Original Article

The Association Between the Dietary Inflammatory Index (DII) and Some Serum Oxidative Stress Markers in Non-Alcoholic Fatty Liver Disease: Case- Control

Abstract

Purpose: Non-alcoholic fatty liver disease (NAFLD) is the most common liver disorder. The purpose of this study was to determine the relationship between the dietary inflammatory index (DII) and the serum oxidative stress markers in patients with NAFLD. **Methods:** In this case-control study, 121 patients with NAFLD and 119 healthy subjects were frequency-matched on gender. DII scores were calculated by using a 168-item food frequency questionnaire (FFQ). Blood samples were collected to measure serum oxidative markers. Linear regression and odds ratio (OR) were also used in this study. **Results:** The mean ± standard deviation of age for case and control group was 38.04 ± 6.7 and 35.6 ± 10.2, respectively. The gender ratio (female to male) for the case and control group was 1:1.42 and 1:1.38, respectively. The mean of the DII in the patient group was significantly higher than the healthy group, (P-values < 0.01). There was a significant negative relationship between TAC and DII (B = -2.63 (95%CI: -4.59, -0.68) and there was also a positive relationship between Malondialdehyde (MDA) and DII (B = 0.15 (95%CI: 0.02, 0.28) in the healthy group, but they were not significant in the case group. After multivariate adjustment, subjects in the most pro-inflammatory DII group had 73 times higher odds of NAFLD compared to subjects in tertile 1 (OR = 72.9; 95%CI (14.3-371.9)). **Conclusions:** Our findings suggest a direct association between the pro inflammatory properties of diet in patient and healthy group, but no relationship between TAC, MDA, and DII in the case group.

Keywords: *Dietary inflammatory index malondialdehyde, non-alcoholic fatty liver disease, total antioxidant capacity*

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Introduction

Non-alcoholic fatty liver disease (NAFLD) is recognized as a clinic pathological condition that may advance to end-stage liver disease. One of the main causes of this disease is insulin resistance in obese people that is characterized as a clinical condition associated with lipid deposition in hepatocytes.[1,2] NAFLD is the most common liver disorder in the United States. Although estimates are different, recent findings show that 34% of the US population aged 30-65 has the disorder.[3] According to the diagnostic means used, the prevalence of NAFLD among the Iranian adult population is 21.5%.[4] Its prevalence is higher in men and increases with age. The incidence of fatty liver is increasing in Western countries.[5] The disease depends on many factors including genetic, metabolic, environmental, microbial, and other factors and is strongly associated with lifestyle.[6]

Some cytokines including Tumor necrosis factor-alpha (TNFα), beta-growth factor, interleukin-8, and interleukin-15 increase intracellular inflammation in liver cells and finally lead to fibrosis.[1,7] Research has shown that C-reactive protein (CRP) and inflammatory cytokines such as Interleukin-6 (IL-6) and TNF-alpha are significantly higher in obese individuals.[8] Chronic inflammation can also lead to insulin resistance and metabolic syndrome.[9] In addition, diet may play an important role in the development of inflammation. For example, the Western dietary pattern, characterized by high meat intake, refined grains, simple carbohydrates, and fried foods is associated with higher levels of IL-6 and TNF-alpha.[10] Some diets such as the Mediterranean diet characterized by whole grains, olive oil, and low amount of red meat and saturated fat is associated with low inflammation. On the other hand,

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high vitamin E and vitamin C intake are associated with lower levels of inflammation.[9]

The dietary inflammatory index was designed to assess the inflammatory potential of individual diets. The purpose of the DII was to develop a tool that could assess a person's diet based on the characteristics of maximal pro-inflammatory to maximal anti-inflammatory markers. Previously, DII was shown to be associated with various inflammatory markers including CRP, IL-6, tumor necrosis factor, and homocysteine.[11]

To the best of our knowledge, few studies have investigated the association between DII and oxidative parameters in patients with NAFLD; therefore, the aim of this study was to examine the association between the DII index, stress oxidative markers such as Malondialdehyde and Total antioxidant capacity in patients with NAFLD and also examine the association between DII and non-alcoholic fatty liver disease in this case-control disease.

