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Association of Opium Addiction with Coronary Artery Ectasia and Coronary Artery Disease

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Original Article

Abstract

Background: Coronary artery ectasia (CAE) is a rare cardiovascular disorder with unknown mechanisms and related risk factors. The roles played by homocysteine in induction of cardiovascular diseases (CVDs) have also been documented previously. This project was designed to assess the relationship between opium and CAE and coronary artery disease (CAD).

Methods: This cross-sectional study was performed on 46 patients with CAE, 30 patients with CAD, and 42 cases without CAE and CAD (controls). Demographic data and information regarding opium consuming and also smoking were collected using a standard checklist. Serum levels of homocysteine, creatinine (Cr), urea, fasting blood glucose (FBG), low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglyceride (TG), and cholesterol were determined.

Findings: Statistical analysis revealed that opium consumers were significantly higher in patients with CAD and CAE when compared to healthy controls. Opium increased serum levels of Cr in the normal controls, and decreased HDL in the patients with CAD. Homocysteine serum levels were not significantly different between the groups.

Conclusion: The results of study showed that opium addiction was associated with increased risk of CAE and CAD, independent of homocysteine serum levels.

Keywords: Coronary artery disease; Opium; Homocysteine

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Introduction

Cardiovascular diseases (CVDs) are the main causes of the morbidity and mortality among Iranian population and also developed and developing countries.[1,2] The diseases are associated with several complications, including coronary artery ectasia (CAE), which play key roles in the induction of the cardiovascular-related morbidities and mortalities.[3] It has been demonstrated that several genetic and environmental factors play significant roles in the induction of CVDs, including CAE and coronary artery disease (CAD).[4-6] CAE, as a rare CVD-related complication, occurs in only 0.3%-4.9% of the patients.[7] The complication is characterized by the non-local coronary artery enlargement to 1.5 times or more, when compared to its normal diameter.[8] It is plausible in 0.22%-1.40% of angiographies and most of them are induced due to the atherosclerosis; however, some of them have the congenital or percutaneous coronary intervention (PCI) sources.[8] It has been hypothesized that CAE may increase the risk of acute myocardial infarction (MI).[7] However, some of the patients with CAD suffer from CAE,[9] implying that different mechanisms are involved in the induction of CAE.

Although the main causes of CAE are yet to be clarified, it has been proposed that inflammation and its related environmental inducers can stimulate the disease ranges.[10] It has been reported that opium is an inducer of inflammation and its significant association with CVDs has been documented by several investigators.[11-15] The roles played by opium in the pathogenesis of CAE are yet to be defined. Due to the relation between opium and CVDs and their related complications,[16] it has been hypothesized that opium may alter the risk of CAE. Therefore, this project was designed to investigate the prevalence of opium consumption in patients with CAD and CAE and possible association between opium consumption and the risk of CAD and CAE in the patients who were under angiography.

Additionally, it has also been documented that serum levels of homocysteine are elevated among the patients who are suffering from CVDs, and it also can be considered as a risk factor for induction of the diseases.[17] Nevertheless, its roles in the pathogenesis of CAE are yet to be clarified completely. Thus, another aim of this project was to explore the association between homocysteine serum levels and CAE/CAD among an Iranian population from Kerman Province, Iran.

Methods

Subjects: In this cross-sectional study, 46 patients with CAE, 30 patients with CAD, and 42 cases without CAE and CAD (controls) were evaluated regarding opium consumption and also serum levels of homocysteine. The participants were selected from the patients under angiography who referred to the Department of Cardiovascular Diseases, Shafa Hospital, Kerman. CAE was defined as dilatation of an arterial segment to a diameter at least 1.5 times that of the adjacent normal coronary artery.[18]

CAD was defined as more than 50% diameter stenosis of one or more major coronary artery. Patients without CAE and any coronary artery stenosis were determined as control group (normal coronary artery).

