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Original article

The relationship between dietary antioxidant intake and physical activity rate with nonalcoholic fatty liver disease (NAFLD): A case – Control study

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SUMMARY

Introduction: Although dietary intakes, especially micronutrients, can be associated with the severity of nonalcoholic fatty liver disease (NAFLD), investigations on the amount of vitamins and antioxidants consumption and their relationship with NAFLD are very limited and incomplete. Therefore, we decided to investigate the relationship between antioxidant compounds intake and physical activity rate with NAFLD.
Methods: In this study, 200 patients with NAFLD for the case group and 400 healthy subjects for the control group were selected. Patients were diagnosed as NAFLD after giving blood tests and performing Ultrasonography by a radiology specialist. Dietary intakes were evaluated through a validated 168-items semi-quantitative food frequency questionnaire (FFQ). Physical activity rate was estimated by a validated short form of International Physical Activity Questionnaire (Short IPAQ).
Results: The study population was between 20 and 60 years old and 46% of them were women. Weight, waist circumference, hip circumference, WHR, and BMI in the cases were higher than the controls. Physical activity comparisons showed that controls had higher physical activity rate than cases. Mean consumption of vitamins C, A, D and alpha-tocopherol in case group was less than the other group. After adjustment for all potential confounder, subjects who were in highest tertile of vitamin A intake -in comparison to those in the lowest tertile of intake-decreased risk of NAFLD (OR = 0.40, 95%CI: 0.30–0.55). The same finding was obtained for vitamin D; [Top category vs. bottom category of vitamin D of intake (OR = 0.35, 95%CI: 0.20–0.61)].
Conclusions: We found that more intakes of vitamins A and D are related to lower risk of NAFLD in this group of Iranian adults. Physical activity rate in cases was less than the controls. Further prospective studies are required to confirm causal association between antioxidant compounds intake and NAFLD.

1. Introduction

Nonalcoholic fatty liver disease (NAFLD) is a pathologic aggregation of fat (mainly triglycerides) in the liver at subjects who have no history intake of alcohol. This disorder is the most prevalent cause of chronic liver diseases [1]. Prevalence of this disease has been estimated about 10–35% of adults in the worldwide. In Iran, 7% of children and 35% of adults have NAFLD [2]. Several variables have been associated to NAFLD, such as diabetes, overweight or

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obesity, hyperlipidemia, jejunoileal bypass and the use of some drugs. The pathophysiology of NAFLD and its progress to fibrosis are still unknown [3]. Several studies have shown the impact of oxidative stress on chronic diseases such as NAFLD [4]. Also, previous studies suggested that oxidative stress has an important role in the pathophysiology and severity of NAFLD [2]. Reactive Oxygen Species)ROS(can block hepatocytes to secret the very low density lipoprotein (VLDL), and result in liver fat aggregation [5].

Some antioxidant defense system components, such as superoxide dismutase enzymes, glutathione peroxidase, catalase, glutathione reductase and uric acid are made inside the body (endogenous), but others, such as vitamins E, C, A and minerals selenium (Se) and zinc (Zn), should be obtained by diet (exogenous) [3].

Some of previous studies have shown that different dietary habits between Asians and Western population had an important role in increasing NAFLD incidence [6]. In some studies, a relationship between some micronutrients and NAFLD was found out. Patients with NAFLD have a lower intake of vitamin D and antioxidant vitamins (E and C) than healthy individuals [7]. But, the use of antioxidants as supplements to prevent NAFLD is not clearly proved. Increasing physical activity (PA) is another key factor in management of NAFLD. Increasing PA could protect against the development of obesity, type 2 diabetes and some other NAFLD related risk factors including dyslipidemia and hypertension [8,9]. Thus, it is assumed that increased PA might be effective in NAFLD risk reduction. A prior study has shown that increasing PA might dose-dependently decrease the risk of NAFLD, and the last guidelines proposed a minimum of 150 min PA each week to moderately decrease the NAFLD risk [10]. However, studies on the physical activity rate and its relationship with NAFLD are very limited and no study has been done in Iran.

Although diet has a major contribution in NAFLD and diet composition, especially micronutrients, affects the severity of the disease, studies on the amount of vitamins and antioxidants consumption and their relationship with NAFLD are very limited and incomplete. Also, studies done in Iran are limited. Therefore, we decided to investigate the relationship between antioxidant compounds intake and physical activity rate with NAFLD in Iranian population.

