BEHAVIORAL AND FAMILIAL RISK FACTORS FOR PREMATURE VERSUS NON-PREMATURE CORONARY ARTERY DISEASE; A CASE-CONTROL STUDY

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Submitted 23 July 2025; accepted 15 September 2025; Published 01 July 2026

ABSTRACT

Background: Premature coronary artery disease (pCAD) presents a growing global health concern due to its association with modifiable risk factors and familial predisposition. This study aimed to identify behavioral and familial risk factors contributing to the prevalence of pCAD and compare them to those associated with non-pCAD.

Methods: A case-control study was conducted over one year, enrolling 350 patients (198 with pCAD and 152 with non-pCAD) from the Azadi Heart Center and related facilities. Premature CAD was defined as CAD onset before age 45 in males and 55 in females. Demographic, clinical, and lifestyle data were collected, and statistical analyses were performed using SPSS version 26, employing Chi-square, Mann-Whitney U test, and logistic regression. A p-value < 0.05 was considered statistically significant.

Results: Premature CAD was significantly associated with female gender, positive family history, non-STEMI presentation, diabetes, hypertension, obesity, and former smoking ($p < 0.05$). Lifestyle factors such as lack of physical exercise, high salt intake, indoor activity, and a vegetarian diet were also significantly linked to pCAD. In contrast, depression and fatty diet were more associated with non-pCAD. Logistic regression analysis demonstrated that these factors collectively predicted pCAD with 70% accuracy. Hyperlipidemia, alcohol consumption, and anxiety showed no significant association.

Conclusion: This study highlights the prominent role of both behavioral and familial factors in the development of pCAD. Emphasis on early identification and lifestyle modification among at-risk individuals—particularly younger females—may reduce the burden of premature coronary artery disease.

Duhok Med J 2026; 20 (1): 36-49.

Keywords: Premature CAD, Risk Factors, Lifestyle, Coronary Artery Disease.

Coronary artery disease is the most common form of heart disease and the leading cause of morbidity and mortality worldwide. The disease represents inadequate blood supply to the myocardium due to the buildup of atherosclerotic plaque and narrowing of coronary arteries[1]. Over 300 million prevalent cases in 2022[1-4]. According to the Global Ranking of Cardiovascular Deaths by Cause[5], ischemic heart disease (IHD) remains at the lead with several attributed burden risk factors, the disease is classified according to the age of the first clinical manifestation[6], and premature coronary artery disease (pCAD) is defined as the occurrence of the first clinical manifestations of CAD in men aged younger than 45 and women less than 55 years[6].

The development of IHD is known to occur based on a complex interplay between genetic, environmental, and lifestyle factors[7]. Familial background plays a significant role in IHD development through the transmission of genetic information between generations and through behavioral habits[7]. The heritability of CAD is estimated at approximately 50-60%[8].

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https://doi.org/10.31386/dmj.2026.20.1.4

CAD risk factors are generally categorized into modifiable and non-modifiable risk factors. The modifiable and behavioral risk factors include hypertension, dietary risks, smoking, second-hand smoking, high body mass index, high fasting blood glucose, hyperlipidemia, and high alcohol use[5, 9-11]. Additionally, patients with pre-existing disease are at greater risk for the development of CAD than healthy individuals[12]. It has been estimated that 1/3 of the patients presenting with CAD and undergoing PCI are categorized under the category of premature CAD[4].

PCAD represents a global health concern with a significant burden on public health. Additionally, more than four-fifths of patients presenting with PCAD have at least one modifiable risk factor[6]. This study aims to identify the modifiable risk factors besides genetic predisposition that contribute to a high prevalence of pCAD and correlate them to non-pCAD.

