International Journal of Health and Life Sciences (IJHLS) is a scholarly, multidisciplinary, open access, peer-reviewed journal that considers articles on the

- Nutrition and Health
- Epidemiology of Communicable and Noncommunicable Diseases
- Environmental Health Hazards
- Occupational Health
- Public Health Interventions and Health Promotion
- Health Economics
- Other disciplines relevant to Public Health.

International Journal of Health [&] Life Sciences

[ISSN: 2383-4390] [eISSN: 2383-4382]

Free O

Charge



Ashtarian H. et al. Int J Health Life Sci. 2016, volume 2 (number 2): pages 29-34. Classification: Nutrition and Health

You can cite this article as follows:

Pasdar Y., Darbandi M., Bagheri A., Niazi P., Sabani S., Nachvak S. M. Non-Alcoholic Fatty Liver Related to Diet and Body Composition: A Case Control Study. *Int J Health Life Sci.* 2016, 2 (2): 29-34.



Non-Alcoholic Fatty Liver Related to Diet and Body Composition: A Case Control Study

Yahya Pasdar^a, Mitra Darbandi^{a*}, Amir Bagheri^b, Parisa Niazi^b, Soraya Siabani^c, Seyed Mostafa Nachvak^b

^a Research center for environmental determinacies of health, School of Public Health, Kermanshah University of Medical Sciences, Kermanshah, Iran.

^b Department of Nutrition, School of Public Health, Kermanshah University of Medical Sciences, Kermanshah, Iran.

^c Department of Health education and health promotion, School of Public Health, Kermanshah University of Medical Sciences, Kermanshah, Iran

ARTICLE INFO				
Article Type:				
Original Article				

ABSTRACT

Article History: Received: 2016-10-21 Accepted: 2016-12-10 ePublished: 2016-12-10

Keywords: Non-alcoholic Fatty Liver Disease Body Composition Body Mass Index Diet Protein

Corresponding author Fatemeh Yavari Email: <u>mitra.darbandi@yahoo.com</u> Tel: +98-83 38281991

Non-alcoholic fatty liver disease (NAFLD) has a common pathogenic background and shares many risk factors such as hypertension, metabolic syndrome and obesity. This study aimed to determine the relationship between dietary factors and body composition in NAFLD suffering patients. A case-control study designed, 250 patients (n = 125 in each group) were studied. Data was collected using a Body Impedance Analyzer and FFQ questionnaires. The data were analyzed in Stata-11 using the Mann-Whitney test, a t-test, and logistic regression. The Mean BMI was 30.41 ± 5.7 and 26.41 ± 3.8 kg/m2 in the case and control groups, respectively (p = 0.001). In patients with NAFLD, 44.7% showed a BMI over 30 (obese), and in the control group it was 12.5%. The amount of protein intake in the case and control groups was 87.74 ± 52.10 and 97.007 ± 75.55 g/d (P = 0.02), respectively. Fiber intake was 23.12 ± 14.57 g/d in the case group and 25.74 ± 34.71 g/d in the control group (P = 0.3). Vitamin E intake in 69.7% of NAFLD and 74.78% of control group was lower than the RDA recommended level. The study showed that NAFLD subjects have a higher BMI than healthy individuals. Obesity and the low intake of vitamin E, protein, and fiber can be a predicting factor of the incidence or progression of fatty liver.

Introduction

Non-alcoholic fatty liver disease (NAFLD) is characterized by fat accumulation within liver cells when no other etiologies for hepatic fat accumulation (*e.g.*, heavy alcohol consumption) are present (1). NAFLD refers to a wide spectrum of liver disorders ranging from simple steatosis (more than 5% hepatocytes showing fat accumulation) to non-alcoholic steatohepatitis (NASH), which increases the risk of liver cirrhosis and hepatocellular carcinoma (2). Its prevalence is thought to be approximately 20% in the general population and up to 70% in patients with type 2 diabetes mellitus(γ). In the US, an estimated onethird of the population has NAFLD and approximately 2–5% have NASH, with the number of affected people increasing (4). The prevalence

of NAFLD and NASH in Iranians varies from 2.9%-7.1% in the general population (5-7). Due to a lack of evidence to support pharmacological treatment options for NAFLD, diet and physical activity play a key role in NAFLD management (8, 9). Several studies have suggested the associations of NAFLD with obesity, abdominal obesity, hyperglycemia, and other components of metabolic syndrome (4). Excess of body fat, and also abdominal fat, is related to NAFLD (10). The first-line treatment is currently the promotion of gradual weight loss through increased physical activity and reduced energy intake, with the aim of improving liver function tests (LFTs), insulin resistance (IR), fasting glucose, and lipid profiles (8, 9). The role of specific dietary nutrients and the influence of body composition on NAFLD pathogenesis remain uncertain. Nevertheless, results from previous