2. Methods and materials

2.1. Participants

The present study has a case–control design. It was approved by the Isfahan University Ethics Advisory Committee. Patients with NAFLD who admitted Isfahan fatty liver research center were included, based on inclusion criteria as the case group.

2.2. Inclusion and exclusion criteria

Individuals entered to the study for case group that were between 20 and 60 years old and were diagnosed as NAFLD patients after giving blood tests and performing Ultrasonography by a radiology specialist [11]. Subjects who were the neighbors of the case group and not suffered from any stage of hepatic steatosis were randomly chosen and were matched with cases in age and sex. Individuals with alcohol consumption, following special diets during the last 6 months, with regular use of any nutritional supplements in the last 4 months, pregnant or breastfeeding women, individuals with type B or C hepatitis and chronic diseases were excluded. Subjects who did not want to continue the study or did not respond to more than 35 food items from the food frequency questionnaires and those who were receiving less than 800 kcal and more than 4200 kcal of energy each day, were eliminated from the study (n = 7).

In this study, according to the following formula, 200 people for case group and 400 for control group were selected.

$$n = \left(\frac{1+\varphi}{\varphi}\right)\frac{\left(z_{1-\frac{\alpha}{2}} + z_{1-\beta}\right)^2}{(\log OR)^2\overline{\pi}(1-\overline{\pi})} = 200$$

[Considering that z = 1.96, OR = 1.5 and prevalence (π) = 0.35]. Controls were twice the case group.

2.3. Method of assessing dietary intake, physical activity rate and other variables

Dietary intakes were assessed through a food frequency questionnaire (FFQ). This semi-quantitative FFQ was previously validated among Iranian population [12]. The FFQ contains 168 food items. For each food item, subjects reported their frequency of consumption (never or 1/month, 2–3/month, 1–3/week, 4–5/week, once a day, twice a day, or more than 2 times a day) in the last year. Then, foods were converted into their ingredients, and their amounts were calculated into gram per day. A modified Nutritionist 4 (Version7; N-squared computing, OR USA) software (NUT4) for Iranian foods was applied to calculate energy, macronutrients and micronutrients intakes of foods (including antioxidant vitamins and minerals, such as vitamin E, C, D, beta-carotene, alpha-tocopherol, Se and Zn). A previous validation study of this FFQ among 132 randomly chosen participants revealed reasonable correlations between dietary intakes assessed by similar FFQ and multiple days of 24-h food recalls completed during the year ($r = 0.3$–0.8; $P < 0.05$) [12]. For instance, the correlation coefficient for vitamin E was 0.79; which means that this FFQ provides reasonably valid and reliable measures of the average long-term intakes of nutrients [12].

Physical activity rate was estimated by the use of a validated short form of International Physical Activity Questionnaire (Short IPAQ) [13]. It contains 7 questions about: low, intermediate and high level of physical activity. Then, physical activity rate of each subject was calculated based on metabolic equivalent task (MET)-minutes/day.

Data about other variables including demographic features age, sex, SES (marital status, education, type of occupation, home status, home type, income and foreign travel), family size, disease history as well as smoking status were collected through a general information questionnaire. Body weight was measured through a digital scale with light clothes and without shoes. Height was measured through a tape measure while participants were standing in a normal position with no shoes. Body mass index (BMI) was calculated as the weight in Kg divided by height in m squared. Also, waist circumference was obtained in the narrowest region of participants' waist between the last rib and the upper part of the pelvic floor through a tape measure. Hip circumference measurement was made through a tape measure while subjects were in a standing normal position and without extra clothes and in the head area of the largest femur; and WHR (waist-to-hip ratio) was calculated.

2.4. Statistical analysis

Continuous variables were presented as mean ± standard error (SE) or median and range (R) and qualitative data were reported as percentages and frequency. We compared continuous variables between cases and controls by independent sample t-test and analysis of covariance, as appropriate. Categorical variables were also compared using Chi-square between two groups. Relationship between anti-oxidant compounds intake and physical activity with NAFLD were assessed using logistic regression. The antioxidants intakes were categorized into three based on the tertiles of intake. The odds ratio (OR) with 95% confidence interval (CI) was presented for the association between intake of antioxidants and NAFLD; while the reference category was the lowest tertile of intake. We used SPSS software version 23 (SPSS Inc, Chicago, IL, USA) for all analyses. We considered P values less than 0.05 as statistically significant.