MATERIAL AND METHODS

This study was conducted as a case-control study, signifying the importance of assessing and comparing pCAD and Non-pCAD. The cases were regarded as Premature CAD patients according to the definitions mentioned below, and they were compared to the control group (Non-Premature CAD), labeled as older age groups presenting with features of ACS. A total of 350 cases were enrolled in this study from the Azadi Emergency Hospital, Coronary Care Unit, and Cardiac Center. The study took 12 months, from October 2023 to October 2024. A formal consent was obtained from every patient included. Patients were recruited upon their presentation to the emergency department following the diagnosis of CAD, as they were complaining of symptoms suggestive of IHD in addition to the Electrocardiogram and Serum High Sensitive Troponin, through using the 2/3 criteria for diagnosis. Inclusion criteria: all cases with CAD were enrolled. Patients under the classification of premature CAD, male patients less than 45 years, and female patients less than 55 years of age presenting as Acute coronary syndrome (STEMI or NSTEMI). Additionally, other factors were included depending on the criteria. Family history was regarded as positive only if the patient had a first-degree relative with IHD within the above limits of age (males less than 45 years and females less than 55 years of age). Previous history of hypertension, diabetes, and hyperlipidemia is regarded as positive if these conditions were previously documented or the patients were already on medical therapy for these conditions. Moreover, any patient who had smoked 20 packs in their lifetime or more than one cigarette/day for one year is regarded as positive, while those who had quit smoking for at least 12 months before the interview were regarded as former smokers[13]. Patients were energy drink consumers only if they drank these products on a regular daily basis[14]. Alcohol addiction was defined as a daily uncontrollable alcohol consumption with interference with daily activities[15]. Patients were considered exercisers if they walked briskly for at least 30-60 minutes per day, for at least 5 days per week[16]. Patients using extra salt during meals, or if the meals of others were regarded as inadequate salt for them, were regarded as having a salty diet. Patients were asked if their diet is high in saturated fat, such as red meat, full-fat dairy products, which are regarded as a fatty diet, while patients who never consume animal products were considered vegetarians[17-18]. Additionally, the Body Mass Index[19] and waist circumference were measured[20].

The data were analyzed using SPSS version 26. The test for normality using Kolmogorov-Smirnov (KS) and Shapiro-Wilk tests assessed the numerical variables. The Mann-Whitney U test was used later, which is a non-parametric test used to assess for significant differences in a scale or ordinal dependent variable by a single dichotomous independent variable.

Additionally, the Chi-square (Fisher's Exact) test was used to assess the association between demographic and clinical factors with pCAD vs. non-pCAD, between lifestyle and stress-related factors with pCAD vs. non-pCAD, and clinical, employment, and behavioral characteristics were analyzed to explore their association with premature and non-premature coronary artery disease. The bivariate analysis was used to identify the multiple significant associations between demographic, clinical, and behavioral characteristics with pCAD. Finally, multiple logistic regression analysis was used to assess the significant predictors of pCAD cases. P values of less than 0.05 were regarded as statistically significant. Pearson's square test was used to analyze the statistics of differences between cases and controls.

RESULTS

Females are significantly more likely to have premature CAD than males ($p < 0.001$, highly significant), compared to males, 70.2% in the non-premature CAD group. Family history of CAD ($p = 0.049$, significant), of premature CAD patients is slightly more frequent in premature CAD patients, but the association is weak, NONSTEMI is more common in premature CAD patients ($p<0.001$, highly significant). diabetes ($p < 0.001$) highly significant and more common in premature CAD patients. Hypertension ($p = 0.005$, significant), is more frequent in non-premature CAD patients. hyperlipidemia ($p = .832$, not significant), does not significantly differ between groups. Former smokers ($p < 0.001$, highly significant) have a higher prevalence of premature CAD, while current smokers have a lower prevalence. Obesity ($p = .005$, significant) is more common in premature CAD patients. Overall, significant predictors of premature CAD ($p < .05$) are female, family history of CAD, NSTEMI type of ACS, diabetes mellitus, hypertension, smoking (former smokers at higher risk), obesity. While hyperlipidemia is not significant.

This analysis suggests that younger females, those with a family history, NONSTEMI type ACS, diabetes, hypertension, obesity, and former smokers are more likely to develop premature CAD [Table 1].