studies have demonstrated significant а association between NAFLD and diet components. The association between reduced levels of carbohvdrates in diets and NAFLD is demonstrated (11). Moreover, an increased level of fat and sucrose in specific diets has promoted NAFLD in the Torres et al study(12). However, due to the interaction among diet components, it is highly recommended by previous studies to investigate the association between whole diets and disorders to find better relationships and modify lifestyles practically (13). Although literature is emerging, it is not clear what type of diet is more likely to cause fatty liver. Since it is very difficult to reduce and maintain weight loss, it looks more feasible for someone to change the dietary composition of a particular diet as a more realistic method to treat NAFLD without the need of decreasing in Kcal intake.

The current study was conducted to assess the relationship between diet and body composition in NAFLD patients in the Kermanshah province, Iran.

Materials and Methods

This case-control study was conducted in Kermanshah (2015). The patients, people with NAFLD, were selected by convenience sampling from patients whose ultrasound results were positive for fatty liver. The control group was selected by simple random sampling from ultrasound negative reports, that is, without fatty liver. The sample size was determined as 125 for each group (total n = 250) based on previous studies, the prevalence of various factors (in the development of metabolic syndrome), and considering the sample loss. The study protocol was approved by the ethical committee of Kermanshah University of Medical Sciences (No: 92423).

The Evaluation of Food Habits

Food intake was assessed by food frequency questionnaire (FFQ) whose validity and reliability has been confirmed in Iran (14). The FFQ includes a list of 161 foods and their standard amount. Nutritional information obtained through FFQ was analyzed in a specific software program. The software was programmed by visual basic 6.0. Standard values for energy, folate, vitamin A, vitamin E, and calcium were considered based on recommended dietary allowances for different age groups. The recommended value for protein intake is 0.8 g/kg body weight and the recommended fiber intake was considered 25 g daily (15). Energy was calculated considering that each gram of protein, carbohydrate, and fat provides 4, 4, and 9 kilocalories of energy, respectively, and the total energy was obtained from the total energy produced by proteins, carbohydrates, and fats.

The Evaluation of body composition

The individual's body composition was measured by using a body analyzer (Jawon Medical Plus model Avis 333) in terms of weight, height, body fat mass (BFM), the percent of body mass index (BMI), soft lean mass (SLM), body fat (PBF), total body water (TBW), body impedance, body protein and mineral, lean body mass and waist-to-hip ratio (WHR). Height was measured in a standing position, without shoes, using a standiometer, while shoulders, heels, and hips were in contact with the wall, with the accuracy of 1centimeter. Women with a WHR 0.80-0.84 and men with a WHR 0.90-0.99 were classified as overweight, while women with a WHR ≥ 0.85 and men with a WHR \geq 1.00 were classified as obese (16).

Data analysis

Finally, all of the data were coded and entered into Stata-11 and analyzed by descriptive statistics (mean, standard deviation, and percentage) and analytical statistics (t-test, Mann-Whitney, and logistic regression) (a p-value less 0.05 was considered significant).

Results

Of the total of 250 participants, 220 that provided full data entered analysis (a response rate of 88.8%). The case group consisted of 103 NAFLD patients, with steatohepatitis confirmed through ultrasound, and the control group included 119 healthy people, with a normal liver ultrasound. The youngest participant was 30 years old and the oldest was 65 years old. Mean weight was 82.1 \pm 16.9 kg in participants with fatty livers, and 70.9 \pm 12.2 kg in participants without fatty livers. The mean body mass index was significantly higher in participants with NAFLD compared to the control group (P = 0.001). The WHR was higher in participants with NAFLD compared to the control group (P = 0.001), and there were significant differences between the two groups in other components of body composition, including MBF, LBM, and TBW (Table 1).

The body mass index (BMI) was greater than 30 (obese) in 44.66% of participants in the NAFLD group and 12.45% of those in the control group, with a significant difference between the two groups (P = 0.001) (Table 2).

