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Healthy eating behaviors may be associated with lower risk of premature coronary artery disease: A multi-center case-control study

Ehsan Shirvani[1], Noushin Mohammadifard[2*], Mahshid Keshavarz[3*], Fereshteh Sattar[1], Motahare Bateni[2], Ehsan Zarepur[2], Fahimeh Haghighatdoost[1], Samad Ghaffari[4], Nahid Salehi[5], Masoud Lotfizadeh[6], Nahid Azdaki[7], Ahmadreza Assareh[8], Mahboobeh Gholipour[9], Masoumeh Sadeghi[10], Alireza Khosravi Farsani[11,12] and Nizal Sarrafzadegan[2,12]

Abstract

Background Despite some evidence on individual eating habits in relation to cardiovascular disease, little is known about the combination of common eating habits in relation to premature coronary artery disease (PCAD).

Objective We investigated the association between a combined eating habits score (EHS) and PCAD risk.

Methods In this case-control study, 2022 patients with PCAD and 1063 healthy control were recruited. Women aged between 18 and 70 and men aged between 18 and 60 years were eligible. PCAD was defined as 75% or more stenosis in a single coronary artery disease or at least 50% in the left main coronary artery. Eating habits were assessed through interview and higher scores represent healthier behaviors.

Results Participants in the fourth quartile of EHS had 22% lower risk of PCAD than those in the first quartile (95% CI: 0.61, 0.99; $P=0.024$). Not adding salt at the table (OR = 0.80, 95% CI: 0.66, 0.97; $P=0.023$), low-salt meals (OR = 0.75, 95% CI: 0.57, 0.99; $P=0.022$), and slow eating (OR = 0.42, 95% CI: 0.24, 0.71; $P<0.001$) were associated with lower risk of PCAD while more water drinking (OR = 1.56, 95% CI: 1.07, 2.27; $P=0.013$), and increased meal frequency (OR = 1.85, 95% CI: 1.09, 3.13; $P=0.025$) were linked with increased risk of PCAD.

Conclusion Healthy eating behaviors score, particularly low salt intake and slow eating were associated with lower risk of PCAD. However, higher meal frequency and more water drinking were associated with increased risk of PCAD. Well-designed prospective cohort studies are required.

Keywords Dietary habits, Dietary behaviors, Eating behaviors, Cardiovascular disease, Coronary artery disease

*Correspondence:
Noushin Mohammadifard
noushinmohammadifard@gmail.com
Mahshid Keshavarz
keshavarzmahta2025@gmail.com

Full list of author information is available at the end of the article

Introduction

The top-ranked causes of DALYs in adults older than 50 years was ischemic heart disease and stroke in 2019 [1]. Despite recent progress in the diagnosis and quality of care in patients with cardiovascular disease (CVD), CVD in younger adults has become a growing problem worldwide [2]. Premature coronary artery disease (PCAD) occurs in females and males aged younger than 65 and 55 years, respectively. PCAD has a multifactorial etiology and both genetic and environmental factors contribute to the onset of PCAD. However, the role of environmental factors remains poorly understood [3, 4].

Lifestyle modifications are prominent approaches for prevention. Poor dietary habits are crucial factors in the occurrence of CVD. Eating habits refer to a set of behaviors related to eating that an individual adopts. habitual decision to choose a specific behavior related to eating or food. Dietary habits may include diverse questions on number of meals a day, eating with family members, breakfast consumption, salt and salty snacks consumption and the common method for cooking. The association of individual eating habits with CVD and its risk factors has been investigated in several studies. For instance, skipping any of the main meal and shorter meal intervals were related to more cardiovascular death in American adults [5]. Breakfast skipping, night-time eating and irregular time-restricted feeding may be also associated with higher inflammatory response and metabolic abnormality [6, 7] and thereby increase cardiovascular disease risk [8]. In addition, higher urinary sodium excretion is associated with greater fat mass as a main risk factor for CVD [9].