Methods

Population

This case-control study was conducted at [removed for blind peer review]' from September 2019 to February 2020; It was performed according to the guidelines laid down in the Declaration of Helsinki and all procedures involving research study participants were approved by the Ethics Committee of '[removed for blind peer review]'. (Ethics code: IR.MUI.REC. 1398.279 and study project code: 398293).

Written informed consent was obtained from all subjects prior to participation and were entitled to withdraw from the study at any time.

The calculation of sample size was based on an alpha level of 0.05 and a power of 90%.[12,13]

Both control and patient groups diagnosed with NAFLD with abdominal ultrasound were recruited from the Hospital of [removed for blind peer review]. In this study, 121 patients (71 males and 50 females) and 119 healthy subjects (69 males and 50 females) by convenience sampling method were selected. They were between 18 and 50 years old. All patients admitted to [removed for blind peer review]'hospital with elevated liver function tests, no alcohol history, no drug usage, with negative viral hepatitis and autoimmune serology were extra evaluated for NAFLD. The women in this study were not be menopausal. The control group, similar to the case group who had been evaluated by an ultrasound specialist from the same place was recruited. The inclusion and exclusion criteria were the same for both groups.

Inclusion criteria for case and control groups

(a) Fatty liver confirmed by abdominal ultrasound, (b) willingness to participate in the study, (c) age between 18 and 50 years, (d) being free from conditions such as pregnancy, lactation, gastric surgery within one year prior to the start of the study, hysterectomy, hepatitis or biliary disease, arthritis or inflammatory diseases, cancer, hemochromatosis, Wilson disease, (e) weight loss over 10% within the previous three months and f) not having followed a specific diet since diagnosis.

Exclusion criteria for case and control groups

(a) Unwillingness to comply with to the study, (b) incomplete demographic or anthropometric information, (c) reporting caloric intake >5000 or <800 kcal/day.[14] After the consent form was signed, all participants underwent abdominal ultrasound by a radiologist. Participants were divided into the case and control groups.

Anthropometric measurements

All anthropometric data were measured by a trained nutritionist. Using a stadiometer, height was measured without shoes and recorded to the nearest 0.5 cm. Body weight was recorded with the least clothing and without shoes and to the nearest 100 g. Body mass index (BMI), was calculated as weight divided by height squared (expressed as kg/m2). Patients were classified as "obese," "overweight," or "normal weight" according to the World Health Organization (WHO) obesity classification of 2004.[15] Waist circumference was measured at the midline of the lower rib margin and iliac crest in standing position with normal breathing at the end of exhalation. Waist and hip circumference were measured with the least possible clothing by using a plastic measuring tape, to the nearest 0.5 cm. Abdominal obesity were classified as waist circumference ≥88 cm and ≥102 cm for women and men, respectively.[16]

Assessment of other variables

A demographic data and lifestyle questionnaire including age, sex, occupation, education, medicine or supplement information, current smoking and economic status and were recorded.[17]

We established the different categories of SES, as a latent variable, using latent class analysis (LCA) technique based on a number of indicator variables such as income, ownership of a house, car, or personal computer, type of house and car and ability to travel. LCA classifies similar individuals in terms of SES into homogeneous latent classes. This leads participants within each latent class are highly similar to each other and uniquely different from the other classes across the set of evaluated items.

Assessment of physical activity

IPAQ questionnaire was used to assess physical activity levels.[18] Participants were asked their daily activities such as walking, exercise, sleep, hours dedicated to watching television, homework and etc., as well as the intensity of

activity report. Total activity was stated for 24 hours and metabolic equivalents (MET) were computed.