3. Results

Demographic, anthropometric and physical activity rate information of 200 cases and 400 controls are listed in Table 1. The case group had higher weight than control group (83.32 ± 0.70 vs. 65.66 ± 0.44; P < 0.001). Physical activity comparisons showed that controls had higher physical activity rate than cases (1574.43 ± 46.34 vs. 1100.81 ± 43.02; P < 0.001). The comparison of the two groups exhibited significant differences in body mass index (BMI) waist circumference, hip circumference and waist to hip ratio (WHR) (P < 0.001 for all).

Prevalence of NAFLD in tertiles antioxidants intake is shown in Table 2. The prevalence of NAFLD was lower in the highest tertile of vitamins A and D (for vitamin A: P = 0.02 and for vitamin D: P < 0.001). Also, it exhibited that highest tertile of Zn and vitamin E intake has significant direct association with prevalence of the disease.

Comparison of antioxidants intake between cases and controls is shown in Table 3. Mean consumption of vitamins C, A, D and alpha-tocopherol in case group was less than other group (P < 0.05). No significant differences were found between vitamin E, Zn and Se intakes in cases and controls. Food groups and macronutrients intake of study participants are shown in Table 4. Mean intake of vegetables in case group was less than the other group (P < 0.05); and mean intake of refined grains and high-fat dairy in case group was higher than controls (P < 0.05).

Multivariable-adjusted odds ratios and 95% confidence interval (CI) for NAFLD across different categories of antioxidants compounds intake are listed in Table 5. For vitamin A, subjects who were in the highest tertile of intake -in comparison to those in the lowest tertile of intake-had 60% lower risk of NAFLD (OR = 0.40, 95%CI: 0.30–0.55), in crude model. This relation was significant even after making adjustment for all confounding variables

Table 1
Basic characteristics and physical activity of the study population (n = 600).[a]

Variable	Cases (n = 200)	Controls (n = 400)	P[e]
Age (yr)	39.10 ± 0.60	37.57 ± 0.44	0.05
Man	111 (56%)	210 (53%)	0.54
Woman	89 (44%)	190 (47%)	
Weight (kg)	83.32 ± 0.70	65.66 ± 0.44	<0.001
Waist- circumference (cm)	102.32 ± 0.51	84.86 ± 0.31	<0.001
Hip circumference (cm)	105.03 ± 0.58	96.64 ± 0.30	<0.001
WHR[b]	0.97 ± 0.005	0.87 ± 0.003	<0.001
BMI[c] (kg/m^2)	30.63 ± 0.27	24.95 ± 0.15	<0.001
MET (minutes/week)	1100.81 ± 43.02	1574.43 ± 46.34	<0.001
SES[d]	8.42 ± 0.08	8.38 ± 0.06	0.71
Family size	3.42 ± 0.07	4.21 ± 0.07	<0.001
Smoking			0.03
Yes	14 (7%)	12 (3%)	
No	186 (93%)	388 (97%)	
Disease history			
Yes	7 (3%)	10 (2.5%)	0.46
No	193 (97%)	390 (97%)	

[a] Data are presented as mean ± SE or percentages and frequency as appropriate.
[b] WHR: waist-*to*-hip ratio.
[c] BMI: body mass index.
[d] SES: family size + education + home status + home type + foreign travel.
[e] Obtained from independent sample t-test for continuous variables and Chi-square for Categorical variables.

Table 2
Prevalence of NAFLD in tertiles antioxidants intake.

	Tertiles of dietary antioxidants intake			P[b]
	T_1	T_2	T_3	
Vitamin C intake (mg/day)[a]	20.76–90.1	90.2–155.3	155.4–479.03	
NAFLD (%)	34.2	30.8	35	0.8
Znc intake (mg/day)	2.89–9.3	9.4–12.1	12.2–28	
NAFLD (%)	27	33.5	39.5	<0.001
Selenium intake (μg/day)	36.74–88.9	89–118.4	118.5–304	
NAFLD (%)	29	33.5	37.5	0.07
Vitamin A intake (RAE)	52.22–321.6	321.7–534.4	534.5–2478	
NAFLD (%)	39.5	31.5	29	0.02
Vitamin D intake (μg/day)	0.03–1.02	1.03–2.43	2.44–6	
NAFLD (%)	40.5	34	25.5	<0.001
Vitamin E intake (mg/day)	4.04–8.9	9–12.7	12.8–38	
NAFLD (%)	25.5	38	36.5	0.02
Alpha-tocopherol intake (mg/day)	2.44–5.96	5.97–8.23	8.24–23	
NAFLD (%)	33	34.5	32.5	0.91

[a] Variables are ranges for each nutrients.
[b] Obtained from ANOVA.