Although the combination of unhealthy eating habits may have synergistic effects and increase the cumulative impact of various risk factors, we are aware of only a few numbers of studies examining the combined eating habits in relation to clinical outcomes. In a cohort study, among Iranian population, after 10 years follow-up, higher adherence to an unhealthy dietary habits score was accompanied by increased odds of abdominal adiposity, low high-density lipoprotein cholesterol (HDL-C), hypertension impaired glucose tolerance and metabolic syndrome [10]. Consistently, in Chinese, individuals with more unhealthy eating habits score were more probably to be obese but no association was found for other cardiometabolic risk factors like hypertension, diabetes and hypercholesterolemia [11]. Given that eating habits are closely related to culture, sociodemographic factors and economic status [12], exploring the association of various eating habits either individually or in combination with each other with cardiovascular disease would be worthwhile. This may provide an insight into the risky behaviors for chronic diseases in each population. Therefore, in the present study on a representative sample of Iranians with different ethnicities, we investigated how an eating habit score is pertinent to the risk of PCAD and how each component plays a role in this association.

Materials and methods

Design and participants

The present study is a part of the Iran premature coronary artery disease (IPAD) project. This project is a multi-center, hospital-based, case-control study which aims at exploring environmental risk factors of PCAD in diverse ethnicities of Iranians including across 14 cities. Considering the wide range of age cut-off for premature (50 to 65 y) [2], we regarded women younger than 70 years and men younger than 60 years as PCAD. Participants were selected from among patients who attended for coronary angiography, knew their own, paternal and maternal ethnicities and tend to participate in the IPAD project. Patients were distinguished from healthy subjects based on the results of coronary angiography. When there was a stenosis of at least 75% in a single coronary artery disease or at least 50% in the left main coronary, individuals were assigned into the case group. Otherwise, they were located in the control group as normal angiography based on PCI registry. Our exclusion criteria were the history of any documented CAD such as coronary artery bypass surgery, balloon angioplasty, or percutaneous coronary intervention. More information in terms of the study design and methodology was described elsewhere [13]. In the current analysis, we enrolled $n = 3200$ individuals ($n = 2022$ cases and $n = 1066$ controls) who had complete data for questions related to eating habits. The protocol of this study was approved by the ethics committee of Isfahan University of Medical Sciences (IR.MUI.MED.REC.1402.136) All participants provided a written informed consent before the recruitment in the project.

Eating behaviors score

Eating behaviors score was developed using ten independent questions (Table 1). The questions were selected based on relevant eating habits in the literature. The questions were about the consumption frequency of fried foods (< 1, 1–3, 4–6, ≥ 7), adding salt at the table (yes/ no), the salt content of homemade foods (low, moderate, high), water consumption (< 4, 4–8, and ≥ 8 glasses/ day), the daily meal frequency (1–2, 3, 4 ≥ times/ day), breakfast consumption (≤ 4 and ≥ 5 days/ week), late night dinner (before 8 PM and after 8 PM), meals with family members (yes/ no), eating rate (slow, moderate, fast), and meal duration (short or long as less than 20 and more than 30 min and optimum as 20 to 30 min). For each item, the unhealthy behavior was given zero, while the healthy behavior was given 1 point. For the questions about salt content of home-made foods, "high" was

Table 1 Development of dietary habits score

Questions	Possible answers	Scores
Do You usually eat fried foods?	< 1	3
	1–3	2
	4–6	1
	≥ 7	0
Do You usually add salt at the table?	Yes	0
	No	1
How salty is your food?	Salty	0
	Normal	1
	Low salt	2
How many glasses of water do you usually drink per day?	< 4	0
	4–8	1
	≥ 8	2
How many meals do you usually eat per day?	1–2	0
	3	1
	≥ 4	2
How many days do you usually eat breakfast in a week?	≤ 4	0
	≥ 5	1
Do you usually eat your meals with family?	No	0
	Yes	1
How fast do you usually eat your meals?	Quickly	0
	Moderately	1
	Slowly	2
How many minutes does it take to eat your meal?	< 20 or > 30 min	0
	20 to 30 min	1
What time do you usually eat your dinner?	After 8 PM	0
	Before 8 PM	1

given zero, "moderate" was given 1 and "low" was given 2 points. The overall score was calculated by the summing up of the scores received for each item by the participants. The possible score range is from zero to 14. Higher scores indicate a healthier eating habits score.