Table 1: Demographical and clinical factors distribution and association in patients with premature and non-pCAD.

Clinical characteristics		PCAD (n=198)	Non-pCAD (n=152)	Total (n=350)	Chi-square	Sig.
		n (%)	n (%)	n (%)		
Gender	Male	54 (29.8)	127 (70.2)	181 (51.7)	28.197	< 0.001*
	Female	98 (58.0)	71 (42.5)	169 (48.3)		
Family history	Yes	73 (38.6)	116 (61.4)	189 (54.0)	3.860	.049*
	No	79 (49.1)	82 (50.9)	161 (46.0)		
Type ACS	STEMI (ACS)	20 (19.8)	81 (80.2)	101 (28.9)	32.257	< 0.001*
	NONSTEMI	132 (53.0)	117 (47)	249 (71.1)		
Diabetes mellitus	Yes	44 (31.4)	96 (68.6)	140 (40.0)	13.676	< 0.001*
	No	108 (51.4)	102 (48.6)	210 (60.0)		
Hypertension	Yes	77 (37.2)	130 (62.8)	207 (59.1)	8.005	0.005*
	No	75 (52.4)	68 (47.6)	143 (40.9)		
Hyperlipidemia	Yes	62 (42.8)	83 (57.2)	145 (41.4)	.045	0.832
	No	90 (43.9)	115 (56.1)	205 (58.6)		
Smoking	Current	5 (11.9)	37 (88.1)	42 (12.0)	19.549	< 0.001*
	Former	106 (48.6)	112 (51.4)	218 (62.3)		
	No	41 (45.6)	49 (54.4)	90 (25.7)		

Clinical characteristics		PCAD (n=198)	Non-pCAD (n=152)	Total (n=350)	Chi-square	Sig.
		n (%)	n (%)	n (%)		
Obesity (BMI=>30)	Yes	64 (53.8)	55 (46.2)	119 (34.0)	7.866	0.005*
	No	88 (38.1)	143 (61.0)	231 (66.0)		
	Total	**198 (56.6)**	**152 (43.4)**	**350 (100.0)**		

CAD; Coronary artery disease, ACS; Acute coronary syndrome
*. The Chi-square statistic is significant at the .05 level.

Alcohol use (p=0.541), not significant difference between alcohol addicts, social drinkers, and non-drinkers in premature vs. non-premature CAD, thereby alcohol consumption does not appear to be a major risk factor for premature CAD. Physical exercise ($p < 0.001$, highly significant) lack of physical exercise is significantly associated with premature CAD. Anxiety ($p = 0.066$, borderline significance); anxiety may have some association with premature CAD, but it is not statistically significant. Depression ($p < 0.001$, highly significant), is significantly more common in non-premature CAD patients. High salt intake ($p = 0.003$, significant), 67.5% of premature CAD patients reported high salt intake vs. 32.5% in non-premature CAD; high salt intake is significantly associated with premature CAD. Overeating ($p = 0.913$, not significant), 57.0% of premature CAD patients overate vs. 43.0% in non-premature CAD, overeating does not show a significant association with premature CAD. Vegetarian diet (p=0.007, significant), a vegetarian diet is significantly associated with premature CAD. This could be due to other confounding dietary habits. Fatty diet intake ($p<0.001$, highly significant), 39.1% of premature CAD patients had a fatty diet vs. 60.9% in non-premature CAD, a fatty diet is significantly more common in non-premature CAD patients. Activity Type ($p = 0.024$, significant), indoor activities is significantly associated with premature CAD, suggesting a sedentary lifestyle may contribute to early disease onset.

Overall conclusion, significant lifestyle factors associated with premature CAD ($p < 0.05$) are lack of physical exercise, high salt intake, vegetarian diet, indoor activity (sedentary lifestyle), while significant lifestyle factors associated with non-premature CAD are depression, fatty diet intake, outdoor activity, lastly, alcohol use, anxiety, and overeating are not significant [Table 2].

