. Concentrations of the adipokines in each sample were calculated from the calibration curve with ORIGIN software version 4.1. At baseline the correlation coefficients were linear in a concentration range between 0.16 and 3.55 ng/mL for adiponectin (r = 0.99); 4.42 and 76.11 ng/mL for leptin (r = 0.99); 7.6 and 9.38 pg/mL for IL-6 (r = 0.99); 4.45 and 31.25 pg/mL for TNF-α (r = 0.99), and after 6 months the correlation coefficients were linear in a concentration range between 0.25 and 4.16 ng/mL for adiponectin (r = 0.99); 2.71 and 64.86 ng/mL for leptin (r = 0.99); 7.56 and 9.09 pg/mL for IL-6 (r = 0.99); and 2.25 and 24.99 pg/mL for TNF-α (r = 0.99).

2.6 Clinical Examination

Blood pressure (BP) was measured by a nurse technician during the pre-consultation after at least five minutes of rest before the measurement. Blood pressure levels (systolic BP and diastolic BP) were classified according to normal standards.

2.7 Imaging Examination

Fatty liver was diagnosed by abdominal ultrasonography (US), which was performed in all women with the same equipment (Voluson 730 Expert, General Electric (GE), Áustria). The examination was performed after fasting for 12 hours in the morning. The diagnosis of NAFLD was based on the presence and degree of liver steatosis, which was classified as mild, moderate or severe according to the stratification proposed by Rumack et al. [16]. The women were divided into the NAFLD or the Control group according to the presence of liver steatosis.

Although US detects steatosis with a sensitivity of 60-94% and a specificity of 88-95%, it has the advantage of being a low-cost assessment tool with no known risks and is available in almost all cities; therefore, this technique is considered a good method for tracking disease [17].

2.8 Intervention

All participants included in the study received individual instructions to change their eating habits and lifestyle. However, no fixed level of energy intake was prescribed; rather, the participants were encouraged to adopt a balanced daily diet based on lower consumption of sugar, sweet beverages and fatty foods and greater intake of vegetables and fruits. Additionally, they were instructed to improve their regular physical activity. No drugs or antioxidants were recommended. After six months, they underwent a new evaluation of the same parameters by the same examiner.

2.9 Statiscal Analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences version 19.0 (SPSS Inc., Chicago, IL, USA) software. Continuous variables were expressed as the mean ± standard error of the mean (SEM). Comparisons between measures at baseline and after 6 months were made using paired Student's t-test or Wilcoxon signed rank test. Comparisons between groups were performed using Student's t-test or the Mann-Whitney test. Categorical data were presented as relative frequencies. The McNemar test and the Wilcoxon signed rank test were used to compare the differences between paired categorical data, and the Chi-square test or Fisher's exact test were used for independent categorical data. The correlation linear analysis of continuous variables was performed using the linear coefficient of Pearson or Spearman. The delta variation (Δ) was used for the statistical analysis obtained from the difference between the baseline and 6-month values for each variable. To reject the null hypothesis, the value of $P< 0.05$ was used.

3. RESULTS

A total of 61 women were included in the study. At baseline, 49% of these women had steatosis (the NAFLD group), which after 6 months was reduced to 41%. The number of obese women with normal liver (the control group) improved from 51 to 59% after 6 months (Fig. 1). Mild, moderate and severe steatosis were reduced from 43 to 38%, from 5 to 3% and from 2 to 0%, respectively, after 6 months ($P= 0.01$).

3.1 NAFLD Group

Significant decreases occurred after 6 months in the anthropometric, biochemical and abdominal adiposity parameters, including body weight, BMI, WC, VA and SA, in AST and GGT enzymes levels and in IL-6 and TNF-α cytokines (Tables 1, 2; Fig. 2). The mean weight loss was 2.0±0.7 Kg (Table 1). The level of physical activity was low during the study (data not shown), and no significant differences were found in food intake, although there was a tendency of reduced consumption of sugar and fats (Fig. 3). According to correlation analyses (Table 3), the Δ in body

weight correlated positively with the Δ in diastolic blood pressure, GGT, CRP and with the Δ in VA, which correlated with the Δ in SA and BMI. Additionally, the Δ in VA and SA associated positively with the Δ in systolic and diastolic blood pressure, and the Δ in IL-6 correlated positively with that of leptin levels.