Calculation of DII scores

A validated 168-item food frequency questionnaire (FFQ) was used to assess typical food intakes over the previous year.[19] Participants were requested to report the frequency of consumption for each food item during the previous year (times per day, week, month or year, or never). Information obtained from the FFQ was evaluated by Nutritionist IV.

The inflammatory potential of the diet was calculated using DII appearance by Shivappa *et al*. Its details and validation have been established.[11,20]

Based on the possible list of the 45 food parameters, a total of 26 were available from the FFQ for the calculation of DII: Vitamin B12, Vitamin B6, beta-carotene, caffeine, carbohydrate, cholesterol, energy, total fat, fiber, folic acid, iron, magnesium, monounsaturated fatty acid (MUFA), polyunsaturated fatty acid (PUFA), niacin, omega-3 fatty acid, omega-6 fatty acid, protein, riboflavin saturated fatty acids (SFA), Thiamine, Vitamin A, Vitamin C, Vitamin D, Vitamin E, Zinc.

Oxidative stress biomarkers

Serum Malondialdehyde (MDA) level was determined by colorimetric assay using thiobarbituric acid reagent. MDA reacts with thiobarbituric acid in an acidic environment to form a pink compound which is read at 532 nm.[21]

The serum total antioxidant capacity (TAC) was determined by measuring the ability of the serum to recover ferric ions. In this method, the antioxidants in the serum ferric ion are converted to ferrous ions at low PH, which in combination with tripyridyltriazine, create a color complex the intensity of which was measured at 593 nm in this study.[22]

Statistical analyzes

In the present study, quantitative variables were reported as mean and standard deviations (SD)[23] and qualitative variables were reported as numbers (percent). The normal distribution of data was evaluated the by Kolmogorov-Smirnov test. Quantitative variables were compared between the two groups using the independent samples t-test and qualitative variables were analyzed using the Chi-square test. The nutrient intake means and the main variables in the DII scales tertile were compared using the ANOVA test. The correlation between DII, TAC and MDA was determined by linear regression data analysis using SPSS Ver 21 software. Linear regression was used to find the association of DII with TAC and MDA.

Odds ratio (OR) and 95% confidence intervals for presence of non-alcoholic fatty liver as outcome were estimated using logistic regression models, adjusting only for age, gender, and education then fitting a model with additional adjustment for age, gender, and education, BMI, SES and physical activity and the other adjustment was in terms of age, gender, and education, BMI, SES, and physical activity, energy, medication, and supplementation. *P* value <0.05 was considered statistically significant.

Results

Two groups were frequency-matched on gender. The demographic characteristics and Dietary inflammatory index (DII) of the participants in the two groups are shown in [Table 1]. This table shows that the means of the age, bodyweight, waist circumference, hip circumference, BMI, and DII in the case group are significantly higher than those in the control group (*P*-value <0.001).

The qualitative variables are shown in Table 2. A significant difference was observed between the education level and occupation in the study groups (*P*-value <0.05). However, there was no significant difference in terms of the gender, smoking habits, and Socioeconomic Status (SES) of the participants (*P*-value >0.05). Table 3 shows that kcal, protein, carbohydrates, fat, SFA, MUFA, PUFA, Zinc, and Selenium means in the case group are significantly higher than the control group (*P*-value <0.05), but the mean of Beta-Carotene in the case group is significantly lower compared to the control (*P*-value <0.001).

Table 4 shows that the TAC means in the control group are significantly higher compared to the case group (*P*-value <0.01). Also, it shows that the MDA means in the control group are significantly lower compared to the case group (*P*-value <0.01).

In both groups, the means of these two indicators were calculated separately according to the tertiles, which TAC and MDA were not significant in the case group (*P*-value = 0.625), but they were significant in the control group (*P*-value = 0.039).

