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RESEARCH ARTICLE **OPEN ACCESS**

Comparison of Dietary Inflammatory Index, Physical Activity Level, and Dietary Macronutrient Intake between Newly Diagnosed Atherosclerotic Patients and Healthy Controls

Hanieh Abdi[1,2], Zohre Karamian[1,2], Kazem Hajiloo[2,3] and Amin Sharifi[2,3,*]

[1]*Student Research Committee, Hamadan University of Medical Sciences, Hamadan, Iran*
[2]*Department of Nutrition and Food Hygiene, Hamadan University of Medical Sciences, Hamadan, Iran*
[3]*Nutrition Health Research Center, Institute of Health Sciences and Technology, Hamadan University of Medical Sciences, Hamadan, Iran*

Abstract:

Introduction: Atherosclerotic cardiovascular disease (ASCVD) is a low-grade inflammation of the vessels that underlies cardiovascular disease (CVD). Dietary factors significantly influence the development of ASCVD, as do traditional CVD risk factors. The Dietary Inflammatory Index (DII) is a tool to evaluate diet-inflammation relationships and their impact on CVD incidence. We aimed to compare the DII, dietary macronutrient intake, and body mass index (BMI) between patients with ASCVD and healthy controls in Hamadan City, Iran, in 2024.

Methods: This case-control study included a total of 103 newly diagnosed ASCVD patients and an equal number of healthy controls. Dietary intake was assessed using a validated food frequency questionnaire. DII was calculated based on 38 food items. Physical activity levels (PAL) were evaluated through two researcher-made questions. DII, PAL, and dietary factors were compared between patients and controls.

Results: ASCVD patients had higher DII and higher intakes of total fat ($p<0.001$), saturated fat ($p=0.032$), polyunsaturated fatty acids (PUFA) ($p=0.023$), and monounsaturated fatty acids (MUFA) ($p<0.001$) than controls. Each unit increase in the DII was associated with a 16.8% increase in ASCVD risk. Higher PAL was associated with a 50.2% reduced risk of ASCVD.

Conclusion: Based on the findings, diets high in proinflammatory foods, higher fat intake, and sedentary behavior are associated with an increased risk of ASCVD.

Keywords: Atherosclerosis, Diet, Dietary inflammatory index, Exercise, Nutrients, Nutrition assessment, Cardiovascular diseases.

*Address correspondence to this author at the Department of Nutrition and Food Hygiene, Hamadan University of Medical Sciences, Hamadan, Iran, and Nutrition Health Research Center, Institute of Health Sciences and Technology, Hamadan University of Medical Sciences, Hamadan, Iran; E-mail: a.sharifi1983@gmail.com

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1. INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of mortality worldwide [1]. Low-grade inflammation of the endothelium is a primary underlying pathologic basis of atherosclerotic cardiovascular disease (ASCVD) [2], where reactive oxygen species and inflammatory cytokines play a substantial role [3]. This inflammatory activity may lead to endothelial dysfunction [4].

Certain dietary factors have been suggested as risk factors for atheroma development, either directly or indirectly through other risk factors, such as plasma lipids and glucose [5]. Healthy dietary patterns, combined with portion control and energy balance, have been proposed to reduce excess weight gain and CVD risk [6, 7].

A substantial body of research demonstrates associations between dietary patterns and inflammation regulation *via* proinflammatory cytokines, suggesting a modulatory role in atherogenesis [8, 9]. Given the significant role of inflammation in ASCVD and its potential modulation through diet, categorizing an individual's diet based on its inflammatory properties may provide valuable insights into the relationships among diet, inflammation, and CVD [10]. The Dietary Inflammatory Index (DII) is a validated tool for assessing the inflammatory potential of the diet and its association with CVD incidence [11]. Studies indicate that individuals with higher (more proinflammatory) DII scores have a higher incidence of CVD and worse cardiometabolic risk profiles, with associations observed for coronary heart disease, stroke, and elevated inflammatory biomarkers [12-17]. This supports the concept that diet-driven systemic inflammation contributes to ASCVD risk.

Some evidence further suggests that DII may be linked to CVD and CVD-related mortality [10, 12, 18-22]. To strengthen this evidence, we conducted a case-control study in Hamadan City, Iran, in 2024, comparing DII and physical activity level (PAL) between individuals with newly diagnosed ASCVD and age- and sex-matched healthy controls, and evaluating the association between DII and ASCVD. We hypothesized that patients with ASCVD would exhibit higher DII scores and lower PAL compared with healthy controls.