A logistic regression model was used to determine the independent effects of WHR and BMI on NAFLD, and after adjusting for other variables, WHR and BMI were found to be significantly related to NAFLD. WHR was higher than the normal range in 55.5% of participants with fatty livers. In other words, the odds of having fatty liver in people with a WHR higher than normal (greater than 0.85 in women and greater than 1 in men) was 2.7 times higher compared to people with a normal WHR. Moreover, BMI was higher than 30 in 74% of people with fatty livers. In other words, the odds of having fatty liver in people with a BMI > 30 was 2.4 time higher compared to people with a normal BMI, and the odds increased to 12.1 times after adjusting for confounding variables (Table 3).

The daily calorie intake was $2329.15 \pm 13.19.38$ Kcal in the case group and 2593.71 ± 1944.25 Kcal in the control groups. The daily protein intake was 87.74 ± 52.1 grams in patients and 97.007 ± 75.55 grams in healthy group, which was significantly higher in the healthy group (P = 0.02). Fiber intake was 23.12 ± 14.57 grams/day in the NAFLD group and 25.74 ± 34.71 grams/day in the healthy group (P = 0.3) (Table 4).

Compared to the Recommended Daily Allowance (RDA), protein intake was less than the RDA in 39 (39.39%) participants from the case group and 31 (26.96%) participants from the control group. Vitamin E intake was less than the RDA in 74.78% of participants with fatty livers and 69.70% of participants without fatty livers.

 Table 1. Components of Body Composition in the two

Body	Mear	Р-	
Composition	NAFLD (-)	NAFLD (+)	value
Weight (kg)	82.1±16.9	70.9±12.2	0.01
BMI(kg/m2)	30.41 ± 5.7	26.41±3.8	0.001
WHR	0.91 ± 0.64	0.77 ± 0.07	0.001
MBF(kg)	27.9 ± 8.83	21.92 ± 6.57	0.03
PBF (%)	33.8±6.86	30.73 ± 6.56	0.001
LBM(kg)	54.2 ± 11.9	49.01 ± 9.23	0.02
SLM(kg)	49.53±11.1	44.97 ± 8.6	0.04
TBW(kg)	39.02 ± 8.61	35.29 ± 6.64	0.001

 Table 2.
 Comparison of body mass index in the two

	groups N (%)			
	Normal Weight	Over Weight	Obese	Total
NAFLD (+)	11(10.68)	46(44.66)	46(44.66)	103(100)
NAFLD (-)	44(36.07)	59(49.58)	16(12.45)	119(100)
Total	55(24.77)	105(47.30)	62(27.93)	222(100)

 Table 3. The role WHR and BMI in terms of having

 fatty liver

Variable		Fatty Crude		adjusted	
		Liver/N			
		Prevalence	OR	OR	
			(95%CI)	(95%CI)*	
WHR	No	25/79	1		
		(31.5%)	T		
	Yes	76/137	2.7	-	
		(55.5%)	(1.5-4.8)		
BMI	18.5-	11/55	1	1	
	24.9	(20%)	1	1	
	25-29.9	46/105	1.1	4.2	
		(44%)	(0.3-1.9)	(1.7-10.4)	
	>30	46/62	2.4	12.1	
		(74%)	(1.5-3.3)	(3.9-37.1)	
* Confidence Interval					

* Confidence Interval

Discussion

The present study was conducted with the aim to determine the relationship between the body composition and NAFLD, and it showed that the odds of fatty liver were higher in people with higher than normal WHR and BMI compared to those with normal or below normal WHR and BMI.

Table 4. The mean of daily food intakes in case and control groups					
Food intakes					
roou makes	NAFLD (+)	NAFLD (-)	Total	- p-value*	
Energy (kcal)	2329.15±1319.38	2593.71±1944.25	2470.32±1685.69	0.5	
Protein (gr)	87.74±52.10	97.007±74.55	92.72±65.76	0.02	
Fat (gr)	93.44±60.51	107.32±87.12	100.90±76.13	0.2	
Carbohydrate (gr)	285.34±157.44	309.46±234.09	298.30±202.31	0.8	
Fiber (gr)	23.12±14.57	25.74±34.71	24.53±27.28	0.3	
Vitamin A (µgr)	992.53±811.18	1011.74±940.84	1002.85±881.24	0.8	
Vitamin E (Mgr)	14.86±17.25	17.33±35.02	16.19±28.19	0.04	

* By using Mann-Whitney test

In other words, high WHR and BMI are among strong predictors of fatty liver. A study conducted by Radmard et al. also proposed WHR and BMI as factors predicting NAFLD (). Other similar studies also reported a positive relationship between BMI and NAFLD (1^{9} , 1^{1}). In a study, liver enzymes, especially ALT, were twice the normal laboratory reference range in overweight people older than 50 years with a BMI of 28 kg/m² or higher (1°).