Table 2: Lifestyle and stress variables distribution and association in patients with pCAD and non-pCAD.

lifestyles and stress characteristics		PCAD (n=198)	Non-pCAD (n=152)	Total (n=350)	Chi-square	Sig.
		n (%)	n (%)	n (%)		
Alcohol use	Addict	2 (33.3)	4 (66.7)	6 (1.7)	1.444	0.541a
	Social	5 (62.5)	3 (37.5)	8 (2.3)		
	No	191 (56.8)	145 (43.2)	336 (96.0)		
Physical exercise	Yes	24 (36.9)	41 (63.1)	65 (18.6)	12.543	< 0.001*
	No	174 (61.1)	111 (38.9)	285 (81.4)		
Anxiety	Yes	103 (52.3)	94 (47.7)	197 (56.3)	3.371	0.066
	No	95 (62.1)	58 (37.9)	153 (43.7)		
Depression	Yes	38 (38.8)	60 (61.2)	98 (28.0)	17.545	< 0.001*
	No	160 (63.5)	92 (36.5)	252 (72.0)		
High salt intake	Yes	79 (67.5)	38 (32.5)	117 (33.4)	8.577	0.003*
	No	119 (51.1)	114 (48.9)	233 (66.6)		
Over eating	Yes	61 (57.0)	46 (43.0)	107 (30.6)	.012	0.913

	No	137 (56.4)	106 (43.6)	243 (69.4)		
Vegetarian	Yes	60 (69.0)	27 (31.0)	87 (24.9)	7.239	0.007*
	No	138 (52.5)	125 (47.5)	263 (75.1)		
Fatty diet intake	Yes	43 (39.1)	67 (60.9)	110 (31.4)	19.952	< 0.001*
	No	155 (64.6)	85 (35.4)	240 (68.6)		
Activity	Outdoor	14 (38.9)	22 (61.1)	36 (10.3)	5.107	0.024*
	Indoor	184 (58.6)	130 (41.4)	314 (89.7)		
	Total	**198 (56.6)**	**152 (43.4)**	**350 (100.0)**		

CAD; Coronary artery disease.
* The Chi-square statistic is significant at the .05 level. a. Fisher's Exact Test used.

Subgroups of CAD ($p < 0.001$, highly significant), STEMI (ST-Elevation Myocardial Infarction) and Non-STEMI presentations are completely segregated, all premature STEMI cases (100%) are classified under the "Not premature STEMI" group, while all premature non-STEMI cases are in the "Not premature non-STEMI" group, the classification of STEMI and Non-STEMI is highly associated with the premature vs. non-premature CAD categorization, showing a statistically absolute relationship. Employment Status ($p = 0.064$, borderline significance), 62.9% of employed individuals had premature CAD, compared to 37.1% with non-premature CAD. there is a trend that employed individuals may be more likely to develop premature CAD, this does not reach statistical significance ($p > 0.05$). Ever Smoking ($p=0.012$, significant), 65.2% of premature CAD patients were ever smokers, compared to 34.8% in non-premature CAD, smoking history is significantly associated with premature CAD, reinforcing its role as a major modifiable risk factor. Ever alcohol use ($p= 0.613$, not significant), 50.0% of premature CAD patients had a history of alcohol use, which is similar to the 50.0% in non-premature CAD, alcohol use does not show a significant association with premature CAD. Obesity (BMI≥30) ($p=0.005$, significant), 46.2% of premature CAD patients were obese, compared to 53.8% in non-premature CAD, obesity is significantly associated with non-premature CAD rather than premature CAD, suggesting other factors may contribute more strongly to early onset CAD. Lack of exercises ($p < 0.001$, highly significant), 61.1% of premature CAD patients lacked exercise vs. 38.9% in non-premature CAD, lack of physical activity is strongly associated with premature CAD, reinforcing the importance of exercise in cardiovascular health.

Overall conclusion, factors significantly associated with premature CAD ($p < 0.05$) are ever smoking, obesity (BMI ≥30), lack of exercise, CAD classification (STEMI/Non-STEMI), while, factors that are borderline significant are employment status, and factors not significantly associated with premature CAD are alcohol use [Table 3].

