



Original Article



Association between Dietary Patterns and Non-alcoholic Fatty Liver Disease: Results from a Case-Control Study

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Abstract

Background: This study aimed to investigate the association between dietary patterns and non-alcoholic fatty liver disease (NAFLD) among Iranian adults.

Methods: This case-control study was conducted on 210 subjects. NAFLD diagnosis was made by ultrasound examination. Anthropometric measures, physical activity, fasting serum levels of glucose, alanine aminotransferase (ALT), aspartate aminotransferase, and lipid profile were assessed. A three-day food diary was used to assess dietary intakes of the subjects. Dietary patterns were determined using factor analysis. To determine the relationship between dietary patterns and NAFLD, multivariable-adjusted odds ratio (OR) obtained from the logistic regression analysis was used.

Results: Two dietary patterns were extracted as follows: vegetables, legumes, fruits, and low-fat dairy products (VLFD); and sweet, hydrogenated fat, red and processed meat, and soft drink (SHMS) dietary patterns. By taking all possible confounders into account, the VLFD dietary pattern was found to be significantly related to lower odds of NAFLD, while the SHMS dietary pattern was independently related to higher odds of NAFLD ($P < 0.05$). Among major food groups, high consumption of processed meat, hydrogenated fats, sweets and desserts, and soft drinks was positively related to NAFLD ($P < 0.05$), whereas vegetable consumption exhibited a protective role against NAFLD ($P = 0.01$).

Conclusion: The VLFD dietary pattern was associated with reduced likelihood of having NAFLD, while the SHMS dietary pattern was associated with higher likelihood. Therefore, the VLFD dietary pattern might be useful in the nutritional strategies for NAFLD patients. Further studies with larger sample sizes and prospective design are warranted.

Keywords: Adult population, Dietary patterns, Factor analysis, Non-alcoholic fatty liver (NAFLD)

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Introduction

With the growing prevalence of obesity worldwide, much attention has been given to the metabolic syndrome as well as its related hepatic manifestation, non-alcoholic fatty liver disease (NAFLD).^{1,2} This disease is defined as a liver pathologic spectrum initiated by simple hepatic steatosis that can progress to steatohepatitis, fibrosis, cirrhosis, and finally to liver cancer.¹ Although the real prevalence of NAFLD may vary depending on the sensitivity of the detection method used, recently, there has been a significant increase in the prevalence of NAFLD. To date, the prevalence rate of NAFLD has been estimated between 8% and 45% worldwide.³ However, NAFLD prevalence is estimated to range from 42% to 93% in obese subjects and in patients with the metabolic syndrome.^{3,4} The prevalence of NAFLD has also increased recently in Asian countries due to the rise of overweight/obesity, diabetes, and the metabolic syndrome among the population in this region.^{5,6} Lifestyle modifications including a healthy

diet, weight reduction, and increasing physical activity remain the first-line therapies for NAFLD.⁷⁻¹² Western lifestyle characterized by decreased physical activity and consumption of diets that are rich in animal fat, saturated fats, processed meat, red meat, refined grains, and sweets is associated with increased risk of NAFLD.^{13,14}

Most of the previous studies assessing the association between diet and NAFLD have focused on the traditional dietary assessment procedures, and investigated the effect of separate nutrients or foods. This method has shown conflicting findings difficult to be interpreted due to problems of isolating the impacts of specific nutrients or foods on the outcome.¹⁵ Rather than looking at individual nutrients, a dietary pattern analysis evaluates the effects of overall diet, including interactions and synergistic effects among nutrients and foods.¹⁶ Therefore, dietary patterns may be more informative for assessing the diet-disease associations compared to individual foods or nutrients.¹⁷

In recent studies, much attention has been paid to

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the association between overall dietary patterns and NAFLD.¹⁸⁻²⁰ Correspondingly, it was demonstrated that a Mediterranean dietary pattern is inversely correlated with NAFLD.^{8,21,22} In contrast, an unhealthy dietary pattern has been shown to increase the risk of NAFLD as well as its risk factors.⁸ However, to the best of our knowledge, there is limited research performed on the association between overall dietary patterns and NAFLD among Iranian adults. Therefore, the current study was designed to examine the relationship between dietary patterns and NAFLD in a sample of Iranian adults using a matched case-control design.