Opium consumption was diagnosed due to the patients' self-declaration.[19] In addition to the variables, sex, age, smoking, and history of CVDs were collected using a standard checklist. Body mass index (BMI) was calculated as weight (kg) divided by height in meters squared (m^2). All the patients who were under angiography and had CAE and CAD were entered to the study except the patients that suffered from other CVDs, heart failure (HF) with a left ventricular ejection fraction (LVEF) < 40%, atrial fibrillation (AF), a history of any revascularization, autoimmune diseases, cancer, splenectomy, active infectious diseases, alcohol drinking, drug administration, allergy, hypersensitivity disorders, and other systematic disorders. All the participants filled out the consent form and the Ethical Committee of Kerman University of Medical Sciences approved the protocol study (code: IR.KMU.AH.REC.1397.031). The study participants' characteristics, including demographic information, screening records, drug use, and clinical features/manifestations were recorded using a checklist.

Angiography: An expert Doctor of Medicine (MD) cardiologist performed the angiography based on the comparison of the damaged to normal vessels. The selective coronary angiography was carried out after local anesthesia using 6 French sheath and judkins catheters (left and right catheters). So, the contrast media

(Visipaque) was injected into the right and left coronary arteries directly, in multiple projections.

Biochemical measurements: High-performance liquid chromatography (HPLC) technique (KNAUER, Germany), coupled with fluorescence detector, was used to evaluate homocysteine serum levels. It was validated over a linearity range of 1-100 µmol/l with 4% and 6% intra-assay and inter-assay coefficient of variation (CV), respectively.

Low-density lipoprotein (LDL), high-density lipoprotein (HDL), fasting blood glucose (FBG), urea, creatinine (Cr), triglyceride (TG), and cholesterol were measured for the participants. To evaluate serum levels of these factors, 5 ml blood samples were collected in the tubes without anticoagulant agents and serums were separated and kept in -20 ^{o}C. Serum levels of FBG, urea, Cr, TG, LDL, and HDL were evaluated using commercial kits (Man Company, Tehran, Iran) according to the manufacturer instructions.

The SPSS software (version 20, IBM Corporation, Armonk, NY, USA) was used to analyse the raw data. Accordingly, for the normal distribution of the data, the groups were compared regarding the variables using the parametric tests. One-way analysis of variance (ANOVA) was used to analyse the differences between the groups regarding age, BMI, ejection fraction (EF), and serum levels of FBG, urea, Cr, TG, cholesterol, LDL, and HDL. To analyse the differences between the groups regarding gender and status of smoking and opium consuming, chi-square test was used.

Results

Data analysis demonstrated that although the groups were not different regarding smoking (P = 0.132), the opium consumers were significantly higher in the patients with CAE and CAD in comparison to the controls (P = 0.001). Table 1 shows the frequency of the cigarette and opium consumers in the case and control groups. Additionally, the one-way ANOVA test revealed that the groups were similar regarding age (P = 0.448), sex (P = 0.219), and BMI (P = 0.113). Table 1 illustrates the raw data regarding the variables.

The statistical analysis revealed that the patients were different regarding opium consumption significantly. Table 2 shows the data regarding EF, FBG, TG, cholesterol, LDL, HDL, urea, Cr, and homocysteine between three groups. The results showed that the serum levels of FBG (P = 0.308), urea (P = 0.430), Cr (P = 0.178), and homocysteine (P = 0.881) were not different between the groups, while serum levels of TG (P < 0.001), cholesterol (P < 0.001), and LDL (P < 0.001) were significantly increased in the CAE when compared to both CAD and control groups. HDL serum levels were significantly decreased in the CAD and CAE groups when compared to the controls (P = 0.024). EF (P < 0.001) was significantly decreased in the CAD in comparison to both CAE and control groups.

Table 1. Demographic data in the patients with coronary artery ectasia (CAE), coronary artery disease (CAD), and the patients without CAE and CAD (control)

Variables	Groups	Value		P
Age (year)	Control	55.97 ± 1.74		0.448
(mean ± SE)	CAE	55.00 ± 1.41		
	CAD	51.21 ± 1.57		
BMI (kg/m^2)	Control	25.33 ± 0.89		0.113
(mean ± SE)	CAE	26.60 ± 0.38		
	CAD	24.61 ± 0.62		
Sex (%)	Control	Male	50.0	0.219
	CAE	Male	54.3	
	CAD	Male	70.0	
Opium (%)	Control	Yes	26.2	0.001
	CAE	Yes	60.9*	
	CAD	Yes	66.7*	
Smoking (%)	Control	Yes	11.9	0.132
	CAE	Yes	26.1	
	CAD	Yes	13.4	