Confounding factors assessment

Information about demographic factors including age, sex, education attainment, and marital status, ethnicity, different lifestyle factors (smoking habits, physical activity level, and dietary habits), and medical history were gathered via interview, conducted by qualified health staff. Physical activity level was determined with the Persian version of the international physical activity questionnaire (IPAQ) [14]. Body weight was determined by using calibrated digital scale while participants were barefoot and minimally clothing and height was determined while shoulders were in normal position using a wall-fixed tape [15]. Body mass index (BMI) was estimated through dividing weight (kg) by the square of height (m^2). The average of two measurements a mercury sphygmomanometer and according to standard protocols [16] was recorded as blood pressure.

Statistical analysis

Participants were categorized into four groups based on the quartiles of eating habits score. Continuous and categorical variables were reported as mean (SD) and frequency (%) across the quartiles of the eating habits score. To assess the differences across quartiles, analysis of variance (ANOVA) for continuous variables and chi-square test for categorical variables were used. Multiple logistic regression was used to evaluate the odds ratio (OR) and 95% confidence interval (CI) for the presence of PCAD across the quartiles of eating habits score in crude and various multivariable-adjusted models. We controlled confounding effects of age (continuous), sex (men/ women) and energy (kcal/d) in the first adjusted model. Model 2 was additionally adjusted for education (illiterate, primary school and higher than primary school), smoking (never/ ex-smoker/ current smoker), physical activity (METS/ minute per week), marital status (married vs. others), alcohol consumption (yes/ no) and ethnicity. Further control was made for BMI (continuous), hypertension (yes/ no), diabetes mellitus (yes/ no), hypercholesterolemia (yes/ no), and aspirin use (yes/ no) in model 3. The associations for individual components of eating habits score in crude and fully-adjusted model were also determined. All analyses were performed at 5% error level, using Statistical Package for Social Sciences (SPSS, Inc., Chicago IL, USA; version 20).

Results

Participants in the top quartile of the eating habits score were older, and less likely to be male, current smoker, or alcohol consumer than those in the first quartile. The prevalence of dyslipidemia was also higher in the fourth quartile while the prevalence of hypertension and diabetes did not differ across the quartiles of the eating habits score (Table 2).

The frequency of different eating habits components across the quartiles of the eating habits score is shown in Table 3. Compared with those in the first quartile, individuals in the fourth quartile were less likely to use fried foods, add salt at the table, and eat salty foods, but more likely to drink more water, have increased meal frequency, eat meals with family members, eat slowly, have breakfast more than 5 days a week, have a meal duration of 20 to 30 min and have early night dinner.

Crude and multivariable-adjusted ORs (95% CIs) for the presence of PCAD across quartiles of eating habits score are presented in Table 4. In the crude model, more adherence to healthy eating habits was associated with a significantly reduced risk of the presence of PCAD (OR = 0.73, 95% CI: 0.50, 0.90; $P = 0.002$). Further adjustment for age, sex and various lifestyle factors slightly weakened the association (OR = 0.80, 95% CI: 0.64, 1.01; $P = 0.047$). In the fully adjusted model, participants with

Table 2 General and demographic variables in subjects with and without premature coronary artery disease