Materials and Methods

Study Population

This matched case-control study was carried out between May and November 2019 in northwestern Iran. Patients were recruited from the gastrointestinal clinic at Imam Reza hospital in northwestern Iran. The present study included 105 subjects newly diagnosed with NAFLD. After recruitment of all cases, 105 healthy individuals without NAFLD (from the same clinic) were recruited as the control group. Each patient was matched to a control of the same sex and age. The sample size of the study was calculated based on the information from the main outcomes of the study, including weight, body mass index (BMI), waist circumference (WC) and alanine aminotransferase (ALT). Considering a confidence of 95%, a power of 95, the sample size was estimated by the PASS 15 software to be 66, 67, 101 and 21 per group, respectively for weight, BMI, WC and ALT.^{23,34} To cover the sample size required for all risk factors of the study, we chose the maximum sample size estimated. The primary information to calculate sample size was obtained from a study by Soleimani et al¹⁸ wherein the information on the main outcome corresponding to the maximum sample size was: WC (cm): cases (mean ± SD): 102.3 ± 13.5, controls (mean ± SD): 96.3 ± 15.9.

The inclusion criteria for the case and control groups were subjects aged 30 to 60 years, and those with ultrasound-diagnosed NAFLD for the cases and without NAFLD as detected by ultrasound for the controls. Patients with liver diseases such as hepatitis and biliary disease and inherited disorders affecting liver, those with kidney, thyroid, or gastrointestinal disorders, and those with diabetes, hypertension, coronary heart disease, inflammatory disease, polycystic ovary syndrome, rheumatoid arthritis, recent surgery, cancer, and stroke were not included. Additional exclusion criteria were: pregnancy, lactation, smoking, alcohol intake during six months prior to the study, adhering to a specific diet within the last six months, use of medications such as corticosteroids, anticoagulant, antidiuretic, hepatotoxic drugs, antidiabetic drugs, lipid-lowering agents, and anti-obesity drugs over three months prior to the study.

Written informed consent was obtained from all participants before enrollment. Subjects underwent in-person interviews to determine medical history.

Dietary Intake and Physical Activity Assessment

A three-day food diary (two weekdays and one weekend day) was used to assess dietary intakes of the subjects. The Nutritionist IV software (First Databank, San Bruno, CA, USA) modified for Iranian foods was applied for dietary data analysis. In this study, twenty-one food groups were included in factor analysis. Data regarding food groups are presented in Table 1.

The level of physical activity of the participants was estimated by international physical activity questionnaire-short form (IPAQ-SF). The validity of this questionnaire has been confirmed previously.²⁵ Responses were converted to Metabolic Equivalent Task minutes per week (MET-minutes/week). The participants were categorized as having high, moderate, or low level of activity, based on the IPAQ scoring protocol.

Assessment of Anthropometric Indices

All anthropometric indices were assessed by a trained nutritionist. Body weight was measured using a scale (Seca, Germany) with a precision of 0.1 kg, in light clothing. Height was measured without shoes using a stadiometer with a precision of 0.1 cm. BMI was calculated as weight (kg) divided by height squared (m²). WC was measured at midpoint between the top of the iliac crest and the lower margin of the least palpable rib, by a stretch-resistant tape. Hip circumference (HC) was assessed at the widest part of the hip at the level of the greater trochanter. Body fat mass was assessed by Bioelectrical impedance analysis (BIA-Tanita MC-780, Amsterdam).