The Regression analysis of the association of DII with TAC, MDA in the case and control groups are shown in Table 5. In the case group, there is no significant relationship between TAC and MDA with DII in either of the models (*P*-value >0.05). In the control group there

Table 1: Demographic characteristics and Dietary inflammatory index (DII) of participants in two groups

Qualitative variables	Patients	Healthy subjects	*P
Age	38.04 (6.7)	35.6 (10.2)	0.037
Height	170.5 (11.1)	168.06 (9.4)	0.062
Bodyweight	82.9 (11.4)	65.7 (12.4)	<0.001
Waist circumference	102.5 (10.7)	81.7 (10.3)	<0.001
Hip circumference	102.2 (10.5)	91.9 (10.5)	<0.001
BMI	28.60 (4.03)	23.29 (4.14)	<0.001
Physical activity	115.9 (14.5)	116.8 (11.9)	0.076
DII	1.18 (1.19)	0.05 (0.79)	<0.001

Values are number mean (SD); **P* based on independent samples *t*-test

Table 2: Qualitative variables (gender, smoking, education, job and SES) of participants in two groups

Qualitative variables	Patients	Healthy subjects	*P
Gender			
Men	71 (58.7%)	69 (58%)	0.91
Women	50 (41.3%)	50 (42%)	
Smoking			0.15
No	109 (90.1%)	113 (95%)	
Yes	11 (9.9%)	6 (5%)	
Education			0.002
School education	20 (16.5%)	15 (12.6%)	
diploma	43 (35.5%)	27 (22.7%)	
Bachelor	39 (32.2%)	41 (34.5%)	
Master and phd	19 (15.7)	36 (30.3%)	
Job			0.001
Employ	36 (29.7%)	17 (14.3%)	
Private	6 (5%)	10 (8.4%)	
Student	2 (1.7%)	35 (29.4%)	
Housewife	36 (29.85)	21 (17.6%)	
Self-employ	32 (26.4%)	22 (18.5%)	
Unemployed	9 (7.4%)	14 (11.8%)	
SES			
Poor	10 (8.3)	10 (8.4)	0.114
Middle	78 (64.5)	90 (75.6)	
Excellent	33 (27.3)	19 (16.0)	
Drug			0.001
No	112 (94.1%)	87 (71.9%)	
Yes	7 (5.9%)	34 (28.1%)	
Supplement			
NO	97 (81.5%)	95 (78.5%)	0.561
Yes	22 (18.5%)	26 (21.5%)	

Values are number (percent); *P based on Chi-square test

is a significant negative relationship between TAC and DII (B = -2.63 (95%CI: -4.59, -0.68), P_value = 0.009) and a positive relationship between MDA and DII. (B = 0.15 (95%CI: 0.02, 0.28), P_value = 0.028).

Table 6 shows the odds ratio between DII and NAFLD. The results were analyzed using logistic regression and three adjusted models and the crude model. According to the results of table 6, the risk of NAFLD in people with high DII are significantly increased.

Discussion

This study assessed the association between the dietary inflammatory index and the oxidative biomarker (MDA) and antioxidant capacity in patients with NAFLD and healthy people.

In the case group, there is no significant relationship between TAC and MDA with DII but in the control group there is a significant negative relationship between TAC and DII and a positive relationship between MDA and DII. In addition, MDA levels and DII in healthy people were lower than those in the NAFLD patients while TAC was higher in the healthy group.

Also, in this case-control study, the relationship between inflammatory potential of diet measured by the DII, and the risk of non-alcoholic fatty liver was examined. We found that subjects with higher DII scores (demonstrating a pro-inflammatory diet) were at increased risk of non-alcoholic fatty liver. Insulin resistance is commonly detected in patients with fatty liver.[24] So, one of the probable mechanisms for the positive association between the DII and the risk of fatty liver might be the effect of a pro-inflammatory diet on insulin resistance.[25] Vahid *et al*. found that people who consumed more anti-inflammatory foods were at a lower risk of diabetes.[26] Previous study have shown that NAFLD is an insulin resistance condition and that low DII may be accompanied by lower risk of insulin resistance.[27] Another study showed that DII was associated with higher BMI, waist circumference, and higher waist to hip ratio.[28] Higher DII scores have also been positively associated with unhealthy metabolic obesity.[29] In our study, higher DII was associated with high risk for NAFLD.