The present study results showed that diet is one of the important and influential factors for the incidence of fatty liver. Analysis of participants' food intake showed that intakes of protein, fiber, and vitamin E were higher in the control group compared to the NAFLD group. Previous studies have shown that the intake of vitamins E and C can dramatically improve the condition of NAFLD patients and prevent progress of this disease (7). Several clinical studies have investigated the effect of nutritional proteins on NAFLD, and some have reported reduced or improved NAFLD due to intake of soy protein. Thus, there is still no definitive evidence about the effect of nutritional proteins on NAFLD (22, 23). A number of interventional studies have reported that weight loss resulting from diet is able to restore liver enzymes and also prevent and control fatty liver. Due to the resulting weight loss and balance in food intakes. diet-based interventions can improve liver enzymes and tissue $(\gamma \gamma)$. Diet has a key role in the management of NAFLD, and quality and quantity of diet are highly effective in the onset and severity of fatty liver (7). Diet, physical activity, and weight control can significantly affect the clinical picture of NAFLD, since most patients with NAFLD have excess body weight and other cardiometabolic risk factors, such as dyslipidemia, hypertension, and diabetes (22, 25). Although

promising pharmacological treatments are emerging, only a significant and stable weight loss is the basis of any treatment plan for patients suffering from NAFLD (26).

Conclusions

In the present study, the odds of NAFLD increased with increasing WHR and BMI. The body composition, especially WHR and BMI as well as diet are strong predictors of NAFLD. Adequately consumed protein, fiber, and vitamin E can be effective in preventing and improving NAFLD. Weight loss is the most effective therapy for NAFLD. Lifestyle changes through improved diet and exercise is the basis of treatment programs for NAFLD patients. Thus, with the right quality and quantity of diet, the body composition can be maintained within the normal range, and the incidence of NAFLD can be significantly prevented.

Acknowledgement

The authors gratefully acknowledge the Research Council of Kermanshah University of Medical Sciences (Grant Number: 92423) for the financial support and Research Center for Environmental Determinants of Health (RCEDH) in School of Health.

References

- [1] Shaker M, Tabbaa A, Albeldawi M, Alkhouri N. Liver transplantation for nonalcoholic fatty liver disease: new challenges and new opportunities. World Journal of Gastroenterology: WJG. 2014;20(18):5320.
- [2] Sofi F, Casini A. Mediterranean diet and nonalcoholic fatty liver disease: new therapeutic option around the corner. World J Gastroenterol. 2014;20(23):7339-46.

- 3. Sattar N, Forrest E, Preiss D. Non-alcoholic fatty liver disease. bmj. 2014;349(8).
- 4. Vernon G, Baranova A, Younossi Z. Systematic review: the epidemiology and natural history of non-alcoholic fatty liver disease and non-alcoholic steatohepatitis in adults. Alimentary pharmacology & therapeutics. 2011;34(3):274-85.
- 5. Alavian SM, Mohammad-Alizadeh AH, Esna-Ashari F, Ardalan G, Hajarizadeh B. Non-alcoholic fatty liver disease prevalence among school-aged children and adolescents in Iran and its association with biochemical and anthropometric measures. Liver International. 2009;29(2):159-63.
- 6. Rogha M, Najafi N, Azari A, Kaji M, Pourmoghaddas Z, Rajabi F, et al. Non-alcoholic steatohepatitis in a sample of iranian adult population: age is a risk factor. International journal of preventive medicine. 2011;2(1).
- 7. Sohrabpour AA, Rezvan H, Amini-Kafiabad S, Dayhim M, Merat S, Pourshams A. Prevalence of nonalcoholic steatohepatitis in Iran: A population based study. Middle East Journal of Digestive Diseases (MEJDD). 2010;2(1):14-9.
- 8. Chalasani N, Younossi Z, Lavine JE, Diehl AM, Brunt EM, Cusi K, et al. The diagnosis and management of non-alcoholic fatty liver disease: Practice Guideline by the American Association for the Study of Liver Diseases, American College of Gastroenterology, and the American Gastroenterological Association. Hepatology. 2012;55(6):2005-23.
- Nascimbeni F, Pais R, Bellentani S, Day CP, Ratziu V, Loria P, et al. From NAFLD in clinical practice to answers from guidelines. Journal of hepatology. 2013;59(4):859-71.
- 10. Bhatt SP, Misra A, Nigam P, Guleria R, Pasha MQ. Phenotype, Body Composition, and Prediction Equations (Indian Fatty Liver Index) for Non-Alcoholic Fatty Liver Disease in Non-Diabetic Asian Indians: A Case-Control Study. PloS one. 2015;10(11):e0142260.
- 11. Gonzalez C, de Ledinghen V, Vergniol J, Foucher J, Le Bail B, Carlier S, et al. Hepatic steatosis, carbohydrate intake, and food quotient in patients with NAFLD. International journal of endocrinology. 2013;2013.
- 12. Torres-Villalobos G, Hamdan-Pérez N, Tovar AR, Ordaz-Nava G, Martínez-Benítez B, Torre-Villalvazo I, et al. Combined high-fat diet and sustained high sucrose consumption promotes NAFLD in a murine model. Ann Hepatol. 2015;14:540-6.
- 13. Willet W. Nutritional epidemiology: issues and challenges. International Journal of Epidemiology. 1987;16(2):312-7.