Biochemical Assays

After 12-14 h overnight fast, blood samples were collected and stored at -70°C until biochemical analysis. Serum concentrations of total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), fasting blood sugar (FBS), ALT, and aspartate aminotransferase (AST) were assessed through enzymatic methods by commercial kits (Pars Azmoon Co., Tehran, Iran). Low-density lipoprotein cholesterol (LDL-C) was measured with the use of the Friedewald formula. The enzyme-linked immunosorbent assay (ELISA) method was used to assess serum insulin level using commercial kits (Monobind, Lake Forest, CA, USA). The homeostatic model (HOMA-IR) was calculated using the following formula:

$$HOMA-IR = [fasting\ insulin\ (\mu IU/mL) \times FBS\ (mg/dL)] / 405$$

NAFLD Diagnosis

All patients underwent ultrasonography for determining

Table 1. Food Grouping Used in the Factor Analysis

Food Groups	Food Items
Vegetables	Lettuce, all kinds of cabbage, cucumber, tomato, spinach, mushroom, garlic, carrot, onions, eggplant, celery, green peas, green beans, turnip, maize, bell peppers, squash, tomato
Legumes	Beans, chickpeas, broad beans, lentils, soybeans, lima beans, mung beans
Fruits	Apple, cherries, grapefruit, apricots, plum, kiwi, strawberry, oranges, grapes, berries, dates, barberry, banana, pomegranate, melon, tangerine, lemon, raisin, mulberry, persimmon, peach, cantaloupe, watermelon, pear, fruit juices
Low-fat dairy	Yogurt, dough (yogurt drink), milk, cheese
White meat	Chicken, turkey, ostrich, fish, seafood
Vegetable oils	Vegetable oils
Whole grains	Barely bread, whole wheat, wheat germ, oat, bulgur, corn flakes
Nuts	Peanut, almond, pistachio, walnut, hazelnut, roasted seeds
Refined grains	White bread, rice, flour, macaroni, noodle, biscuit, crackers, cake
Sweets and desserts	Jam, honey, candy, sugar, chocolate, pastry
Hydrogenated fats	Animal fats, butter, margarine
Red and processed meat	Beef, lamb, camel, sausages, hamburger, processed meats, organ meats
Soft drinks	Soft drinks
High-fat dairy	Whole milk, Chocolate milk, high-fat yogurt, cream cheese, cream, ice cream, pizza cheese, curd
Fried potato	French fries, chips
Pizza	Pizza
Egg	Egg
Cooked potato	Cooked potato
Pickle	Pickle
Tea and coffee	Tea and coffee
Salt	Salt

fatty liver by a single expert radiologist. Echogenicity grading was performed using SonoAce X4 ultrasound system (South Korea). NAFLD was defined as the presence of moderate-severe hepatic steatosis in the absence of excessive intake of alcoholic beverages.

Statistical Analysis

All statistical procedures were done using SPSS software (IBM, Armonk, USA). Dietary patterns were determined through factor analysis. The eigenvalues and the scree plot were applied to decide on the number of factors retained. We performed a principal component analysis with varimax rotation in order to make the factors that interpreted the maximum variability of the data. A factor score was created for participants by summing intakes of food groups weighted by their factor loading. The factor scores were further classified into tertiles, where the first tertile represented low adherence and the third tertile displayed high adherence to the dietary pattern. Homogeneity of variance was tested using Leven's test for equality of variances and in all cases, the results showed that the assumption was satisfied for all variables. The normal probability plot was applied to check the normal distribution of data. Independent-sample *t*-test was applied to compare the means of normally distributed variables. For values with non-normal distribution, the Mann-Whitney U test was used. Normality was confirmed for all variables except for HOMA-IR. The chi-square test was applied to determine the association between two

categorical variables. To assess the relationship between food groups and NAFLD, multivariable-adjusted odds ratio (OR) obtained from the logistic regression analysis was performed. The relationship between dietary patterns and NAFLD was also determined from the ORs using the multivariate logistic regression adjusted for potential confounders. We defined two models to compute multivariable-adjusted ORs. The models were as follow: the first model was the crude model. The second model was adjusted for sex, education, physical activity, BMI, and WC. In addition to matching age in the design of the study, we entered it in the models and adjusted the relationships and effect sizes for the residual effects of this variable.