Dietary patterns play a key role in the inflammatory process.[30,31] In addition, the accumulation of visceral fat can be the cause of inflammation and inflammatory processes.[32] Researchers have found that people with higher liver fat accumulation and obesity (BMI more than 30 kg/M^2) have a higher level of inflammation and DII.[33] BMI has been reported as an independent predictor of fatty liver.[34,35] Fatty liver can be caused by metabolic syndrome or other hepatic disorders such as steatohepatitis and liver failure.[36,37]

A study by Mazidi *et al*. Showed that a pro-inflammatory diet (high DII) was more likely to result in fatty liver and poor liver function tests in people with lower BMI.[38] This is exactly the same as our result because in our study, people on a pre-inflammatory diet were more likely to develop nonalcoholic fatty liver disease.

Ruiz-Canella *et al*. have shown that DII can be a useful tool for predicting the inflammatory capacity of the diet. It has also been shown that an increase in DII score is associated with an increase in CRP and glucose intolerance.[39] The results of our study show that increasing the DII score can increase serum inflammatory markers in control group.

Namazi *et al*. showed there was no significant relationship between metabolic syndrome and DII.[40] Due to the variety of studies and different cut-points in different studies may affect the result.

Previous studies have shown that high intake of antioxidant nutrients such as beta-carotene, flavonoids, and healthy fatty acids are inversely correlated with fatty liver. In the present research, the dietary intakes of beta-carotene in healthy people were significantly higher than those in NAFLD patients.[41] In addition, a diet with a higher inflammatory potential suggests that diet-induced

Table 3: The comparison of the mean (SD) of nutrients in the patients and healthy subjects groups by DII

Variables	Tertiles of dietary inflammatory index		Patients	Healthy subjects	**P
Kcal	Total		3651.08 (552.7)	2497.2 (553.4)	<0.001
	DII	Tertile 1	4113.79 (416.67)	2563.42 (463.15)	
		Tertile 2	3823.58 (507.36)	2476.30 (657.11)	
		Tertile 3	3402.24 (482.30)	2288.84 (526.62)	
	*P		<0.001	0.21	
Pro	Total		102.07 (20.9)	82.4 (20.8)	<0.001
	DII	Tertile 1	125.19 (15.56)	87.01 (18.70)	
		Tertile 2	109.38 (17.36)	79.55 (23.76)	
		Tertile 3	90.38 (15.53)	73.89 (15.88)	
	*P		<0.001	0.04	
Cho	Total		500.3 (84.8)	385.2 (91.1)	<0.001
	DII	Tertile 1	570.61 (67.76)	396.45 (77.50)	
		Tertile 2	524.53 (72.04)	382.84 (108.09)	
		Tertile 3	463.67 (77.70)	350.70 (82.57)	
	*P		<0.001	0.20	
Fat	Total		145.5 (28.1)	78.4 (21.3)	<0.001
	DII	Tertile 1	158.71 (24.97)	80.26 (19.54)	
		Tertile 2	151.44 (27.66)	78.03 (22.75)	
		Tertile 3	137.95 (27.41)	73.10 (24.38)	
	*P		<0.001	0.49	
Chol	Total		291.6 (123.9)	219.00 (104.7)	0.120
	DII	Tertile 1	335.91 (123.17)	229.35 (107.80)	
		Tertile 2	311.74 (119.82)	205.99 (106.34)	
		Tertile 3	265.77 (122.01)	216.65 (89.75)	
	*P		0.04	0.54	
Sfa	Total		31.7 (12.60)	15.63 (4.18)	0.007
	DII	Tertile 1	31.15 (6.30)	16.63 (3.71)	
		Tertile 2	33.59 (9.71)	14.80 (4.61)	
		Tertile 3	30.87 (15.33)	14.23 (3.93)	
	*P		0.57	0.03	
Monofat	Total		36.5 (10.10)	20.8 (7.17)	0.005
	DII	Tertile 1	40.88 (9.99)	20.64 (6.53)	
		Tertile 2	37.71 (8.94)	21.27 (7.90)	
		Tertile 3	34.56 (10.37)	20.38 (7.79)	
	*P		0.03	0.87	
Polyfat	Total		58.28 (12.71)	29.7 (11.49)	<0.001
	DII	Tertile 1	66.07 (11.23)	30.09 (11.12)	
		Tertile 2	60.01 (13.84)	29.90 (11.79)	
		Tertile 3	54.76 (11.26)	28.02 (12.64)	
	*P		<0.001	0.81	
Magnesium	Total		317.4 (79.92)	310.6 (70.07)	0.484
	DII	Tertile 1	429.16 (57.85)	330.61 (68.13)	
		Tertile 2	351.87 (44.95)	299.20 (65.77)	
		Tertile 3	261.42 (44.23)	268.45 (66.49)	
	*P		<0.001	<0.001	
Zinc	Total		10.2 (2.9)	9.28 (2.4)	0.009
	DII	Tertile 1	13.63 (2.41)	10.01 (2.30)	
		Tertile 2	11.69 (2.43)	8.78 (2.57)	
		Tertile 3	8.26 (1.69)	8.05 (1.98)	
	*P		<0.001	<0.001	