2. MATERIALS AND METHODS

This case-control study included 103 newly diagnosed (within the recent three months) ASCVD patients in Hamadan City (Iran) as the case group and 103 healthy controls. The controls were selected among friends or neighbors of each ASCVD patient, ensuring gender matching and a similar age range (±5 years).

The inclusion criteria for the case group required a definitive diagnosis of ASCVD [23, 24] by cardiologists within the past three months, along with non-adherence to a prescribed diet or significant changes in dietary habits during the three months preceding the study.

All participants completed a 168-item food frequency questionnaire (FFQ) [25]. Calorie, carbohydrate, protein, total fat, saturated fatty acids (SFA), monounsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA), and dietary fiber intake were estimated using ShaFA software [26] based on the FFQ data.

The DII for each participant was calculated using the methodology proposed by Shivappa *et al*. [11]. Due to the absence of dietary intake data for specific components, only 38 of the original 45 food parameters were included in the computation. Specifically, eugenol, flavan-3-ol, flavones, flavonones, flavonols, anthocyanidins, and isoflavones were not included.

Physical activity was assessed at two levels using two researcher-made questions:

- **Level 1:** No exercise or regular walking.
- **Level 2:** One or more sessions of exercise or regular walking per week.

2.1. Statistical Analysis

The normality of quantitative data was assessed using the Kolmogorov-Smirnov test. Based on the results, either the independent t-test or the Mann-Whitney U test was used to compare the two groups. Qualitative variables were compared between groups using the chi-square test. Additionally, logistic regression analysis was performed to evaluate the association between predictor variables and the occurrence of the outcome. Statistical analyses were conducted using SPSS software (IBM Corp., Armonk, New York, USA). A two-sided significance level of 0.05 was considered statistically significant.

2.2. Ethics Approval and Consent to Participate

The study was conducted after receiving approval from the Ethics Committee at Hamadan University of Medical Sciences (ethics code: IR.UMSHA.REC.1401.790). Informed written consent was obtained from all participants prior to the study.

3. RESULTS

A total of 103 newly diagnosed ASCVD patients and 103 healthy controls were included in the study. No significant differences were observed between the two groups in dietary energy, protein, or cholesterol intake. However, ASCVD patients had higher dietary intakes of total fat, saturated fatty acids (SFA), polyunsaturated fatty acids (PUFA), and monounsaturated fatty acids (MUFA), as well as higher Dietary Inflammatory Index (DII) scores compared with the control group. In contrast, dietary intake of carbohydrates and fiber was higher in the control group (Table **1**).

In the multiple logistic regression model, which included dietary intake of protein, carbohydrates, fiber, and total fat, dietary total fat ($p<0.001$) and fiber ($p=0.005$) were significant predictors of ASCVD incidence (Table **2**).

Logistic regression analysis revealed that a one-unit increase in the Dietary Inflammatory Index (DII) was associated with a 16.8% increase in the risk of developing ASCVD (Odds Ratio [OR]: 1.167; 95% Confidence Interval [CI]: 1.008–1.352, $p=0.039$).

Table 1. Descriptive statistics and comparisons of age, body mass index (BMI), dietary inflammatory index (DII), energy intake, and the consumption of macronutrients and fatty acids between atherosclerotic patients and healthy control groups.