Results

In this study, two major dietary patterns were extracted. We labeled these factors as follows: the vegetable, legume, fruit, and low-fat dairy product (VLFD) dietary pattern; and the sweet, hydrogenated fat, red and processed meat, and soft drink (SHMS) dietary pattern (Table 2).

As shown in Table 3, NAFLD patients were less physically active compared to controls ($P < 0.001$). Moreover, weight, WC, BMI, body fat mass, serum AST, ALT, TG, and HOMA-IR were significantly higher in NAFLD patients compared to controls ($P < 0.05$).

Participants in the third tertile of the VLFD dietary pattern showed significantly lower weight, WC, BMI, body fat mass, serum levels of ALT and TG, and a lower

Table 2. Factor Loading Matrix for Major Dietary Patterns

Food Groups	Dietary Patterns	
	VLFD	SHMS
Vegetables	0.733	
Legumes	0.611	
Fruits	0.497	
Low-fat dairy	0.441	
White meat	0.409	0.311
Vegetable oils	0.402	
Whole grains	0.391	
Nuts	0.361	
Refined grains	-0.309	0.447
Sweets and desserts		0.711
Hydrogenated fats		0.678
Red and processed meat		0.658
Soft drinks		0.571
High-fat dairy		0.532
Fried potato		0.471
Pizza		0.425
Egg		0.382
Cooked potato		0.374
Pickle		0.325
Tea and coffee		0.321
Salt		0.307
Variance explained	10.71%	12.16%

VLFD, vegetables, legumes, fruits, and low-fat dairy; SHMS, sweets, hydrogenated fats, red processed meat, and soft drinks. Absolute values <0.3 were not displayed for simplicity.

consumption of energy, carbohydrate, and fat, but a higher protein intake and a greater serum level of HDL-C compared with those in the first tertile. However, there were no significant difference in physical activity level, serum levels of AST, TC, LDL-C, FBS, and HOMA-IR across tertiles of this pattern. Moreover, subjects in the third tertiles of the SHMS dietary exhibited higher weight, WC, BMI, body fat mass, serum levels of ALT and TG, and a greater intake of energy, carbohydrate, and fat, but a lower protein intake compared with those in the lowest tertile (Table 4).

Participants in the third tertile of red or processed meat consumption (OR: 2.68; 95% CI: 1.31–4.16), hydrogenated fats (OR: 2.66; 95% CI: 1.42–4.70), sweets and desserts (OR: 2.59; 95% CI: 0.89–3.48), and soft drinks (OR: 2.61; 95% CI: 1.32–5.22), showed a greater risk of NAFLD than those in the first tertile. Conversely, patients in the highest tertile of vegetable consumption exhibited a lower risk of NAFLD (OR: 0.34; 95% CI: 0.16–0.81) compared with those in the lowest tertile (Table 5).

As shown in Table 6, the VLFD dietary pattern was significantly associated with lower odds of NAFLD. In the first model, high adherence to the VLFD dietary pattern was related to lower odds of NAFLD compared to low adherence. Following further adjustment based on model 2, a significant downward trend was observed in the odds