	Quartiles of eating habits score				P value*
	1 (low quality) (n = 776)	2 (n = 533)	3 (n = 570)	4 (high quality) (n = 809)	
Sex (Male), n (%)	519 (66.9)	304 (57.0)	304 (53.3)	393 (48.6)	< 0.001
Age, (years)**	52.39 ± 7.72	54.04 ± 7.87	53.86 ± 7.54	54.40 ± 7.33	< 0.001
Married, n (%)	713 (91.9)	474 (88.9)	507 (88.9)	725 (89.4)	0.207
Ethnicity n (%):					< 0.001
Fars	464 (59.9)	280 (52.8)	317 (55.7)	429 (53.2)	
Azari	66 (8.5)	44 (8.3)	39 (6.9)	40 (5.0)	
Kurd	95 (12.3)	60 (11.3)	53 (9.3)	81 (10.0)	
Lor	40 (5.2)	38 (7.2)	28 (4.9)	33 (4.1)	
Bakhtiari	32 (4.1)	34 (6.4)	35 (6.2)	75 (9.3)	
Qashqaei	26 (3.4)	21 (4.0)	29 (5.1)	34 (4.2)	
Arab	18 (2.3)	14 (2.6)	19 (3.3)	26 (3.2)	
Gilak	29 (3.7)	37 (7.0)	48 (8.4)	87 (10.8)	
Smokers, n (%)	233 (30.0)	113 (21.2)	104 (18.2)	133 (16.4)	< 0.001
Alcohol drinking n (%)	45 (5.8)	36 (6.8)	11 (1.9)	25 (3.1)	< 0.001
Physical activity (MET-min/wk)**	1929.06 ± 4115.78	1999.23 ± 3798.77	1940.72 ± 3986.85	1838.20 ± 3797.25	0.900
Education n (%):					0.472
< 6 years	353 (45.5)	269 (50.5)	296 (51.9)	416 (51.4)	
6–12 years	327 (42.1)	204 (38.3)	217 (38.1)	303 (37.5)	
13–16 years	75 (9.7)	48 (9.0)	44 (7.7)	68 (8.4)	
> 16 years	21 (2.7)	12 (2.3)	13 (2.3)	22 (2.7)	
Aspirin use n (%)	210 (27.5)	146 (27.5)	147 (26.1)	224 (27.9)	0.905
Dyslipidemia, n (%)	760 (97.9)	526 (98.7)	568 (99.6)	805 (99.5)	0.005
Hypertension, n (%)	763 (98.3)	525 (98.5)	562 (98.6)	795 (98.3)	0.962
Diabetes, n (%)	684 (88.1)	451 (84.6)	474 (83.2)	698 (86.3)	0.056
Body mass index (kg/m^2)**	28.30 ± 4.99	28.20 ± 5.80	28.36 ± 4.85	28.72 ± 5.34	0.243
Waist circumference (cm)**	99.80 ± 12.73	99.27 ± 13.11	99.03 ± 12.38	99.85 ± 12.23	0.574
Energy intake (Kcal)**	2148.91 ± 841.27	2099.18 ± 786.76	2077.63 ± 808.16	2070.66 ± 839.01	0.225

*Derived from 1-factor ANOVA and chi-square test for continuous and categorical variables, respectively

**Values are means ± SDs

higher scores of eating habits score had 22% lower risk of PCAD compared with those with the lowest scores (OR = 0.78, 95% CI: 0.61, 0.99; $P = 0.024$).

Crude and multivariable-adjusted ORs (95% CIs) for the presence of PCAD across quartiles of individual eating habit components are indicated in Table 5. In the crude model, more frequent meals and breakfast consuming were associated with increased risk of PCAD whereas having family meals and slow eating were associated with decreased risk of PCAD. After adjustment for potential confounders, not adding salt at the table (OR = 0.80, 95% CI: 0.66, 0.97; $P = 0.023$), low-salt meals (OR = 0.75, 95% CI: 0.57, 0.99; $P = 0.022$), and slow eating (OR = 0.42, 95% CI: 0.24, 0.71; $P < 0.001$) were associated with lower risk of PCAD while more water drinking (OR = 1.56, 95% CI: 1.07, 2.27; $P = 0.013$), and increased meal frequency (OR = 1.85, 95% CI: 1.09, 3.13; $P = 0.025$) were linked with increased risk of PCAD. Fried food consumption, late night dinner and shorter meal duration were not related to PCAD risk either in crude or adjusted model.

Discussion

In this study, we found a significant relationship between eating habits score and PCAD risk; as individuals in the highest quartile of the eating habits score had lower risk of PCAD compared with those in the first quartile, who had the unhealthiest eating habits. Amongst different components of the eating habits score, not adding salt at the table, low-salt homemade foods and slow eating rate were related to the lower risk of PCAD while higher meal frequency and greater water drinking were associated with increased risk of PCAD.

Some earlier studies examining the association of eating behaviors with CVD showed that non-optimal dietary behaviors were associated with increased risk of various cardiovascular events [12, 17, 18]. In a large cohort of Japanese, non-optimal dietary behaviors, consisting of frequent breakfast skipping, late night dinner and bedtime snacking, were associated with increased risk of stroke, heart failure, myocardial infarction, and angina after a follow-up duration of 2.5 years [17]. In another cohort study of Japanese, breakfast skipping and eating quickly were associated with incident major

Table 3 The frequency of individual eating behaviors across eating behaviors score quartiles