3.2 Control Group

After 6 months, this group showed improvement in VA and SA parameters, HDL-cholesterol levels, the AST/ALT ratio and IL-6 and TNF-α cytokine levels (Tables 1, 2; Fig. 2). No significant weight loss occurred (1.2±1.0 Kg; Table 1). The level of physical activity was low and food intake was unchanged (Fig. 3). A positive association occurred between the Δ in VA and the Δ in body weight, BMI and TNF-α (Table 3). The Δ in SA was positively correlated with Δ body weight, diastolic blood pressure and glucose (Table 3).

3.3 Comparison between the Groups

In the NAFLD group, visceral adiposity, fasting serum glucose and insulin levels, HOMA-IR, QUICKI and GGT were higher during the study compared with the Control group (Tables 1, 2). The frequency of comorbidities at baseline between the NAFLD and Control groups, respectively, for hypertension (50 vs. 39%), dyslipidemia (20 vs. 19%) and diabetes (17 vs. 16%) were similar between groups. The Control group presented higher practice of physical activity related to the NAFLD group after 6 months (64% vs. 37%, respectively); walking was the most common activity reported in both groups and periods (data not shown). Although no significant difference occurred between the groups and in both periods for the FFQ based on the Adapted Food Pyramid for the Brazilian population (Fig. 3), the women tended to consume lower amounts of fats, sugar and sweets and, this trend being more pronounced in the NAFLD group, after 6 months.

4. DISCUSSION

Current studies emphasize that even small changes in body weight or even weight maintenance in conjunction with improved dietary and/or physical activity habits can bring about improvements in metabolic and liver profile in NAFLD patients [18].

After 6 months, we observed a body weight loss of 2% in the NAFLD group and none in the Control group. According to study reported by Okita et al. [19] a mean of 1.6 Kg of weight loss after 8 weeks and 2.4 Kg after 24 weeks was observed in obese patients with NAFLD who consumed an energy-restricted diet. Another study evaluated overweight individuals with NAFLD after lifestyle interventions that included a low-lipid diet and encouraged activity; a weight loss of 3% resulted after 6 months [20].

Table 1. Anthropometric and clinical analysis of NAFLD and control groups at baseline and after 6 months

Variables	NAFLD (N= 30)			Control (N= 31)		
	Baseline	6 months	Δ value	Baseline	6 months	Δ value
Age (years)	43.7±1.9	-	-	39.6±2.1	-	-
Body weight (Kg)	93.2±3.2	91.1±3.3*	-2.0±0.7	92.8±3.3	91.6±3.3	-1.2±1.0
BMI (Kg/m^2)	38.3±1.1	37.4±1.2*	-0.9±0.3	36.6±1.1	36.2±1.2	-0.4±0.3
WC (cm)	114.6±2.6	112.5±2.5*	-2.1±0.8	112.6±2.2	113.5±2.5	1.0±1.2
WHR	0.99±0.01	0.99±0.01	0.001±0.01	0.96±0.01	0.97±0.01	0.02±0.01
VA (cm)	5.2±0.2	4.7±0.2*	-0.5±0.1	4.1±0.2$^{\#}$	3.8±0.2$^{\#\#*}$	-0.3±0.1
SA (cm)	4.0±0.2	3.5±0.2*	-0.5±0.1	4.3±0.2	3.9±0.2*	-0.4±0.1
Water (%)	42.1±1.1	41.7±0.8	-0.4±0.6	41.4±0.6	42.4±0.8	1.0±0.8
Bone (Kg)	2.6±0.05	2.5±0.05	-0.06±0.03	2.6±0.05	2.6±0.1	0.05±0.05
Muscle (Kg)	48.8±1.0	47.6±1.0	-1.2±0.5	48.3±1.1	49.0±1.7	0.7±1.0
Fat (%)	43.9±1.4	44.0±1.2	-9.0±6.4	44.6±0.9	43.5±1.0	-5.9±6.0
Systolic BP (mmHg)	125.20±3.8	129.4±3.5	4.2±3.8	120.0±2.8	126.0±3.2	6.0±3.1
Diastolic BP (mmHg)	82.8±2.6	80.3±2.1	-2.4±2.4	81.4±1.8	85.04±2.1$^{\#\#}$	3.6±2.5

BMI: body mass index; WC: waist circumference; WHR: waist-to-hip ratio; VA: visceral adiposity; SA: subcutaneous adiposity; Systolic BP: systolic blood pressure; Diastolic BP: diastolic blood pressure. Values expressed in mean ± SEM. * *P< 0.05 comparison of baseline vs. after 6 months in the same group. Paired Student's t-test or Wilcoxon signed rank test.* $^{\#}$ *P< 0.05 comparison of NAFLD vs. Control group at baseline.* $^{\#\#}$ *P< 0.05 comparison of NAFLD vs. Control group after 6 months. Comparison in Δ values of NAFLD vs. Control groups. Student's t-test or Mann Whitney test*