Table 3. Characteristics of NAFLD Cases and Controls

	Cases (n = 105)	Controls (n = 105)	P
Age (years)	45.6 ± 9.1	45.4 ± 9.3	0.74 ^a
Male (n%)	42.8	42.8	0.50 ^b
Physical activity* (%)			0.001 ^b
Low	64 (60.9)	45 (42.8)	
Moderate	35 (33.3)	49 (46.6)	
High	6 (5.8)	11(10.4)	
Weight (kg)	87 ± 17.5	79.1 ± 14.5	0.01 ^a
Waist circumference (cm)	102.3 ± 13.5	96.3 ± 15.9	<0.001 ^a
BMI (kg/m ²)	33.8 ± 7.7	30.3 ± 6.5	<0.001 ^a
Body fat mass (kg)	31.1 ± 5.1	27.30 ± 6.9	<0.01 ^a
AST (U/L)	24.9 ± 8.1	20.3 ± 7.6	<0.001 ^a
ALT(U/L)	31.7 ± 13.1	21.4 ± 9.3	<0.001 ^a
TC (mg/dL)	189 ± 21.9	181.7 ± 24.8	0.12 ^a
TG (mg/dL)	132.8 ± 31.8	107.3 ± 39.1	<0.01 ^a
LDL-C (mg/dL)	124.7 ± 28.9	119.6 ± 36.2	0.33 ^a
HDL-C (mg/dL)	38.37 ± 14.6	40.5 ± 13.9	0.06 ^a
FBS (mg/dL)	94.3 ± 10.7	91.2 ± 13.7	0.23 ^a
HOMA-IR	3.9 (1.1, 6.3) ^d	2.2 (1.4–6.8) ^d	<0.01 ^c

NAFLD, Non-alcoholic fatty liver disease; BMI, Body mass index; MET-h, Metabolic equivalent hours; AST, Aspartate aminotransferase; ALT, Alanine aminotransferase; TC, Total cholesterol; TG, Triglyceride; LDL-C, Low-density lipoprotein cholesterol; HDL-C, High-density lipoprotein cholesterol; FBS, Fasting blood sugar; HOMA-IR, Homeostasis model assessment of insulin resistance.

Data are reported as mean ± SD or percentage as appropriate.

^a Independent samples t-test using equal variances (assessed and satisfied through Leven's test).

^b Chi-squared test.

^c Mann-Whitney U test.

^d Data were expressed as Median (25th and 75th percentiles)

*<600 MET/h per week: "low", 600-3000 MET/h per week: "moderate", >3000 MET/h per week: "high".

of NAFLD with increasing the adherence to this pattern. In contrast, adherence to the SHMS dietary pattern was significantly related to the increased risk of NAFLD. A significant increase was also observed in the odds of NAFLD with increased adherence to this pattern after adjustment for cofounders based on model 2 (Table 6).

Discussion

Dietary patterns identified by factor analysis provide a more realistic representation of daily eating habits.^{26,27} Based on our extensive search, this is the first study to assess the relationship between dietary patterns and NAFLD incidence among Iranian adults. In the current study, two major dietary patterns were extracted including VLFD and SMHS dietary patterns. The results demonstrated that the VLFD and SMHS dietary patterns were associated with NAFLD inversely and directly, respectively. Additionally, the VLFD dietary pattern was inversely associated with weight, WC, BMI, body fat mass, serum levels of ALT and TG, and intakes of energy, carbohydrate, and fat. However, the SHMS pattern was indicated to be positively associated with weight, WC, BMI, body fat mass, serum levels of ALT and TG, and intakes of energy, carbohydrate, and fat. Besides, among the major food groups, vegetable

Table 4. Characteristics of the Study Participants Across Tertiles (T) of the Dietary Pattern Scores