	Quartiles of eating habits score				*P* value
	1 (low quality)	**2**	**3**	**4 (high quality)**	
Fried food, **n (%)**					< 0.001
0	19 (2.8%)	4 (0.3%)	0	0	
1	179 (26.3%)	141 (11.6%)	38 (6.5%)	8 (1.1%)	
2	386 (56.8%)	728 (60.1%)	296 (50.5%)	268 (37.4%)	
3	96 (14.1%)	338 (27.9%)	252 (43%)	441 (61.5%)	
Adding salt at the table, **n (%)**					
Salt content of homemade food, **n (%)**					< 0.001
Low	89 (9.3)	136 (21.6)	255 (37.6)	607 (65.5)	
Medium	521 (54.3)	384 (61.0)	353 (52.0)	296 (31.9)	
High	349 (36.4)	109 (17.3)	71 (10.5)	24 (2.6)	
Water glasses/day, **n (%)**					< 0.001
1–3	542 (56.5)	295 (46.9)	278 (40.9)	244 (26.3)	
4–7	347 (36.2)	258 (41.0)	298 (43.9)	413 (44.6)	
≥ 8	70 (7.3)	76 (12.1)	103 (15.2)	270 (29.2)	
Meal frequency, **n (%)**					< 0.001
1–2	189 (19.7)	62 (9.9)	37 (5.4)	24 (2.6)	
3	737 (76.9)	536 (85.2)	602 (88.7)	824 (88.9)	
≥ 4	33 (3.5)	31 (4.9)	40 (5.9)	79 (8.4)	
Eating breakfast, **n (%)**					< 0.001
≤ 4	218 (22.7)	74 (11.8)	51 (7.5)	25 (2.7)	
≥ 5	741 (77.3)	555 (88.2)	628 (92.5)	902 (97.4)	
Family meals (yes), **n (%)**	627 (65.4)	533 (84.7)	621 (91.5)	901 (97.2)	
Eating rate, **n (%)**					< 0.001
Slow	111 (11.6)	145 (23.1)	179 (26.4)	378 (40.8)	
Moderate	710 (74.0)	450 (71.5)	477 (70.3)	528 (57.0)	
Fast	138 (14.4)	34 (5.4)	23 (3.4)	21 (2.3)	
Meal duration, **n (%)**					< 0.001
< 20 or > 30 min	753 (78.6)	438 (69.7)	392 (57.7)	362 (39.0)	
20–30 min	206 (21.4)	191 (30.3)	287 (42.3)	565 (60.9)	
Late night dinner, **n (%)**					< 0.001
Before 8 pm	237 (24.7)	225 (35.8)	296 (43.6)	496 (53.5)	
After 8 pm	722 (75.3)	404 (64.2)	383 (56.4)	431 (46.5)	

Table 4 Odds ratio (95% confidence interval) of premature coronary artery disease risk according to eating behaviors score quartiles

	Crude	Model 1	Model 2	Model 3
Q1	1 (Ref)	1 (Ref)	1 (Ref)	1 (Ref)
Q2	1.02 (0.80, 1.30)	1.08 (0.83, 1.40)	1.08 (0.83, 1.41)	1.08 (0.82, 1.43)
Q3	0.90 (0.71, 1.13)	0.99 (0.77, 1.28)	0.99 (0.77, 1.28)	0.93 (0.71, 1.22)
Q4	0.73 (0.59, 0.90)	0.80 (0.64, 1.01)	0.80 (0.64, 1.01)	0.78 (0.61, 0.99)
P for trend[1]	0.002	0.047	0.047	0.024

[1]Derived from a Mantel-Haenszel extension chi-square test

Model 1: Adjusted for age, sex and energy intake

Model 2: Additionally adjusted for education, smoking (never/ ex-smoker/ current smoker), physical activity (METS/ minute per week), marital status, alcohol consumption and ethnicity

Model 3: Additionally adjusted for body mass index (Kg/ m^2), hypertension (yes/ no), diabetes mellitus (yes/ no), hypercholesterolemia (yes/ no), aspirin use (yes/ no)

adverse cardiovascular events (MACE) in subjects with normal body weight but not overweight individuals [18]. Although various non-optimal eating behaviors have been linked with different health concerns, data are not consistent for a specific behavior in different populations. Eating behaviors may affect the risk of CVD via their close link with the hyperactivity of the hypothalamic-pituitary-adrenal axis following psychological stress and circadian rhythm [19–21]. Furthermore, non-optimal eating behaviors might be associated with unhealthy food choices [22] which affect the development of diseases. Nevertheless, reasons for discrepancies between studies