Table 2. Biochemical analysis of NAFLD and control groups at baseline and after 6 months

Variables	NAFLD (N= 30)			Control (N= 31)		
	Baseline	6 months	Δ value	Baseline	6 months	Δ value
Glucose (mg/dL)	124.6±9.0	112.6±7.0	-9.0±6.4	98.2±6.0$^{\#}$	92.3±1.4$^{\#\#}$	-5.9±6.0
Insulin (μIU/mL)	18.5±1.8	20.0±2.0	1.5±2.5	12.5±1.2$^{\#}$	12.4±0.7$^{\#\#}$	-0.01±1.3
HOMA-IR	5.2±0.5	5.5±0.5	0.3±0.7	2.7±0.2$^{\#}$	2.8±0.2$^{\#\#}$	0.05±0.3
QUICKI	0.308±0.005	0.304±0.004	-0.003±0.006	0.335±0.005$^{\#}$	0.330±0.003$^{\#\#}$	-0.005±0.005
TC (mg/dL)	200.4±7.0	204.7±10.7	4.2±9.4	188.3±7.3	190.7±6.6	2.4±5.6
VLDL (mg/dL)	33.5±3.0	30.5±4.8	-3.0±3.6	24.8±2.2$^{\#}$	22.2± 2.2	-2.6±2.3
LDL (mg/dL)	125.4±5.1	129.3±7.6	3.8±7.8	121.4±6.6	123.3±6.0	1.8±4.3
HDL (mg/dL)	42.8±1.3	45.0±1.3	2.2±1.2	42.9±1.3	45.7±1.5*	2.7±1.2
TAG (mg/dL)	161.0±13.4	152.4±22.7	-8.6±16.0	131.8±13.8$^{\#}$	117.1±13.5	-14.7±10.3
ALT (IU/L)	29.1±3.6	25.2±2.0	-3.8±3.4	33.4± 9.1	31.4± 5.0	-2.0±6.3
AST (IU/L)	23.0±2.1	17.5±0.9*	-5.5±1.9	23.6± 2.6	21.1± 2.0	-2.5±2.5
AST/ALT ratio	0.9±0.03	0.7±0.04	-0.1±0.05	1.0±0.1	0.9±0.1*	-0.1±0.06
GGT (U/L)	42.3±3.6	32.0±2.5*	-10.3±3.4	32.0±3.6$^{\#}$	28.7±3.2$^{\#\#}$	-3.3±2.7
CRP (mg/dL)	10.5±2.0	9.3±1.3	-1.1±1.7	7.2±2.2$^{\#}$	7.3± 1.6	0.1±1.5
Adipo (ng/mL)	1.3±0.2	1.2±0.2	-0.1±0.1	1.4±0.2	1.6±0.2	0.2±0.2
Leptin (ng/mL)	33.0±4.6	33.1±4.0	0.1±3.8	29.4±3.5	28.6±3.2	-0.8±4.8
IL-6 (pg/mL)	8.6±0.1	8.2±0.1*	-0.4±0.1	8.6±0.1	8.1±0.1*	-0.5±0.1
TNF-α (pg/mL)	14.7±1.7	7.8±1.3*	-7.0±2.2	12.8±1.6	7.3±1.0*	-5.5±2.0

HOMA-IR: homeostatic model assessments insulin resistance; QUICKI: quantitative insulin sensitivity check index; TC: total cholesterol; VLDL: very low density lipoprotein; LDL: low density lipoprotein; HDL: high density lipoprotein; TAG: triacylglycerol; GGT: gamma-glutamyltranferase; ALT: alanine aminotransferase; AST: aspartate aminotransferase; CRP: c-reactive protein; Adipo: adiponectin; IL-6: interleukin 6; TNF-α: tumor necrosis factor- α. Values expressed in mean±SEM. * P< 0.05 comparison of baseline vs. after 6 months in the same group. Paired Student's t-test or Wilcoxon signed rank test. $^{\#}$P< 0.05 comparison of NAFLD vs. Control groups at baseline. $^{\#\#}$ P< 0.05 comparison of NAFLD vs. Control groups after 6 months. Comparison in Δ values of NAFLD vs. Control groups. Student's t-test or Mann Whitney test