*P < 0.001 versus control group
SE: Standard error; BMI: Body mass index; CAE: Coronary artery ectasia; CAD: Coronary artery disease

The statistical analysis revealed that the patients were different regarding EF, TG, cholesterol, HDL, and LDL serum levels significantly. Table 3 shows the data regarding EF, FBG, TG, cholesterol, LDL, HDL, urea, Cr, BMI, and homocysteine between the smokers vs. non-smokers and opium consumers vs. non-consumers in participants. Statistical analysis revealed that smoking was associated with decreased EF and opium consumption was associated with decreased serum levels of HDL in the patients (Table 3). In addition, smoking and opium were associated with increased serum levels of Cr (Table 3).

The statistical analysis revealed that smoking and opium were associated with changes in EF, HDL, and Cr serum levels.

Table 2. Comparison of ejection fraction (EF), fasting blood glucose (FBG), triglyceride (TG), cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), urea, creatinine (Cr), body mass index (BMI), and homocysteine between control, coronary artery ectasia (CAE), and coronary artery disease (CAD) groups

Group	EF	FBG	TG	Cholesterol	LDL	HDL	Urea	Cr	Homocysteine
Control	55.97 ± 0.43	104.78 ± 3.66	168.92 ± 12.57	167.82 ± 5.23	92.30 ± 4.40	43.16 ± 1.78	30.26 ± 1.68	0.96 ± 0.03	9.67 ± 0.74
CAE	55.04 ± 0.55	106.27 ± 3.82	214.88± 10.92*	199.02 ± 7.89*	130.67 ± 6.98*	38.11 ± 1.09$^{\#}$	33.04 ± 1.61	1.01 ± 0.02	9.38 ± 0.91
CAD	51.21 ± 1.20†	97.75 ± 4.10	144.86 ± 13.03	165.33 ± 5.45	96.08 ± 4.33	39.33 ± 1.01$^{\#}$	33.29 ± 1.74	1.06 ± 0.03	9.12 ± 0.57
P	< 0.001	0.308	< 0.001	< 0.001	< 0.001	0.024	0.430	0.178	0.881

*P < 0.05 versus non-smokers, opium consumers, and non-consumers, $^{\#}$P < 0.05 versus control group, †P < 0.001 versus control and coronary artery ectasia (CAE) groups
CAE: Coronary artery ectasia; CAD: Coronary artery disease; EF: Ejection fraction; FBG: Fasting blood glucose; TG: Triglyceride; LDL: Low-density lipoprotein; HDL: High-density lipoprotein; Cr: Creatinine
Data are presented mean ± standard deviation (SD)

Table 3. Comparison of ejection fraction (EF), fasting blood glucose (FBG), urea, creatinine (Cr), triglyceride (TG), cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), body mass index (BMI), and homocysteine between the smokers vs. non-smokers and opium consumers vs. non-consumers in participants

		EF	FBG	TG	Cholesterol	LDL	HDL	Urea	Cr	Homocysteine
Smoking	Yes	46.22 ± 2.33*	91.30 ± 4.44	153.00 ± 15.53	157.33 ± 11.93	81.81 ± 4.16	45.11 ± 11.31	22.30 ± 11.50	1.11 ± 0.06†	25.53 ± 1.53
	No	53.13 ± 1.33	100.11 ± 22.03	162.37 ± 22.57	157.83 ± 5.38	83.83 ± 7.91	50.41 ± 9.97	28.66 ± 5.70	0.88 ± 0.02	23.53 ± 2.54
Opium	Yes	56.91 ± 0.88	96.18 ± 13.63	155.34 ± 11.69	171.56 ± 16.93	88.45 ± 7.90	44.44 ± 3.34$^{\#}$	32.23 ± 11.96	1.15 ± 0.05†	24.63 ± 1.33
	No	57.66 ± 0.48	101.21 ± 3.53	146.31 ± 19.30	158.12 ± 9.69	85.67 ± 6.42	56.00 ± 3.55	25.65 ± 13.82	0.81 ± 0.03	24.55 ± 1.63