Table 3: Premature subgroups, employment, and further lifestyles variables distribution and association in patients with pCAD and non-pCAD.

Clinical, Employment & Behavior characteristics		**PCAD (n=198)**	**Non-pCAD (n=152)**	**Total (n=350)**	**Chi-square**	**Sig.**
		n (%)	**n (%)**	**n (%)**		
Subgroups	Premature STEMI	0 (0.0)	20 (100.0)	20 (5.7)	350.000	< 0.001*
	Not premature STEMI	81 (100.0)	0 (0.0)	81 (23.1)		
	Premature Not STEMI	0 (0.0)	132 (100.0)	132 (37.7)		
	Not premature not STEMI	117 (100.0)	0 (0.0)	117 (33.4)		

Employment	Yes	83 (62.9)	49 (37.1)	132 (37.7)	3.432	0.064
	No	115 (52.8)	103 (47.2)	218 (62.3)		
Ever Smoking	Yes	86 (65.2)	46 (34.8)	132 (37.7)	6.350	< 0.012*
	No	112 (51.4)	106 (48.6)	218 (62.3)		
Ever alcohol use	Yes	7 (50.0)	7 (50.0)	14 (4.0)	.256	0.613
	No	191 (56.8)	145 (43.2)	336 (96.0)		
Obesity (BMI=>30)	Yes	55 (46.2)	64 (53.8)	119 (34.0)	7.866	0.005*
	No	143 (61.9)	88 (38.1)	231 (66.0)		
Lack Exercise	Yes	174 (61.1)	111 (38.9)	285 (81.4)	12.543	< 0.001*
	No	24 (36.9)	41 (63.1)	65 (18.6)		
	Total	**198 (56.6)**	**152 (43.4)**	**350 (100.0)**		

*. The Chi-square statistic is significant at the .05 level.

The bivariate analysis revealed multiple significant associations between demographic, clinical, and behavioral characteristics with pCAD. However, these associations are likely influenced by confounding factors, as many risk factors such as smoking, obesity, and physical inactivity are interrelated. For instance, obesity and lack of exercise may independently contribute to CAD while also being linked to other risk factors like hypertension or diabetes. Additionally, the observed associations may be affected by differences in demographic or lifestyle characteristics. To accurately determine the independent predictors of premature CAD and control for potential confounders, a multivariable logistic regression analysis were used as seems to be essential. This approach will allow for the adjustment of confounding variables, ensuring that the identified risk factors remain significant even after accounting for other influencing variables, ultimately providing a clearer understanding of the true determinants of premature CAD.

The improved stepwise multiple logistic regression final model with accuracy (predicts premature CAD more effectively) of 70.0%, sensitivity (correctly predicting premature CAD cases) of 63.2%, and specificity (correctly predicting non-premature CAD cases) is 75.3%, shows improvement (R2 = 33.8) with previously of (28.5%), and Good fit ($p > 0.05$), meaning model predictions match observed data well [Tables 4, and 5].

Table 4: Model Performance (Classification Tables & Model Summary)

Step	Correctly Predicted Premature CAD (%)	Correctly Predicted Non-Premature CAD (%)	Overall Accuracy (%)
Step 1	64.50%	64.10%	64.30%
Step 2	40.80%	88.90%	68.00%
Step 3	47.40%	85.40%	68.90%
Step 4	59.90%	76.80%	69.40%
Step 5 (Final Model)	63.20%	75.30%	70.00%

Table 5: Model Fit & Goodness-of-Fit Tests

Test	Value
Nagelkerke R^2	33.80%
Hosmer-Lemeshow Test (p = 0.422)	Good fit ($p > 0.05$), meaning model predictions match observed data well.