Table 3. Correlations analyses*

Groups	Δ Variables	r#	P
NAFLD			
Δ Body weight (Kg)	Diastolic BP (mmHg)	0.394	0.034
	GGT (U/L)	0.477	0.010
	CRP (mg/dL)	0.426	0.030
	VA (cm)	0.588	0.001
Δ VA (cm)	Systolic BP (mmHg)	0.396	0.033
	Diastolic BP (mmHg)	0.401	0.031
	BMI (Kg/m^2)	0.537	0.004
	SA (cm)	0.418	0.021
Δ SA (cm)	Systolic BP (mmHg)	0.508	0.005
	Diastolic BP (mmHg)	0.495	0.006
Δ IL-6 (pg/mL)	Leptin (ng/mL)	0.485	0.026
Control	**Δ Variables**	**r#**	**P**
Δ VA (cm)	Body weight (Kg)	0.357	0.048
	BMI (Kg/m^2)	0.417	0.020
	TNF-α (pg/mL)	0.485	0.019
Δ SA (cm)	Body weight (Kg)	0.586	0.001
	Diastolic BP (mmHg)	0.433	0.021
	Glucose (mg/dL)	0.386	0.039

*GGT: gamma-glutamyltransferase. CRP: C-reactive protein. VA: visceral adiposity. SA: subcutaneous adiposity. BP: blood pressure. BMI: body mass index. IL-6: interleukin-6. TNF-α: tumor necrosis factor-α. * P< 0.05 Linear Coefficient of Pearson or Spearman. #Positive correlations. Groups NAFLD: N=30; Control: N=31*

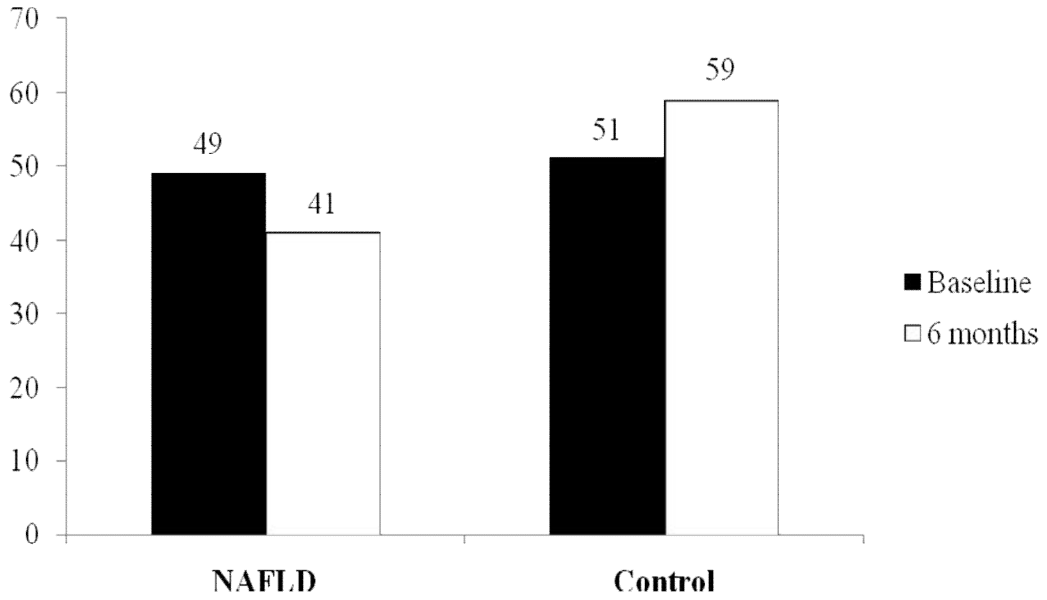

Fig. 1. Frequency (%) of steatosis (NAFLD) and normal liver (control) in obese women at baseline and after 6 months

Comparison of baseline vs. after 6 months in the same group, McNemar test.
Groups NAFLD: N=30; Control: N=31