Table 5 Odds ratio (95% confidence interval) of premature coronary artery disease risk according to individual eating behaviors categories

	Categories of eating behaviors	Crude	Model 1
Fried food	< 1	1 (Ref)	1 (Ref)
	1–3	1.15 (0.96, 1.36)	1.14 (0.93, 1.39)
	4–6	0.85 (0.64, 1.13)	0.86 (0.62, 1.21)
	≥ 7	0.95 (0.34, 2.64)	0.90 (0.27, 3.05)
	P for trend[1]	0.921	0.942
Adding salt at the table	Yes	1 (Ref)	1 (Ref)
	No	0.85 (0.72, 1.00)	0.80 (0.66, 0.97)
	P for trend[1]	0.050	0.023
Salt content of homemade food	High	1 (Ref)	1 (Ref)
	Medium	1.04 (0.83, 1.31)	0.93 (0.71, 1.21)
	Low	0.92 (0.72, 1.16)	0.75 (0.57, 0.99)
	P for trend[1]	0.306	0.022
Water glasses/day	1–3	1 (Ref)	1 (Ref)
	4–7	1.01 (0.84, 1.20)	1.17 (0.95, 1.43)
	≥ 8	1.31 (1.95, 1.81)	1.56 (1.07, 2.27)
	P for trend[1]	0.235	0.013
Meal frequency	1–2	1 (Ref)	1 (Ref)
	3	1.51 (1.15, 1.98)	1.24 (0.90, 1.72)
	≥ 4	2.26 (1.43, 3.57)	1.85 (1.09, 3.13)
	P for trend[1]	< 0.001	0.025
Eating breakfast	≤ 4	1 (Ref)	1 (Ref)
	≥ 5	1.45 (1.14, 1.84)	1.21 (0.91, 1.61)
	P for trend[1]	0.003	0.181
Family meals	No	1 (Ref)	1 (Ref)
	Yes	0.70 (0.56, 0.88)	1.06 (0.80, 1.40)
	P for trend[1]	0.002	0.685
Eating rate	Fast	1 (Ref)	1 (Ref)
	Moderate	0.77 (0.50, 1.19)	0.55 (0.33, 0.93)
	Slow	0.60 (0.38, 0.94)	0.42 (0.24, 0.71)
	P for trend[1]	0.002	< 0.001
Meal duration	< 20 or > 30 min	1 (Ref)	1 (Ref)
	20–30 min	0.85 (0.72, 1.00)	0.89 (0.74, 1.08)
	P for trend[1]	0.057	0.242
Late night dinner	After 8 pm	1 (Ref)	1 (Ref)
	Before 8 pm	0.94 (0.80, 1.11)	1.05 (0.87, 1.27)
	P for trend[1]	0.456	0.617

[1]Derived from a Mantel-Haenszel extension chi-square test

Model 1: Adjusted for age, sex, energy intake, education, smoking (never/ ex-smoker/ current smoker), physical activity (METS/ minute per week), marital status, alcohol consumption, ethnicity, body mass index (Kg/ m^2), hypertension (yes/ no), diabetes mellitus (yes/ no), hypercholesterolemia (yes/ no), aspirin use (yes/ no)

might be related to variations in eating behaviors. For instance, while Kaneko et al. defined breakfast skipping as more than 3 times a week [17], we considered 4 days per week. There was similar inconsistence for late dinner. While we considered a specific time for all individuals regardless of their bedtime, they defined late night dinner as eating two hours before their bedtime for more than three times a week [17].

Although some studies have shown a beneficial association between increased meal frequency and various cardiometabolic risk factors and cardiovascular disease [23–25], there is uncertainty with respect to optimal meal frequency [26] while decreased meal frequency may even indicate more favorable serum lipids levels [26, 27] and fasting diets have emerged as a strategy to deal with obesity and comorbidities. Furthermore, in a randomized controlled trial, individuals with one meal a day had lower body weight and fat mass compared to those with three meals a day [28]. The potential mechanisms underlying the inverse association between eating rate and PCAD might be attributed to lower dyslipidemia and adiposity in slow-eaters compared with fast-eaters [29–32].