DISCUSSION

In this study, PCAD was more likely to be seen among females than males, while males were more likely to develop NPCAD. Additionally, Family history plays a significant role in the development process of the disease and its risk factors. Patients with positive family history have an approximately 1.5- to 2.0-fold higher risk of CHD independent of conventional risk factors[21]. Similarly, patients with PCAD had a slightly more contribution of family history compared to NPCAD cases. The role of genetics, through the family history, is well identified. Across a long-term follow-up, patients with a premature family history of CAD showed a significant association with a persistent increase in mortality of CHD[22, 23]. A family history of CHD is an independent risk factor for premature CHD[24]. Chacko and colleagues found a prevalence of 24%, and the risk increases linearly with the number of family members affected[24]. In this study, 54% of the cases had a positive family history of CAD and a statistically slightly more significant toward PCAD. Despite that, until now, Family history has not been added to the available cardiovascular (CV) risk scores (SCORE, ESC 2016-; PCE, AHA 2019-; Framingham Heart Study Risk Score and QRISK3, NICE 2023)[25]. Mechanisms of PCAD include inherited pathologies such as familial hypercholesterolemia, genetic factors that affect atherosclerosis development, inherited risk factors such as hypertension, and shared lifestyle such as physical inactivity[25].

As for the type of presentation, PCAD was more likely to present as NSTEMI. Furthermore, one of the significant modifiable risk factors associated with ACS was hypertension. Its presence alone with CAD can significantly elevate cardiovascular-related comorbidities such as diabetes mellitus and obesity and the risk of all-cause mortality[26]. In this study, nearly half of the cases of PCAD were hypertensive. Similarly, Li and Wang et al. also found a high prevalence of hypertension among the younger age groups[26]. This study shows that the presence of Hypertension is a significant factor for PCAD.

Former smokers, patients with diabetes, and obesity all were significantly correlated to PCAD. All of these factors were regarded as thrombotic and atherosclerotic with significant contribution to the development of CAD[27-30]. Another case-control study conducted in India supported the presence of both thrombotic and atherosclerotic risk factors that were significant among young populations[30]. Despite that, Hyperlipidemia is regarded as one of the significant risk factors of CAD[31], and lipid-lowering therapy is one of the key treatments among patients with CAD[32] in this study there was no significant difference between both groups indicating that hyperlipidemia doesn't affect the maturity of CAD plaque. However, further studies are required.

Many other factors contribute to the development of CAD, such as lifestyle and stress-related factors. In this study, alcohol[33] had no significant difference between both groups and this could be attributed to the community as the vast majority are non-alcohol drinkers. Lack of physical activity has been associated with the development of PCAD in our study and similar strong findings were seen for indoor activities. Both factors are among the significant modifiable risk factors of CAD and all-cause mortality [34]. This indicates that a sedentary lifestyle is one of the major contributing factors for early disease onset and PCAD.

Additionally, despite the complex and multifactorial relationship between stress and CAD[35], other psychological disturbances such as anxiety, and depression can play a role in the development of CAD[36-37]. Nevertheless, anxiety did not show a strong association

with PCAD, while Depression was significantly more common among NPCAD and was independently associated with high cardiovascular-related morbidity and mortality[37].

A salty diet is one of the contributing factors to CAD[38]. Presence of this habit at high percentages, 67.5% in our study, among young populations can indicate a major future health related consequence[39] starting form CAD and in our study, the correlation was significant with PCAD. As for over-eating, no significant difference can be seen. Despite that fatty diet was more significant among NPCAD.

Strength Points and Limitations

One of the limitations which was faced in this study it included only patients and there were no healthy matching group to be compared with. Another study is required to assess the patients with PCAD to healthy groups with matched age and gender. Additionally, a larger sample size is required. However, this represents a single center study. In this study all potential biases were avoided as much as possible.

Conclusion and Recommendation

Highly significant findings were seen and discussed in this study. Patients with PCAD have significant predictors which could indicate the high riskiness of the case. This indicates that younger females, those with a family history of CAD, NSTEMI type of ACS, D.M., Hypertension, Former smokers, and obesity, presence of anyone or more of these factors can lead significantly to PCAD, with P values < 0.05. Additionally, lifestyle and food factors such as lack of physical activity, in door activity, high salt intake and vegetarian diet were significantly corelated to PCAD. This study comes to the conclusion that a greater focus shall be placed on the premature CAD as well as the usual risk factors which contribute to the pathogenesis of the disease through awareness campaigns. It is recommended to starts a large scaled public awareness on the importance of avoiding risk factors of ACS among population generally and young adults specifically.