Variables	VLFD Dietary Pattern			SHMS Dietary Pattern		
	T1 (Lowest) (n = 70)	T3 (Highest) (n = 70)	P	T1 (Lowest) (n = 70)	T3 (Highest) (n = 70)	P
Age (years)	49.7±5.9	48.3±4.1	0.43	48.1±4.8	46.3±3.3	0.09
Physical activity (%)			0.08			0.1
Low	26 (37.1)	25(35.7)		28 (40.1)	29 (41.4)	
Moderate	33 (47.1)	34 (48.5)		30 (42.8)	31 (44.3)	
High	10 (14.2)	11 (15.7)		12 (17.1)	10 (14.3)	
Weight (kg)	81.4 ± 6.7	77.2 ± 4.3	0.001	78.7 ± 11.2	83.3 ± 9.9	0.001
Waist circumference (cm)	94.2 ± 10.7	87.2 ± 9.4	0.006	88.9 ± 10.8	95.6 ± 12.1	<0.001
BMI (kg/m ²)	29.7 ± 4.7	27.9 ± 5.3	0.001	26.1 ± 5.1	28.9 ± 6.9	<0.001
Body fat mass (kg)	31.1 ± 6.2	30.3 ± 8.3	0.03	28.3 ± 7.9	30.6 ± 8.8	0.003
Energy intake (kcal/d)	2245.1 ± 321	1987.8 ± 183	<0.001	1954.3 ± 211	2311.9 ± 196	<0.001
Carbohydrate intake (g/d)	291 ± 18.8	276 ± 23.9	0.002	265.6 ± 33.5	284.2 ± 41.4	0.003
Fat intake (g/d)	68.4 ± 18.4	61.3 ± 13.9	0.001	58.8 ± 11.7	67.7 ± 12.3	0.001
Protein intake (g/d)	61.7 ± 11.1	64.5 ± 9.7	0.007	67.4 ± 18.3	59.7 ± 14.7	0.001
AST (U/L)	23.5 ± 6.9	23.9 ± 7.2	0.27	24.3 ± 7.7	25.4 ± 9.4	0.43
ALT (U/L)	27.4 ± 9.8	25.1 ± 10.4	0.02	24.4 ± 8.1	27.3 ± 6.6	0.02
TC (mg/dL)	189.4 ± 18.4	191 ± 23.9	0.61	194.6 ± 34.6	201.6 ± 31.3	0.14
TG (mg/dL)	112.8 ± 21.8	87 ± 11.7	<0.001	91.6 ± 14.2	104.9 ± 21.9	0.001
LDL-C (mg/dL)	129.5 ± 28.8	126 ± 32.2	0.54	131 ± 34.2	133.5 ± 38.1	0.16
HDL-C (mg/dL)	42.4 ± 11.1	45.4 ± 13.6	0.003	46.3 ± 12.9	45.9 ± 10.8	0.29
FBS (mg/dL)	92.9 ± 13.6	91.7 ± 17.6	0.13	92.7 ± 11.8	94.7 ± 17.9	0.14
HOMA-IR	2.2 (1.1,6.3)	2.04 (1.2,5.8)	0.11	2.1 (1.1,6.1)	2.3 (1.01,6.4)	0.63

BMI, Body mass index; AST, Aspartate aminotransferase; ALT, Alanine aminotransferase; TC, Total cholesterol; TG, Triglyceride; LDL-C, Low-density lipoprotein cholesterol; HDL-C, High-density lipoprotein cholesterol; FBS, Fasting blood sugar; HOMA-IR, Homeostasis model assessment of insulin resistance.

VLFD, vegetables, legumes, fruits, and low-fat dairy; SHMS, sweets, hydrogenated fats, red processed meat, and soft drinks.

Mean ± SD are presented for normally distributed data; Median (25th and 75th percentiles) are presented for data not normally distributed (HOMA-IR).

Independent samples t-test using equal variances (assessed and satisfied through Levene's test).