Table 3
Comparison of antioxidants intake of study participants.

Variable		Cases	Controls	P[b]
Vitamin C (mg/d)	Mean ± SE	123.16 ± 6.50[a]	144.62 ± 4.23	0.01
	Median(R)	121.03 (20.00–479.03)	119.01 (6.00–576.00)	
Zinc (mg/d)	Mean ± SE	11.16 ± 0.18	11.03 ± 0.12	0.59
	Median (R)	11.00 (2.00–28.00)	10.00 (3.00–28.00)	
Selenium (μg/d)	Mean + SE	111.33 ± 2.70	108.50 ± 1.75	0.42
	Median (R)	106.00 (36.00–304.00)	100.00 (33.00–386.00)	
Vitamin A (RAE)	Mean ± SE	419.14 ± 24.37	514.39 ± 15.84	<0.001
	Median (R)	382.00 (52.00–2478.03)	426.00 (39.00–2757.00)	
Vitamin D (μg/d)	Mean ± SE	1.36 ± 0.13	2.26 ± 0.08	<0.001
	Median (R)	1.00 (0.03–6.00)	1.00 (0.05–20.00)	
Vitamin E (mg/d)	Mean ± SE	11.05 ± 0.35	11.75 ± 0.22	0.13
	Median (R)	11.00 (4.04–38.00)	10.00 (2.00–59.02)	
alpha-tocopherol (mg/d)	Mean ± SE	6.99 ± 0.23	7.75 ± 0.15	0.01
	Median(R)	7.00 (2.00–23.00)	6.00 (1.00–37.00)	

[a] Data are presented as mean ± SE (adjusted for age, BMI, MET, energy) and Median (Range).
[b] Obtained from independent sample t-test.

Table 4
Comparison of food groups and macronutrients intake of study participants.[a]

	Cases	Controls	P[b]
Energy (kcal/d)	2335.03 ± 43.20	2194.10 ± 31.16	0.85
Proteins (% of energy)	13.38 ± 0.15	13.64 ± 0.11	0.64
Fats (% of energy)	31.72 ± 0.53	31.87 ± 0.33	0.01
Carbohydrates (% of energy)	57.08 ± 0.53	56.98 ± 0.34	0.05
Dietary fibre (g/d)	39.77 ± 1.64	35.48 ± 0.93	0.06
Whole grains (g/d)	88.69 ± 6.16	86.74 ± 4.72	0.37
Refined grains (g/d)	393.83 ± 14.08	308.45 ± 7.65	0.01
Red meat (g/d)	18.16 ± 1.13	18.86 ± 0.90	0.34
Fruit (g/d)	315.80 ± 15.72	307.58 ± 11.17	0.86
Vegetables (g/d)	245.90 ± 8.70	294.22 ± 7.36	0.001
Nuts, soy and legumes (g/d)	21.35 ± 1.32	22.17 ± 0.99	0.80
Low fat dairy (g/d)	174.12 ± 9.85	230.13 ± 8.82	0.06
High fat dairy (g/d)	241.88 ± 10.85	122.92 ± 5.65	<0.001

[a] All values are means ± standard error (SE).
[b] Obtained from independent sample t-test.

Table 5
Multivariable-adjusted odds ratios and 95% confidence interval (CI) for NAFLD across of tertiles of antioxidants compounds intake.