The direct link between water and CVD risk might be related to the hardness of drinking water [33–35], or its high level of arsenic [36, 37]. In a study of 41 samples of drinking water in Iran, arsenic content of almost 85% of samples was greater than the limit suggested by the World Health Organization (WHO), that is, 10 µg/L [38]. Moreover, nutrient deficiencies in patients with specific needs may disrupt the pathway of arsenic metabolism [39] and therefore its adverse effects may occur even with low levels of arsenic in drinking water. In addition, although the hardness of drinking water is a protective factor against CVD [33], a null association was observed in previous studies among Iranians [40] and some other studies [34, 35]. This may suggest that the detrimental effects of high content of arsenic in drinking water outweigh the favorable effects of ions in water.

Considering limitations is in order when interpreting these findings. Despite taking into account the effect of various confounders to extract associations, residual and unmeasured confounders could not be ruled out. Due to the case-control design of the study, we were not able to explore any causality. On the other hand, it is possible that patients have changed their dietary behaviors towards healthier ones. The imbalance between the number of cases and controls is another limitation of our study which may affect findings. However, the large sample size of our study may mitigate this issue. Moreover, since there is no standard for eating behaviors definition, different studies used their own specific definition which can affect the results. We also failed to assess some other behaviors which can potentially affect health status such as meal timing and snack consumption. Finally, the small number of participants in some categories of dietary behaviors might be the reason behind the null association in our findings and future studies with larger sample size are required. However, our study has its own strengths including a large representative sample of Iranians with different ethnicities and sociodemographic variability which increase the external validity of our findings and identifying patients from healthy controls through angiography test.

In conclusion, eating behaviors score and some of its components including lower salt intake and slow eating were associated with lower risk of PCAD in Iranian adults. Conversely, increased meal frequency and more water consumption were identified as a risk factor for PCAD in our study. Our results may highlight the potential relevance of combined effect of various eating behaviors; however, well-designed prospective cohort studies are warranted to confirm these results.

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Author contributions
Ehsan Shirvani: Conceptualization (equal); writing– original draft (equal); writing– review and editing (equal). Noushin Mohammadifard: Data curation (lead); project administration (equal); supervision (lead); writing– review and editing (equal). Mahshid Keshavarz: Writing– original draft (equal). Fereshteh Sattar: Writing– original draft (equal). Motahareh Bateni: Formal analysis (lead). Ehsan Zarepur: Resources (equal). Fahimeh Haghighatdoost: Conceptualization (equal); writing– review and editing (equal). Samad Ghaffari: Resources (equal). Nahid Salehi: Resources (equal). Masoud Lotfizadeh: Resources (equal). Nahid Azdaki: Resources (equal). Ahmadreza Assareh: Resources (equal). Mahboobeh Gholipour: Resources (equal). Masoumeh Sadeghi: Resources (equal). Alireza Khosravi: Resources (equal). Nizal Sarrafzadegan: Funding acquisition (lead); project administration (equal); resources (equal).

Data availability
Data will be available on request from the corresponding author.

Declarations

Competing interests
The authors declare no competing interests.

Conflict of interest
There are no conflicts of interest.

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Author details
[1]Interventional Cardiology Research Center, Cardiovascular Research Institute, Isfahan University of Medical Sciences, Isfahan, Iran
[2]Isfahan Cardiovascular Research Center, Cardiovascular Research Institute, Isfahan University of Medical Sciences, Isfahan, Iran
[3]Department of Cardiology, School of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran
[4]Cardiovascular Research Center, Tabriz University of Medical sciences, Tabriz, Iran
[5]Cardiovascular Research Center, Health Institute, Kermanshah University of Medical Sciences, Kermanshah, Iran
[6]Department of Community Health, Shahrekord University of Medical Sciences, Shahrekord, Iran
[7]Cardiovascular Diseases Research Center, Birjand University of Medical Sciences, Birjand, Iran
[8]Atherosclerosis Research Center, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran
[9]Cardiovascular Diseases Research Center, Department of Cardiology, School of Medicine, Heshmat Hospital, Guilan University of Medical Sciences, Rasht, Iran
[10]Cardiac Rehabilitation Research Center, Cardiovascular Research Institute, Isfahan University of Medical Sciences, Isfahan, Iran
[11]Hypertension Research Center, Cardiovascular Research Institute, Isfahan University of Medical Sciences, Isfahan, Iran
[12]Iranian Network of Cardiovascular Research (INCVR), Tehran, Iran

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