Table 5. Odds Ratios (95% CI) for NAFLD Across Tertiles (T) of Food Groups

Food Groups	T1 (Low Adherence)	T2	T3 (High Adherence)	P-trend
Vegetables	Ref	0.73 (0.31–1.29)	0.34 (0.16–0.81)	0.01
Legumes	Ref	0.68 (0.21–1.32)	0.74 (0.61–1.67)	0.24
Fruits	Ref	0.63 (0.16–1.71)	0.54 (0.19–1.56)	0.52
Low-fat dairy	Ref	0.51 (0.14–1.51)	0.46 (0.16–1.32)	0.14
White meat	Ref	1.36 (0.64–3.94)	1.11 (0.48–4.17)	0.63
Vegetable oils	Ref	1.05 (0.38–1.87)	0.74 (0.29–1.44)	0.52
Whole grains	Ref	0.61 (0.38–1.44)	0.53 (0.31–1.21)	0.21
Nuts	Ref	0.93 (0.72–1.48)	0.84 (0.61–1.09)	0.08
Refined grains	Ref	1.33 (0.69–2.65)	2.11 (0.72–4.37)	0.32
Sweets and desserts	Ref	1.32 (0.72–2.41)	2.59 (1.11–3.48)	0.01
Hydrogenated fats	Ref	1.74 (1.24–3.11)	2.66 (1.42–4.70)	0.02
Red and processed meat	Ref	1.34 (0.89–2.91)	2.68 (1.31–4.16)	<0.01
Soft drinks	Ref	1.31 (0.63–2.45)	2.61 (1.32–5.22)	<0.01
High-fat dairy	Ref	1.21 (0.64–1.41)	2.01 (0.81–3.11)	0.28
Fried potato	Ref	1.01 (0.84–1.69)	1.44 (0.91–2.78)	0.46
Pizza	Ref	1.29 (0.71–2.79)	2.22 (0.88–3.45)	0.41
Egg	Ref	1.18 (0.51–2.31)	1.38 (0.71–2.59)	0.54
Cooked potato	Ref	1.19 (0.79–2.34)	2.04 (0.86–3.26)	0.59
Pickle	Ref	1.11 (0.71–1.41)	2.01 (0.84–2.61)	0.61
Tea and coffee	Ref	1.09 (0.56–1.33)	1.52 (0.61–2.44)	0.66
Salt	Ref	1.29 (0.72–1.55)	2.31 (0.88–3.59)	0.22

NAFLD, Non-alcoholic fatty liver disease.

Table 6. Odds ratios (95% CI) for NAFLD Across Tertiles (T) of Dietary Pattern Scores

Dietary Pattern	T1 (Low Adherence)	T2	T3 (High Adherence)	P-trend
VLFL (n = 210)				
Model 1 ^a				
Age (years)				
30–39	Ref	0.52 (0.27–1.27)	0.31 (0.18–0.95)	0.001
40–49	Ref	0.61 (0.33–1.33)	0.41 (0.11–0.83)	0.006
50–59	Ref	0.71 (0.21–1.27)	0.66 (0.16–0.94)	0.032
Model 2 ^b				
Age (years)				
30–39	Ref	0.39 (0.17–1.41)	0.27 (0.12–0.79)	0.012
40–49	Ref	0.44 (0.24–1.27)	0.33 (0.11–0.84)	0.025
50–59	Ref	0.55 (0.31–1.22)	0.28 (0.16–0.93)	0.033
SHMS (n = 210)				
Model 1 ^a				
Age (year)				
30–39	Ref	1.53 (0.55–3.01)	2.79 (1.21–3.87)	0.041
40–49	Ref	1.64 (0.74–3.44)	2.73 (1.31–5.83)	0.033
50–59	Ref	2.01 (1.05–4.42)	4.44 (1.81–7.12)	0.039
Model 2 ^b				
Age (year)				
30–39	Ref	1.69 (0.69–3.79)	2.93 (1.54–5.12)	0.026
40–49	Ref	1.21 (0.65–3.45)	3.17 (1.04–6.66)	0.033
50–59	Ref	1.71 (0.77–4.44)	4.22 (1.22–6.91)	0.013

NAFLD, Non-alcoholic fatty liver disease; VLFL, vegetables, legumes, fruits, and low-fat dairy; SHMS, sweets, hydrogenated fats, red processed meat, and soft drinks.

Multivariable-adjusted odds ratio (OR) obtained from the logistic regression analysis.

^a Crude model (model 1).

^b Adjusted for sex, education, physical activity, BMI, and waist circumference (model 2).

consumption was found to play a protective role against NAFLD.