-		Men		Women		Total	
		Controls (n=52)	Cases (n=53)	Controls (n=51)	Cases (n=50)	Controls (n=103)	Cases (n=103)
Age (years)	Mean	60.7	62.6	61.6	64.7	61.1	63.7
	SD	11.9	12.1	12.2	12.2	12.0	11.6
	Median	60.0	62.0	59.0	64.0	59.0	63.0
	IQR	13.0	11.0	15.0	14.0	14.0	14.0
	Min	34.0	30.0	44.0	44.0	34.0	30.0
	Max	91.0	90.0	102.0	90.0	102.0	90.0
	p	0.398		0.179		0.124	
Body mass index (kg/m^2)	Mean	25.5	25.8	27.0	26.9	26.2	26.3
	SD	2.8	4.4	4.0	5.1	3.5	4.8
	Median	25.8	25.3	27.2	26.3	26.1	26.0
	IQR	3.8	5.1	4.4	6.3	4.6	6.1
	Min	19.6	18.7	16.8	17.8	16.8	17.8
	Max	33.5	40.1	34.1	41.6	30.1	41.6
	p	0.677		0.894		0.888	
Energy (Calorie)	Mean	2176.6	2237.4	1777.9	1764.8	1979.2	2007.9
	SD	833.6	597.1	655.9	485.0	773.6	592.5
	Median	2135.3	2275.1	1583.4	1778.1	1721.2	2031.6
	IQR	1373.1	611.0	656.9	680.3	1066.3	797.6
	Min	691.7	979.1	883.8	770.1	691.7	770.1
	Max	3970.7	3526.5	3587.7	2806.8	3970.7	3526.5
	p	0.668		0.909		0.765	
Carbohydrate (g)	Mean	288.1	244.8	222.6	202.4	255.7	224.2
	SD	122.3	62.7	83.8	61.0	109.6	65.1
	Median	295.8	255.4	204.5	203.3	228.9	231.6
	IQR	190.6	68.3	76.1	87.6	162.5	89.1
	Min	78.9	116.3	89.3	83.9	78.9	83.9
	Max	553.7	375.2	443.9	344.3	553.7	375.2
	p	0.024		0.169		0.013	
Protein (g)	Mean	77.7	75.3	62.2	56.8	70.0	66.3
	SD	31.8	19.9	26.1	14.6	30.0	19.7
	Median	73.8	74.4	53.5	57.1	61.8	62.6
	IQR	52.5	27.7	34.1	20.2	40.6	27.1
	Min	28.0	35.6	26.6	31.9	26.6	31.9
	Max	147.5	124.1	135.9	91.2	147.5	124.1
	p	0.638		0.208		0.297	
Total fat (g)	Mean	86.5	110.8	76.2	84.7	81.4	98.1
	SD	36.9	36.5	33.9	26.0	35.6	34.3
	Median	77.8	110.4	65.4	81.8	73.4	99.1
	IQR	47.9	48.2	31.8	43.7	41.8	45.9
	Min	31.2	38.8	30.3	31.6	30.3	31.6
	Max	185.2	192.5	176.6	147.6	185.2	192.5
	p	0.001		0.158		<0.001	
Saturated fat (g)	Mean	25.9	34.2	25.0	25.1	25.5	29.8
	SD	13.8	15.1	15.1	11.4	14.4	14.2
	Median	21.6	31.7	19.7	23.6	20.5	26.7
	IQR	15.0	19.2	13.9	12.9	14.6	18.7
	Min	6.8	10.7	9.2	7.8	6.8	7.8
	Max	74.2	88.6	88.5	55.0	88.5	88.6
	p	0.004		0.975		0.032	

(Table 1) contd.....

-		Men		Women		Total	
		Controls (n=52)	Cases (n=53)	Controls (n=51)	Cases (n=50)	Controls (n=103)	Cases (n=103)
Polyunsaturated fatty acids (g)	Mean	20.1	23.7	15.6	18.0	17.9	20.9
	SD	11.0	9.3	9.6	6.1	10.5	8.4
	Median	16.7	23.2	13.2	18.1	14.1	20.7
	IQR	12.9	11.0	9.7	10.0	10.6	11.4
	Min	7.6	7.5	2.8	7.0	2.8	7.0
	Max	57.7	54.8	53.0	30.7	57.7	54.8
	p	0.075		0.141		0.023	
Monounsaturated fatty acids (g)	Mean	32.9	44.8	29.3	35.4	31.1	40.2
	SD	16.2	16.6	15.7	13.1	16.0	15.7
	Median	30.0	44.0	26.9	31.5	27.3	38.2
	IQR	16.5	18.9	15.3	21.3	15.5	23.1
	Min	14.3	14.1	4.8	13.9	4.8	13.9
	Max	87.8	93.6	85.0	63.9	87.8	93.6
	p	<0.001		0.035		<0.001	
Cholesterol (mg)	Mean	295.3	319.0	237.9	230.2	266.9	275.9
	SD	125.3	107.3	91.7	67.5	113.1	100.2
	Median	280.3	309.1	223.6	217.9	252.5	252.5
	IQR	148.4	136.8	126.9	70.4	139.9	127.2
	Min	60.4	149.5	66.7	106.0	60.4	106.0
	Max	613.4	557.8	471.8	414.4	613.4	557.8
	p	0.3		0.635		0.544	
Dietary fiber (g)	Mean	26.1	20.7	21.1	18.0	23.6	19.4
	SD	12.2	6.3	11.4	6.3	12.0	6.4
	Median	25.9	20.2	15.8	16.8	18.3	18.6
	IQR	21.1	7.9	14.1	9.7	19.1	9.5
	Min	7.4	10.1	6.3	6.8	6.3	6.8
	Max	48.7	34.8	56.5	32.4	56.5	34.8
	p	0.006		0.098		0.002	
Dietary inflammatory index	Mean	-0.195	0.650	1.161	1.429	0.476	1.028
	SD	2.255	1.384	2.101	1.328	2.273	1.406
	Median	-1.088	0.713	1.976	1.505	1.263	1.023
	IQR	4.286	1.799	3.643	2.006	4.371	1.971
	Min	-3.236	-2.049	-2.489	-2.069	-3.236	-2.069
	Max	3.842	4.197	4.470	3.959	4.470	4.197
	p	0.022		0.445		0.037	

Note: SD: Standard deviation; IQR: Inter-quartile range.