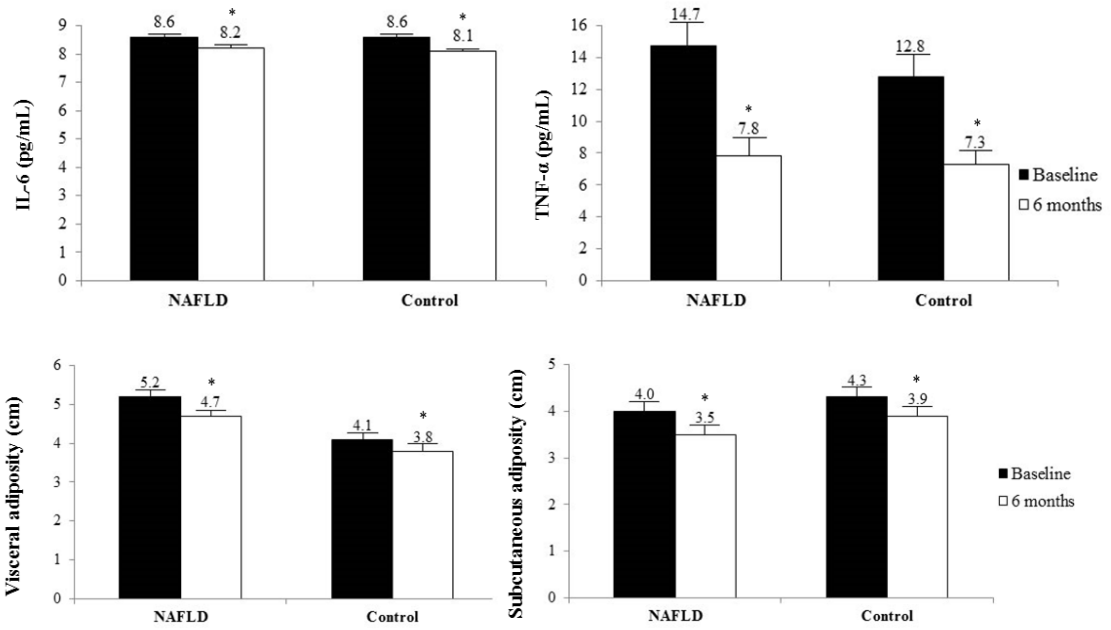

Fig. 2. Visceral and Subcutaneous adiposity, Interleukin 6 (IL-6) and Tumor necrosis factor-α (TNF- α) cytokines in NAFLD and Control groups

**P< 0.05 comparison of baseline vs. after 6 months in the same group. Paired Student's t-test or Wilcoxon Signed Rank test.Comparison of NAFLD vs. Control groups at baseline and after 6 months. Student's t-test or Mann Whitney test.Groups NAFLD: N=30; Control: N=31*

No significant difference in food intake was found in our study, but both groups tended to have lower intake of fats, sugar and sweets. However, compared to control, the NAFLD group had more reduction in food intake of fats (10% vs. 3%) and, in sugar and sweets (20% vs. 13%). The consumption of vegetables, cereals and fruits, remained high during the study in both groups. Additionally, we observed a reduction in liver steatosis from 49 to 41% and, improvements in abdominal adiposity (visceral and subcutaneous), as the reduction in visceral adiposity was correlated with body weight and BMI.

In study described by de Piano et al. [21], obese adolescents with and without NAFLD were evaluated for 1 year and showed a weight loss of approximately 10% in both groups and a significant decrease in intake of total energy, macronutrients and saturated and monounsaturated fatty acids. Oza et al. [8] observed a reduction in body weight in Japanese patients with NAFLD during 6 months of a home-based lifestyle intervention that was associated with improved visceral fat accumulation, liver fat deposition and liver function. On the other hand, Koda et al. [22] and Finelli and Tarantino (2012) verified that visceral adiposity accumulation was the most important factor for the development of hepatic steatosis and had the strongest correlation with the variation in body weight and, showed the correlation between WC and VA as a marker for abdominal adiposity [2].

During the study, high insulin resistance and low insulin sensitivity occurred in both groups, although these characteristics were more pronounced in NAFLD group. Sun et al. [23] also did not observe improvement in insulin resistance after 6 months but only after 12 months of intervention in NAFLD and Control groups.

On the other hand, our study showed in both groups persistently low levels of adiponectin and high levels of leptin. However, after 6 months of treatment, a reduction in the pro-inflammatory cytokines IL-6 and TNF-α was detected. Visceral fat deposition releases various adipokines, such as TNF-α, IL-6, resist in, leptin and adiponectin; among these, low adiponectin levels are associated with NAFLD [3,24]. These adipokines, particularly IL-6, stimulate hepatic synthesis and

the secretion of other markers of inflammation. Jarrar et al. [25] demonstrated that serum levels of cytokines such as TNF-α and IL-6 were higher in NAFLD patients in comparison with obese controls, whereas adiponectin levels were not different between groups; however, lower levels of adiponectin and higher TNF-α levels were related to NAFLD severity. There is evidence that VA produces more pro-inflammatory cytokines, such as TNF-α and IL-6, than does SA [26,27].