Variable		T_1	T_2	T_3	p trend[d]
VitC (mg/d)	Crude Model	1.00 (reference)	0.44 (0.33–0.60)	0.53 (0.40–0.72)	<0.001
	Model 1[a]	1.00 (reference)	0.76 (0.50–1.15)	0.93 (0.62–1.40)	0.41
	Model 2[b]	1.00 (reference)	0.69 (0.41–1.18)	0.85 (0.49–1.48)	0.40
	Model 3[c]	1.00 (reference)	0.72 (0.42–1.22)	0.89 (0.51–1.56)	0.46
Zinc (mg/d)	Crude Model	1.00 (reference)	0.50 (0.37–0.67)	0.65 (0.49–0.86)	<0.001
	Model 1	1.00 (reference)	1.10 (0.73–1.66)	1.41 (0.95–2.10)	0.21
	Model 2	1.00 (reference)	1.10 (0.59–2.02)	1.84 (0.85–3.96)	0.18
	Model 3	1.00 (reference)	1.12 (0.60–2.07)	1.85 (0.84–4.07)	0.20
Selenium (μg/d)	Crude Model	1.00 (reference)	0.50 (0.37–0.67)	0.60 (0.45–0.79)	<0.001
	Model 1	1.00 (reference)	1.02 (0.68–1.53)	1.16 (0.78–1.72)	0.73
	Model 2	1.00 (reference)	1.35 (0.75–2.42)	1.58 (0.80–3.11)	0.40
	Model 3	1.00 (reference)	1.37 (0.76–2.49)	1.60 (0.79–3.23)	0.40
Vit A (RAE)	Crude Model	1.00 (reference)	0.46 (0.34–0.62)	0.40 (0.30–0.55)	<0.001
	Model 1	1.00 (reference)	0.65 (0.43–0.97)	0.58 (0.38–0.88)	0.02
	Model 2	1.00 (reference)	0.66 (0.39–1.13)	0.43 (0.24–0.78)	0.02
	Model 3	1.00 (reference)	0.66 (0.39–1.14)	0.43 (0.23–0.78)	0.02
Vit D (μg/d)	Crude Model	1.00 (reference)	0.51 (0.38–0.69)	0.34 (0.24–0.47)	<0.001
	Model 1	1.00 (reference)	0.69 (0.46–1.03)	0.47 (0.31–0.71)	<0.001
	Model 2	1.00 (reference)	0.52 (0.30–0.89)	0.34 (0.19–0.60)	<0.001
	Model 3	1.00 (reference)	0.51 (0.30–0.88)	0.35 (0.20–0.61)	<0.001
Vit E (mg/d)	Crude Model	1.00 (reference)	0.61 (0.46–0.81)	0.57 (0.43–0.76)	<0.001
	Model 1	1.00 (reference)	1.53 (1.01–2.32)	1.50 (0.99–2.29)	0.07
	Model 2	1.00 (reference)	1.41 (0.81–2.48)	1.71 (0.89–3.31)	0.25
	Model 3	1.00 (reference)	1.37 (0.78–2.41)	1.70 (0.88–3.28)	0.28
alpha-tocopherol (mg/d)	Crude Model	1.00 (reference)	0.49 (0.36–0.66)	0.52 (0.39–0.70)	<0.001
	Model 1	1.00 (reference)	0.85 (0.57–1.27)	0.96 (0.64–1.43)	0.73
	Model 2	1.00 (reference)	0.55 (0.30–1.01)	0.74 (0.44–1.27)	0.15
	Model 3	1.00 (reference)	0.40 (0.19–0.84)	0.61 (0 0.34–1.10)	0.04

[a] Model 1: adjusted based on sex and age.
[b] Model 2: adjusted based on sex, age, energy, MET, smoking, BMI and SES.
[c] Model 3: adjusted based on sex, age, energy, MET, smoking, BMI, SES, fiber, fat and vitamin E.
[d] Obtained by the use of tertiles of each nutrient as an ordinal variable in the model.

(OR = 0.43, 95%CI: 0.23–0.78). The same results were shown for vitamin D (crude model: OR = 0.34, 95%CI: 0.24–0.47, after adjustment for confounders: OR = 0.35, 95%CI: 0.20–0.61). Highest intake of vitamins E and C, Zn and Se intake, in comparison to lowest intake exhibited a significant inverse relation with NAFLD in crude model (Vitamin E: OR = 0.57, 95%CI: 0.43–0.76; Vitamin C: OR = 0.53, 95% CI: 0.40–0.72; Zn: OR = 0.65, 95%CI: 0.49–0.86; Se: OR = 0.60, 95%CI: 0.45–0.79); but after adjustment for confounding variables, these associations disappeared. After making adjustment for all confounders, subjects who were in second tertile of alpha-tocopherol intake had 60% lower risk of NAFLD(OR = 0.40; 95%CI: 0.19–0.84).

4. Discussion

Our study examined the relationship between antioxidant compounds intake and physical activity rate with NAFLD. Results indicated that control group have higher physical activity rate than patients and weight in the case group were higher than the other group. More vitamin A and D intakes were also related to lower risk of NAFLD.