Table 2. Logistic regression model for atherosclerosis risk adjusted for dietary protein, carbohydrate, fiber, and total fat.

Variables	OR	SE	95% CI		*P*
Protein (g)	0.980	0.017	0.947	1.015	0.261
Carbohydrate (g)	0.996	0.004	0.987	1.004	0.306
Fiber (g)	0.889	0.037	0.819	0.966	0.005
Total Fats (g)	1.057	0.011	1.037	1.078	<0.001
Constant	0.862	0.437	0.319	2.328	0.770

Table 3. The number and percentage of cases and controls based on the physical activity level (PAL). There was a significant difference between the two groups (p = 0.028).

	Controls	Cases
PAL 1 (No exercise or regular walking)	21 (20.4%)	35 (34.0%)
PAL 2 (One or more times of exercise or regular walking per week)	82 (79.6%)	68 (66.0%)

There was a significant difference in physical activity level (PAL) between the two groups (Table **3**). Compared with activity level one (no exercise or regular walking), activity level two (one or more sessions of exercise or regular walking per week) was associated with a 50.2% reduction in the risk of ASCVD incidence (OR: 0.498; 95% CI: 0.265–0.934, p=0.03).

Participants were divided into two subgroups based on their DII scores (≤1 and >1). Logistic regression analysis within these subgroups showed that higher PAL was associated with a reduced risk of ASCVD only in the group with DII ≤1 (OR: 0.152; 95% CI: 0.032–0.724, p=0.018), whereas in the group with DII >1, higher PAL had no significant effect on ASCVD prevention (OR: 0.652; 95% CI: 0.298–1.426, p=0.284).

4. DISCUSSION

In this study, dietary intakes of total fat, saturated fatty acids (SFA), polyunsaturated fatty acids (PUFA), monounsaturated fatty acids (MUFA), and Dietary Inflammatory Index (DII) scores were higher in ASCVD patients compared with the control group. In contrast, dietary intake of carbohydrates and fiber was higher in the control group. Additionally, higher physical activity level (PAL) was associated with a lower risk of ASCVD development.

The incidence of non-communicable diseases is significantly influenced by diet [27] and diet-related metabolites [28]. Among the modifiable risk factors for CVD (*i.e.*, metabolic, environmental, and behavioral), dietary factors rank highest. It has been estimated that dietary risk factors contribute to 53% of CVD deaths and 58% of CVD-related disabilities [29].

Numerous dietary patterns have been proposed to reduce CVD events, including diets low in total or saturated fats [30, 31]. For example, a meta-analysis found that Mediterranean diet and low-fat dietary programs probably reduce the risk of mortality and myocardial infarction [32]. In our study, dietary intake of total fat, SFA, PUFA, and MUFA was higher in patients with ASCVD than in the control group, indicating that higher fat intake, even of unsaturated types, is associated with a higher risk of developing ASCVD. This suggests that total dietary fats may act as an independent risk factor for CVD regardless of fatty acid type. However, some studies suggest that substituting SFA with PUFA and MUFA may decrease CVD risk [33-36].

Conversely, increased consumption of whole grains, nuts, seeds, beans, legumes, fruits, and vegetables (which are fiber-rich sources), as well as fish, lean meats, and low-fat dairy products, contributes to lower CVD mortality [37-49].

Currently, it is believed that dietary cholesterol has a limited impact on serum low-density lipoprotein (LDL) cholesterol levels and the risk of CVD compared with other dietary and lifestyle factors. A recent study involving over 17,700 participants from 50 countries supported this perspective, showing no significant association between egg consumption and increased blood lipids, blood pressure, or CVD-related mortality [50]. Similarly, in our study, no significant differences were observed between the two groups in terms of cholesterol intake, supporting the view that dietary cholesterol may not significantly affect CVD incidence.

In the present study, foods higher in carbohydrates and fiber demonstrated a protective effect against ASCVD, consistent with previous studies highlighting the benefits of fiber-rich foods, such as whole grains, vegetables, and fruits [51-55]. This protective effect may be mediated through alterations in the gut microbiota, promoting fiber fermentation and short-chain fatty acid (SCFA) production [56, 57].