According to George et al. [18] which reported the effects of a comprehensive lifestyle intervention as opposed to intense weight loss alone in patients with liver disease, our study showed that small changes in eating habits may have contributed to the reduction in body weight and in abdominal adiposity, which resulted in a reduction in liver steatosis.

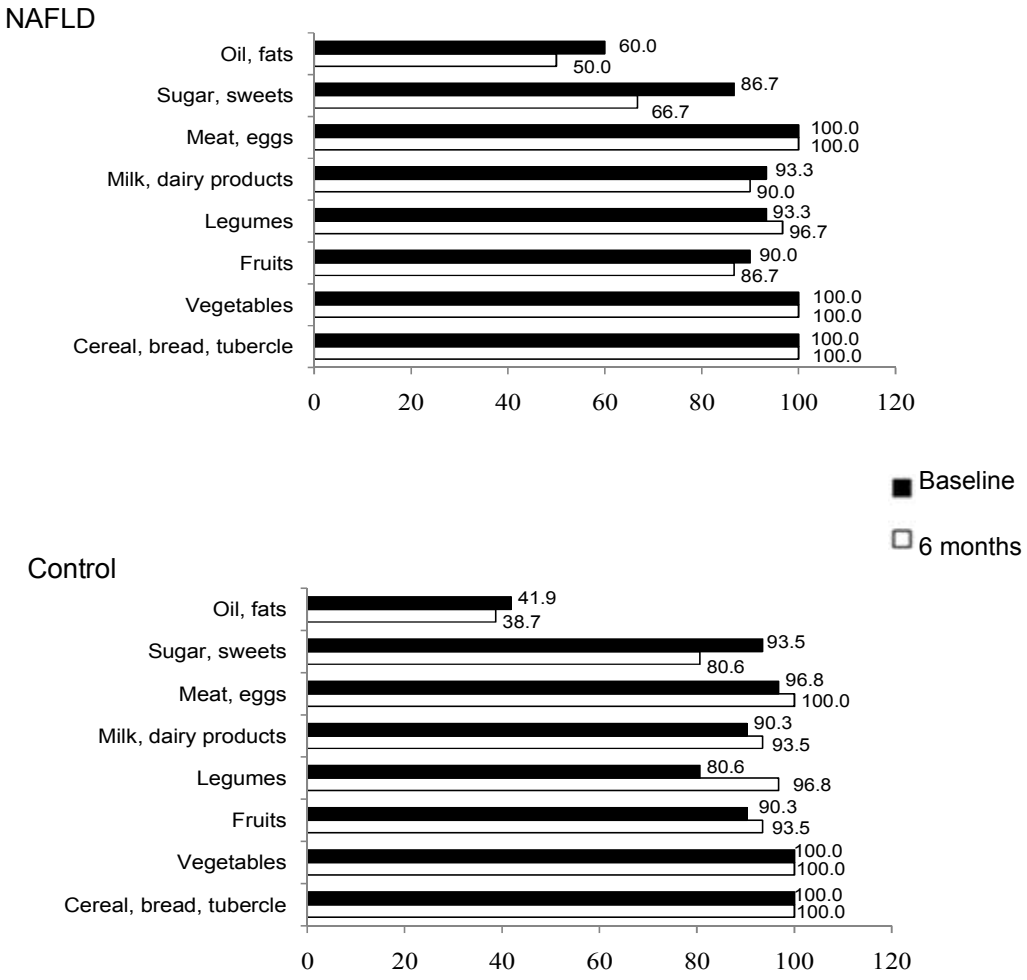

Fig. 3. Frequency (%) of food intake in NAFLD and control groups at baseline and after 6 months

Comparison of baseline vs. after 6 months in the same group, McNemar test. Comparison of NAFLD vs. Control groups in the same period, Chi-square test or Fisher exact test.
Groups NAFLD: N=30; Control: N=31

5. CONCLUSION

We concluded that even a small modification in lifestyle can play an important role in the improvement of nutritional and metabolic profile of obese women with NAFLD.

However, the establishment of methods to encourage greater adherence to lifestyle modifications are necessary to achieve better results. Our data also highlight the need for larger studies employing instruments that will ensure adequate quantitative and qualitative assessment of diet and physical activity.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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