Conflict of Interest

None to Declare

Funding

None to declare

Clinical Trial Number

Not applicable

Ethical approval and Consent to Participate

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the General Directorate of Health-Duhok. The study has been given a reference number of (31012024-1-13). The study was adherent to the ethical standards of the Declaration of Helsinki and national guidelines. Consent were obtained from each patient prior of including their data.

Availability of data and materials

The raw data generated and analyzed during the current study are not publicly available. Only their final results are. However, they are ready to be shared with the Editor upon request, taking into consideration about patients' privacy.

Authors Contributions

M.H.M. is the only author of this article and has single-handedly taken the cases one by one to the final form. The whole article is done by him with some help of statistics from experts.

Acknowledgment

The author would like to thank all the staff of the Azadi-Heart Center for their help and support. Additionally, I would like to thank all the patients who have had participated in this study.

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هۆکارە مەترسیدارە ڕەفتاری و خێزانییەکان بۆ نەخۆشی زووەکانی شەریانی تاجی دڵ بەرامبەر نەخۆشی نازووەکان: توێژینەوەیCase-Control

پوختە

پێشەکی و ئارمانج: نەخۆشی زووەکانی شەریانی تاجی دڵ (pCAD) بووەتە کێشەیەکی گەورەی تەندروستی لە ئاستی جیهاندا، چونکە پەیوەندی هەیە بە هۆکارە مەترسیدارە گۆڕانکارەکان و مێژووی خێزانی. ئامانجی ئەم توێژینەوەیە دیارپکردنی هۆکارە مەترسیدارە ڕەفتاری و خێزانییەکان بوو کە بەشدارن لە زیادبوونی pCAD و بەراوردکردنیان لەگەڵ هۆکارەکانی نەخۆشی شەریانی تاجی دڵی نازووە.

ڕێکێن قەکولینێ: توێژینەوەیەکی Case-Control ماوەی ساڵێک ئەنجامدرا و 350 نەخۆش تێیدا بەشداریان کرد (198کەس بە pCAD و 152 کەس بە نەخۆشی شەریانی تاجی دڵی نازووە (لە ناوەندی دڵی ئازادی و ناوەندە پەیوەندیدارەکان pCAD .بەو شێوەیە پێناسە کرا کە نەخۆشییەکە پێش تەمەنی 45 ساڵ لە پیاوان و 55 ساڵ لە ژنان دەست پێ بکات. زانیارییە دیمۆگرافی، کلینیکی و شێوازی ژیان کۆکراوە و شیکردنەوەی ئاماری بە بەکارهێنانی بەرنامەی SPSS وەشانی 26 ئەنجامدرا، بە بەکارهێنانی تاقیکردنەوەیChi-square ، تاقیکردنەوەی-Mann Whitney U.و logistic regression بەهای p-value < 0.05 بە ماندار لە ڕووی ئامارییەوە هەژمار کرا.

ئەنجام: pCADبە شێوەیەکی ماندار پەیوەندی هەبوو بە ڕەگەزی مێ، مێژووی خێزانی ئەرێنی، دەرکەوتنی-Non STEMI، شەکرە، بەرزبونی فشاری خوێن، قەڵەوی و جگەرەکێشانی پێشوو .(p < 0.05) هەروەها هۆکارەکانی شێوازی ژیان وەک کەمی وەرزش، خواردنی زۆری خوێ، چالاکی ناومالی و ڕژێمی خواردنی ڕووەکی بە شێوەیەکی ماندار پەیوەندیان هەبوو بە .pCAD لە بەرامبەردا، خەمۆکی و ڕژێمی خواردنی پڕ لە چەوری زیاتر پەیوەندییان بە نەخۆشی شەریانی تاجی دڵی نازووەوە هەبوو. شیکردنەوەی logistic regression نیشانی دا کە ئەم هۆکارانە بە کۆی گشتی دەتوانن pCAD بە وردییەکی 70% پێشبینی بکەن. فرەچەوری خوێن، خواردنەوەی ئەلکۆل و دڵەڕاوکێ هیچ پەیوەندییەکی ماناداریان پیشان نەدا.