Contd...

Table 3: Contd...

Variables	Tertiles of dietary inflammatory index		Patients	Healthy subjects	**P
Selenium	Total		0.06 (0.034)	0.05 (0.032)	<0.001
	DII	Tertile 1	0.10 (0.03)	0.05 (0.03)	
		Tertile 2	0.07 (0.04)	0.05 (0.03)	
		Tertile 3	0.05 (0.03)	0.05 (0.04)	
	*P		<0.001	0.92	
Betacaroten	Total		632.08 (506.7)	1471.7 (1185.12)	<0.001
	DII	Tertile 1	1165.94 (722.73)	1726.26 (1475.63)	
		Tertile 2	676.12 (348.11)	1173.81 (716.59)	
		Tertile 3	432.13 (345.25)	1352.73 (791.77)	
	*P		<0.001	0.06	

Values are mean (SD); **P*-Values is based on ANOVA; ***P*-Values is based on independent samples *t*-test

Table 4: The comparison of the mean (SD) of TAC, MDA in the patients and healthy subjects groups by DII

			Patients	Healthy subjects	**P
Total antioxidant capacity	Total		13.78 (9.41)	17.82 (8.67)	0.001
	DII	Tertile 1	14.92 (9.03)	19.81 (8.80)	
		Tertile 2	12.58 (6.87)	16.23 (8.26)	
		Tertile 3	14.07 (10.74)	14.88 (7.96)	
	*P		0.625	0.039	
Malondialdehyde	Total		2.65 (0.99)	1.32 (0.59)	<0.001
	DII	Tertile 2	2.87 (0.91)	1.17 (0.54)	
		Tertile 2	2.82 (1.17)	1.51 (0.62)	
		Tertile 3	2.49 (0.88)	1.37 (0.51)	
	*P		0.151	0.014	

Values are mean (SD); **P*-Values is based on ANOVA; ***P*-Values is based on independent samples *t*-test

Table 5: Regression analysis of the association of DII with TAC, MDA in the patients and healthy subjects groups