The association of DII with CVD incidence has been examined in several studies. Analysis of data from the National Health and Nutrition Examination Survey (NHANES) revealed a direct association between the 28-item DII and the risk of ASCVD development, which was more pronounced in women [58]. However, dietary intake in this study was based on a single 24-hour dietary recall, which may not accurately reflect dietary fluctuations. A similar finding was observed in the Prevención con Dieta Mediterránea (PREDIMED) study, where CVD risk increased across DII quartiles [20]. Furthermore, following over 18,000 Spanish university graduates for 8.9 years, the Seguimiento Universidad de Navarra (SUN) Cohort study found that participants in the highest DII quartile were at approximately twice the risk of CVD incidence compared with those in the lowest quartile [21]. A meta-analysis also suggested that each unit increase in the DII increases CVD risk by 8% [19]. Conversely, studies conducted in Iran [59] and Australia [23] found no association between DII and CVD.

In the present study, each unit increase in the DII was associated with a 16.8% increase in the risk of developing ASCVD. Moreover, at higher DII levels, even higher physical activity levels (PAL) did not prevent the incidence of ASCVD.

Many studies indicate that adequate physical activity level (PAL) can prevent or delay CVD. PAL significantly lowers CVD risk by reducing serum triglycerides, increasing serum high-density lipoprotein (HDL) cholesterol, and decreasing coronary artery calcium [60, 61]. In our study, higher PAL was associated with a lower risk of ASCVD, and this protective effect was more pronounced in individuals with lower Dietary Inflammatory Index (DII) scores.

Certain variables, including carbohydrate, total fat, saturated fat, fiber intake, and DII, did not show statistically significant differences within the female subgroup, despite reaching significance in the male subgroup. This discrepancy may reflect genuine gender-based physiological differences or may be attributable to limited statistical power in the subgroup analysis.

The limitations of this study include the lack of use of validated PAL questionnaires, such as the International Physical Activity Questionnaire (IPAQ), due to the potential for a low participation rate, as anthropometric assessment and completion of the FFQ were time-consuming and tedious. Another limitation is recall bias, which is inherent in retrospective questionnaires such as the FFQ. Additionally, the number of participants, particularly in subgroup analyses, was relatively small.

CONCLUSION

Our findings support the notion that patients with ASCVD consumed higher amounts of fat and exhibited a proinflammatory dietary pattern. In contrast, the control group showed higher intakes of carbohydrates and fiber. Furthermore, increased PAL was associated with a decreased risk of ASCVD. Future studies with larger sample sizes and different ethnicities are required.

AUTHORS' CONTRIBUTIONS

The authors confirm contribution to the paper as follows: Z.K. and K.H.: Contributed to the design of the study, acquisition of the data, revision of the manuscript, and provided final approval of the version to be submitted. They also agreed to be accountable for all aspects of the work; H.A.: Contributed to the design of the study, acquisition of the data, and drafting of the manuscript; H.A.: Provided final approval of the version to be submitted and agreed to be accountable for all aspects of the work; A.Sh.: Contributed to the literature search, conception and design of the study, supervision and execution of the study, acquisition, statistical analysis, and interpretation of the data, and drafting of the manuscript; A. Sh.: Provided final approval of the version to be submitted and agreed to be accountable for all aspects of the work.

LIST OF ABBREVIATIONS

ASCVD	=	Atherosclerotic Cardiovascular Disease
DII	=	Dietary Inflammatory Index
LDL	=	Low-Density Lipoprotein
FFQ	=	Food Frequency Questionnaire
SFA	=	Saturated Fatty Acids
MUFA	=	Monounsaturated Fatty Acids
PUFA	=	Polyunsaturated Fatty Acids

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This project was conducted in accordance with the approval of the Ethics Committee of Hamadan University of Medical Sciences, Iran [ethics code: IR.UMSHA.REC.1401.790].

HUMAN AND ANIMAL RIGHTS

All procedures performed in studies involving human participants were in accordance with the ethical standards of institutional and/or research committee and with the 1975 Declaration of Helsinki, as revised in 2013.

CONSENT FOR PUBLICATION

Informed written consent was obtained from all volunteers before the study.

STANDARDS OF REPORTING

STROBE guidelines were followed.

AVAILABILITY OF DATA AND MATERIALS

The data of current study are available from corresponding author, [A.S.], on a reasonable request.

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CONFLICT OF INTERESTS

The authors declare no conflict of interest, financial or otherwise.

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