دەرئەنجام: ئەم توێژینەوەیە گرنگیی ڕۆڵی هەردوو هۆکارە ڕەفتاری و خێزانییەکان لە پەرەسەندنی pCAD نیشان دەدات. گرنگیدان بە ناسینەوەی زوو و گۆڕینی شێوازی ژیان لە تاکەکانی مەترسیدار — بە تایبەتی ژنانی گەنجتر — دەتوانێت بارگرانی نەخۆشی زووەکانی شەریانی تاجی دڵ کەم بکات.

عوامل الخطورة السلوكية والعائلية لمرض الشريان التاجي المبكر مقارنة بغير المبكر: دراسة حالات وشواهد

الخلاصة

الخلفية والأهداف: يعد مرض الشريان التاجي المبكر (pCAD) مصدر قلق صحي عالمي متزايد بسبب ارتباطه بعوامل خطورة قابلة للتعديل واستعداد عائلي. هدفت هذه الدراسة إلى تحديد عوامل الخطورة السلوكية والعائلية التي تسهم في انتشار مرض الشريان التاجي المبكر ومقارنتها بالعوامل المرتبطة بمرض الشريان التاجي غير المبكر.

الطرق: أجريت دراسة حالات وشواهد على مدى عام واحد، شملت 350 مريضا (198 مصابا بمرض الشريان التاجي المبكر و152 مصابا بمرض الشريان التاجي غير المبكر) من مركز آزادي للقلب والمراكز ذات الصلة. وتم تعريف مرض الشريان التاجي المبكر على أنه حدوث المرض قبل عمر 45 سنة لدى الذكور و55 سنة لدى الإناث. جمعت البيانات الديموغرافية والسريرية ونمط الحياة، وأجريت التحليلات الإحصائية باستخدام برنامج SPSS الإصدار 26، مع تطبيق اختبار كاي-تربيع، واختبار مان–ويتنيU، والانحدار اللوجستي. واعتبرت القيمة الاحتمالية (p-value) أقل من 0.05 ذات دلالة إحصائية.

النتائج: ارتبط مرض الشريان التاجي المبكر بشكل معنوي بالجنس الأنثوي، والتاريخ العائلي الإيجابي، وظهور المتلازمة التاجية غير المصحوبة بارتفاع مقطع ST (Non-STEMI)، وداء السكري، وارتفاع ضغط الدم، والسمنة، والتدخين السابق ($p < 0.05$). كما ارتبطت عوامل نمط الحياة مثل قلة ممارسة النشاط البدني، والإفراط في تناول الملح، والأنشطة الداخلية، والنظام الغذائي النباتي بشكل معنوي بمرض الشريان التاجي المبكر. في المقابل، كان الاكتئاب والنظام الغذائي الغني بالدهون أكثر ارتباطا بمرض الشريان التاجي غير المبكر. وأظهر تحليل الانحدار اللوجستي أن هذه العوامل مجتمعة تنبأت بمرض الشريان التاجي المبكر بدقة بلغت 70%. ولم يظهر كل من فرط شحميات الدم، واستهلاك الكحول، والقلق ارتباطا معنويا.

الاستنتاجات: تبرز هذه الدراسة الدور البارز لكل من العوامل السلوكية والعائلية في تطور مرض الشريان التاجي المبكر. وقد يسهم التركيز على التشخيص المبكر وتعديل نمط الحياة لدى الأفراد المعرضين للخطر - وخاصة الإناث الأصغر سنا - في تقليل عبء مرض الشريان التاجي المبكر.