	Patients		Healthy subjects	
	B (95%CI)	*P	B (95%CI)	*P
Total antioxidant capacity				
Crude Model	0.04 (-1.40, 1.48)	0.952	-2.63 (-4.59, -0.68)	0.009
Model 1	0.71 (-0.75, 2.17)	0.336	-2.56 (-4.48, -0.63)	0.010
Model 2	0.92 (-0.57, 2.42)	0.224	-2.38 (-4.34, -0.43)	0.017
Model 3	0.69(-1.06,2.46)	0.43	-2.41(-4.41,0.42)	0.01
Malondialdehyde				
Crude Model	-0.13 (-0.28, 0.02)	0.086	0.15 (0.02, 0.28)	0.028
Model 1	-0.12 (-0.28, 0.03)	0.124	0.15 (0.02, 0.28)	0.027
Model 2	-0.10 (-0.26, 0.06)	0.226	0.16 (0.02, 0.30)	0.024
Model 3	-0.046(-0.23,0.14)	0.6	0.16 (0.02,0.30)	0.02

**P*-value is based on linear regression model. Model 1: Adjusted for Age, Sex & Education. Model 2: Adjusted for Age, Sex, Education, physical activity, BMI & SES. Model 3: Adjusted for Age, Sex, Education, physical activity, BMI & SES, energy, taking medication and supplements

Table 6: Logistic Regression of the association of DII with NAFLD

	Dietary Inflammatory Index OR (95% CI)			
DII	Tertile1	Tertile2	Tertile3	*P
Crude Model	1	2.29 (1.18-4.46)	11.23 (5.36-23.56)	<0.001
Model 1	1	2.24 (1.12-4.46)	11.20 (5.21-24.09)	<0.001
Model 2	1	2.9 (1.21-7.24)	20.59 (7.27-58.25)	<0.001
Model 3	1	5.90 (1.33-26.1)	72.9 (14.3-371.9)	<0.001

**P*-value is based on binary logistic regression model. Model 1: Adjusted for Age, Sex and Education. Model 2: Adjusted for Age, Sex, Education, physical activity, BMI and SES. Model 3: Adjusted for Age, Sex, Education, physical activity, BMI and SES, energy, taking medication and supplements

inflammation may increase obesity, especially central obesity, in an overweight or obese population. Diet can alter body metabolism, oxidation of fats, and increase and accumulate fat in the liver which leads to elevated concentrations of inflammatory markers such as C-reactive protein and IL-6.[20,42]

Hermsdorff showed that dietary TAC values were inversely related to lipid and glucose biomarkers as well as central fat size in healthy young adults and demonstrated that dietary TAC as a useful means to measure the health benefits of cumulative antioxidant capacity from food intake.[28] In our study, there was a significant difference between the case and control groups in terms of TAC and was greater in the healthy group. Also, in the healthy group, a significant relationship was observed between DII and TAC.

On the other hand, MDA can inactivate superoxide dismutase enzymatic. Antioxidant protection systems may be elaborated in the development of NAFLD.[43] Both of these biomarkers can predict the body's oxidative state.

This is the first study to evaluate the association between DII and non-alcoholic fatty liver disease and its association with TAC and MDA. One of the strengths of our research was the use of a valid and repeatable 168-item FFQ for Iranian participants which helped to understand the questionnaire better.

One of the limitations of our study was the use of ultrasound instead of fibro-scan due to financial limitations. Ultrasound sonography may be associated with an error in the diagnosis of NAFLD. The FFQ is still the most appropriate tool for collecting nutritional data in large epidemiological studies, but it has some limitations. Accurate reporting relies on respondent memory. Bias may be introduced with respondents reporting eating according to social desirability, thus resulting in over-estimation of certain foods and under-estimation of other items.[44] Finally, our findings also showed that there was a significant negative relationship between the DII score and TAC in healthy individuals. Additionally, a low DII score was significantly correlated with a low MDA level. There was no significant relationship between serum TAC and MDA with DII in NAFLD patients.

Conclusions

To sum up, people who have more pro-inflammatory diets was in NAFLD group. Therefore, consuming more foods with anti-inflammatory nutrients, including omega-3 fatty acids and plant-rich foods, carotenoids and phytochemicals, and reducing the consumption of inflammatory agents such as fried foods, processed foods, refined carbohydrates, and saturated fatty acids should be encouraged.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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