- 14. Mirmiran P, Esfahani FH, Mehrabi Y, Hedayati M, Azizi F. Reliability and relative validity of an FFQ for nutrients in the Tehran Lipid and Glucose Study. Public health nutrition. 2010;13(05):654-62.
- 15. Amirifar A, Saberi M. Modern Human Kraus.second edition. Tehran: FE, editor: Book Publishing Mir; 2006.
- 16. Dalton M, Cameron A, Zimmet P, Shaw J, Jolley D, Dunstan D, et al. Waist circumference, waist-hip ratio and body mass index and their correlation with cardiovascular disease risk factors in Australian adults. Journal of internal medicine. 2003;254(6):555-63.
- 17. Radmard AR, Rahmanian MS, Abrishami A, Yoonessi A, Kooraki S, Dadgostar M, et al. Assessment of Abdominal Fat Distribution in Non-Alcoholic Fatty Liver Disease by Magnetic Resonance Imaging: a Population-based Study. Archives of Iranian Medicine. 2016 Oct;19(10):693-9. PubMed PMID: WOS:000386933000003.
- 18. Abenavoli L, Di Renzo L, De Lorenzo A. Body Composition and Non-alcoholic Fatty Liver Disease. Journal of Lifestyle Medicine. 2016;6(1):47.
- 19.Zelber-Sagi S, Shoham D, Zvibel I, Abu-Abeid S, Shibolet O, Fishman S. Predictors for advanced fibrosis in morbidly obese non-alcoholic fatty liver patients. World Journal of Hepatology. 2017;9(2):91.
- 20. Ratziu V, Giral P, Charlotte F, Bruckert E, Thibault V, Theodorou I, et al. Liver fibrosis in overweight patients. Gastroenterology. 2000;118(6):1117-23.
- 21. Miller ER, Pastor-Barriuso R, Dalal D, Riemersma RA, Appel LJ, Guallar E. Meta-analysis: high-dosage vitamin E supplementation may increase all-cause mortality. Annals of internal medicine. 2005;142(1):37-46.
- 22. McCarthy EM, Rinella ME. The role of diet and nutrient composition in nonalcoholic fatty liver disease. Journal of the Academy of Nutrition and Dietetics. 2012;112(3):401-9.
- 23. Tovar AR, Torre-Villalvazo I, Ochoa M, Elías AL, Ortíz V, Aguilar-Salinas CA, et al. Soy protein reduces hepatic lipotoxicity in hyperinsulinemic obese Zucker fa/fa rats. Journal of lipid research. 2005;46(9):1823-32.
- 24. Promrat K, Kleiner DE, Niemeier HM, Jackvony E, Kearns M, Wands JR, et al. Randomized controlled trial testing the effects of weight loss on nonalcoholic steatohepatitis. Hepatology. 2010;51(1):121-9.
- 25. Zivkovic AM, German JB, Sanyal AJ. Comparative review of diets for the metabolic syndrome: implications for nonalcoholic fatty liver disease. The

American journal of clinical nutrition. 2007;86(2):285-300.

26. Loria P, Adinolfi L, Bellentani S, Bugianesi E, Grieco A, Fargion S, et al. Practice guidelines for the diagnosis and management of nonalcoholic fatty liver disease: A decalogue from the Italian Association for the Study of the Liver (AISF) Expert Committee. Digestive and Liver Disease. 2010;42(4):272-82.