The results obtained in this study are in line with those of some previous studies. In this context, a case-control study on the elderly Brazilian population reported that NAFLD is inversely associated with adherence to the healthy dietary pattern and is also related to the regional snacks pattern directly, which is characterized by the presence of regional foods with high carbohydrates and high glycemic load, after adjusting for confounding variables.¹⁹ Similarly, in another study, high-carbohydrate/sweet pattern scores were positively related to the prevalence of NAFLD in female subjects.²⁸ In a study on 999 Chinese adults, after adjusting for potential confounders, “animal food” and “grains-vegetables” dietary patterns were found to be directly and inversely associated with the prevalence of NAFLD, respectively.²⁹ In a Greek case-control study, subjects in the 4th quartile of the “fast-food type” dietary pattern showed 3.9 times higher odds for NAFLD compared to the individuals in the 1st quartile, after adjustment for confounders.³⁰ Similarly, a high meat/fast food pattern significantly increased the risk of NAFLD in Lebanese patients, whereas the “traditional Lebanese” pattern which is characterized by high consumption of vegetables, legumes, and vegetable oil/olives was inversely related to the odds of NAFLD.³¹ Moreover, a Western dietary pattern was related to the increased risk of NAFLD in a population-based cohort of adolescents.¹⁴ However,

no association was found between the Western and high-carbohydrate pattern and NAFLD risk in Korean adults.²⁷ Dietary patterns change among ethnicities, cultural groups, gender, and they may also vary as a result of personal preferences and food availability, which may explain the discrepancy in the obtained results.^{32,33}

The Mediterranean diet, which is rich in fibers, antioxidants, and monounsaturated fats, has been demonstrated to be protective against NAFLD.^{34,35} In this regard, Kontogianni et al³⁶ investigated the effect of adherence to the Mediterranean diet on the presence and severity of NAFLD in adult patients. Their findings demonstrated that higher adherence to the Mediterranean diet was associated with lower degrees of insulin resistance (IR) as well as less severe liver disease in patients with NAFLD. In the present study, the “VLFL” pattern had the major components of the Mediterranean diet containing a wide range of nutraceuticals such as phytosterols and polyphenols, which can attenuate hepatic steatosis through antioxidant and anti-inflammatory activities. Polyphenols may have hepatoprotective effects by increasing fatty acid oxidation and modulation of IR, oxidative stress, and inflammation, which are known as the major factors involved in the pathogenesis of NAFLD.³⁷

The “SHMS” dietary pattern includes most of the main components of the Western diet. Also, it has been reported that an inappropriate dietary pattern like the Western food style, which is characterized by high consumption of soft

drinks, saturated fats, trans fats, red and processed meat, and refined grains, may contribute to hepatic steatosis risk through increased total and visceral fat mass, IR, and increased hepatic *de novo* lipogenesis.^{4,14,38,39} In this study, although the consumption of tea and coffee was not associated with odds for NAFLD significantly, coffee and tea were included in the SMHS dietary pattern. In contrast to our findings, some previous studies have reported that consumption of tea and coffee can have protective effects against NAFLD.^{18,40} The discrepancy between these results might be due to the consumption of sugar and sweets along with tea or coffee. In the current study, the associations of VLFD and SMHS dietary patterns with NAFLD remained to be significant even after controlling for confounding variables, which indicated that these dietary patterns directly act on NAFLD.

The most important strength of the current research was that this is the first study investigating the association between dietary patterns and NAFLD in patients newly diagnosed with NAFLD, receiving no treatment. However, a few limitations of this study are also notable. Firstly, some inflated OR estimates and confidence limits may be an indication of sparse-data bias.^{41,42} Finally, due to the case-control design of this study, the causality effect could not be indicated; therefore, our findings need to be confirmed in future prospective studies.

In conclusion, the present study indicated that the VLFD dietary pattern was associated with reduced likelihood of having NAFLD, while the SHMS dietary pattern was related to higher likelihood. Thus, the VLFD dietary pattern might be useful in the nutritional strategies for NAFLD patients. However, further studies with larger sample sizes and prospective design are warranted to confirm our findings.

Authors' Contribution

HT drafted the manuscript and analyzed and interpreted the data; MAJ was the statistic counsellor; AO and MSA contributed to the final revision of the manuscript. All authors approved the final version of this manuscript.

Conflict of Interest Disclosures

All authors declare that there is no conflict of interest.

Ethical Statement

The Ethical Committee of Tabriz University of Medical Sciences approved the study protocol.

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