While interpreting the findings, it should be considered that NAFLD patients are a heterogeneous group and this disorder has different etiologies in the populations. Life style modification, especially weight reduction is the best therapeutic option for NAFLD. At least a 7–9% weight loss is required to reduce hepatic inflammation [14]. Yoshiike et al. found that obesity is known to be one of the most important risk factors for nonalcoholic steatohepatitis (NASH) and NAFLD [15]. In the study conducted by Capristo et al. it was shown that weight and body fat percentage of patients with NAFLD were more than control group [16]. Durazzo et al. indicated that there is an evidence for the positive effect of weight reduction and physical exercise on controlling NAFLD severity and progression [17]. Although we found a higher intake of certain micronutrients was associated with a lower prevalence of NAFLD, this might simply be due to the fact that thinner people are more likely to follow a healthier lifestyle and the only reason for the lower risk of NAFLD might be their lower weight. We observed that those in the highest category of vitamins A and D had lower prevalence of NAFLD. Comparison of the two groups exhibited that mean consumption of vitamins C, A, D, alpha-tocopherol and vegetables in case group was less and mean consumption of refined grains and high fat dairy in case group was higher than the other group. This study revealed that in both crude and adjusted model for all potential confounders, subjects who were in highest tertile of vitamins A and D intake were associated with reduced risk of NAFLD. Our study was also suggested inverse associations between all antioxidants intake and NAFLD in crude model.

Some previous studies indicated that parameters of oxidative stress increased in NAFLD and endogenous antioxidants including vitamin E and glutathione (GSH) levels reduced in this disorder [18]. Vitamin C is involved in the regulation of circulating and hepatic lipid homeostasis [19]. Also, Han et al. reported a significant lower intake vitamin C in nonalcoholic steatohepatitis (NASH) patients in comparison to the controls [20]. Foster et al. have reported that 20 mg Atorvastatin in combination with vitamins C and E were more effective in odds reduction of hepatic steatosis by 71% in NAFLD patients, after a 4-year of active therapy [21]. On the other hand, other clinical trials in children with NAFLD reported that supplementation with both vitamin C and E was not a better treatment than lifestyle intervention [22,23]. One cross-sectional study reported that intake of vegetables, which are rich in vitamins E, C, A, K and folic acid, was lower in NAFLD patients than the controls [20]. Shi et al. suggested that NAFLD patients in comparison to controls had lower intake of vegetables, vitamin C and A, dietary fiber and PUFA [24]. In a study, Yang et al. suggested that oxidative stress have an important role in NAFLD pathogenesis, since increasing ROS production could result in lipid peroxidation [25]. Unfortunately, we could not measure lipid profile and plasma antioxidants concentration in the present study. Most previous studies revealed that, in subjects that ate higher dietary antioxidant compounds, prevalence of NAFLD was less than the others.

The present study had some strengths; this was the first investigation that examined the relationship between some dietary antioxidants intake and NAFLD in a large sample of adults. Also, it was the first study that examined association between physical activity rate with NAFLD in the Iranian population. Furthermore, we controlled in the statistical analysis for several potential confounding variables. First limitation of the current study is that due to the case–control design of the study, it cannot explain the causal relationships. Second, the plasma antioxidants concentration, glycemic indices, lipid profiles and liver enzymes were not measured in the whole population of the study, since our funds were limit. Third, individuals in the case group were diagnosed as NAFLD patients through blood tests and ultrasonography by a radiology specialist; but we did not have information regarding NAFLD grading. In addition, we did not have enough data to differentiate NASH from simple NAFLD. We suggest that more clinical trials and cohort studies should be conducted to confirm a causal relationship between dietary vitamins or antioxidant compounds intake and NAFLD.

In conclusion, we found that more intakes of vitamins A and D were related to lower risk of NAFLD in this group of Iranian adults. Physical activity rate in cases were less than the controls. Further prospective studies are required to confirm causal association between antioxidant compounds intake and NAFLD.

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Authors' contribution

AL and RG contributed in conception, design, data collection, statistical analysis and drafting of the manuscript. PS contributed in conception and drafting of the manuscript. AH and AS contributed in conception and data collection. RG supervised the study. All authors read and approved the final version of the paper.

Conflicts of interest

Authors declared no potential conflicts of interest.

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