*P < 0.05 versus non-smokers, opium consumers, and non-consumers, $^{\#}$P < 0.05 versus opium non-consumers, †P < 0.05 versus non-smokers and opium non-consumers
EF: Ejection fraction; TG: Triglyceride; LDL: Low-density lipoprotein; HDL: High-density lipoprotein; Cr: Creatinine; FBG: Fasting blood glucose; BMI: Body mass index
Data are presented mean ± standard deviation (SD)

Discussion

The results demonstrated that the prevalence of opium addiction was significantly higher in CAE and CAD groups compared to control group (Table 1). Due to the results, it appears that opium addiction increases the risk of CAE and CAD. As mentioned previously, opium is a risk factor for deterioration of CVDs.[16] Additionally, the roles played by opium in the cardiovascular-related complications, such as atherosclerosis and coronary microvascular dysfunction (CMD), have also been documented previously.[20] Our results also showed that there was an association between opium consumption and the risk of either CAE or CAD. The main mechanisms used by opium to increase the risk of the disease are yet to be clarified. Meanwhile, previous investigations revealed that homocysteine was a risk factor for CVDs.[21,22] The results of the current study demonstrated that homocysteine levels were not changed between cases and controls. Smoking and opium consumption also did not have significant effects on the serum levels of homocysteine. Thus, it may be concluded that opium can probably induce CAE or CAD in homocysteine independent pathways and it may alter other risk factors for the diseases. For example, it has been reported that opium can lead to CAD via up-regulation of plasminogen activator inhibitor (PAI).[23] In parallel with our results, Azdaki et al. revealed that opium had no effects on the homocysteine serum levels.[24] Although there are some investigations showing increased plasma/serum levels of homocysteine in the opium-addicted patients, their participants did not suffer from CVDs.[25,26] Therefore, it may be hypothesized that probably opium increases the risks of CAE and CAD incidence, but it did not induce the disorders via up-regulation of homocysteine.

The results showed that the smoking was associated with decrease in EF compared to non-smokers, opium consumers, and non-consumers. Besides, opium consumption was associated with decrease in HDL serum levels when compared to non-addicted patients. Therefore, it may be hypothesized that opium may increase the risk of CAD indirectly via down-regulation of HDL-related molecules, which needs to be explored by further investigations. Moreover, opium and smoking may be considered as risk factors for other disorders including kidney disease, as the results demonstrated that smokers and opium consumers were associated with higher levels of Cr than non-smokers and non-opium consumers. Consistent with these results, other studies have shown that opium and smoking are associated with impaired renal function and elevated serum Cr levels.[27-30]

Collectively, it appears that opium and smoking are two important risk factors for deterioration of CAE and CAD, independent of homocysteine. Additionally, it appears that smoking and opium may be considered as risk factors for kidney diseases.

Conclusion

Due to the results, it may be concluded that there is an association between opium consumption and smoking with the risk of CVDs (including CAD and CAE), independent of homocysteine and kidney diseases incidence.

Conflict of Interests

The Authors have no conflict of interest.

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Authors' Contribution

Data curation, data analysis, funding, investigation, methodology, software, supervision, writing-original draft, review and editing: NB; conceptualization, data curation, funding, investigation, methodology, project administration, resources, writing-review and editing: GA; conceptualization, data curation, funding acquisition, investigation, methodology, project administration, supervision, resources, writing–reviewing and editing: MM.

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بررسی ارتباط مصرف تریاک با اکتازی عروق کرونر و بیماری عروق کرونر

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مقاله پژوهشی

چکیده

مقدمه: اکتازی عروق کرونر (Coronary artery ectasia یا CAE) یک بیماری نادر قلبی و عروقی با مکانیسم ناشناخته و عوامل خطر مرتبط می‌باشد. نقش هموسیستئین در ایجاد بیماری‌های قلبی- عروقی پیش‌تر ثابت شده است. پژوهش حاضر به منظور بررسی ارتباط بین مصرف تریاک، اکتازی عروق کرونر و بیماری عروق کرونر (Coronary artery disease یا CAD) طراحی شد.

روش‌ها: این مطالعه مقطعی بر روی ۴۶ بیمار مبتلا به CAE، ۳۰ بیمار مبتلا به CAD و ۴۲ بیمار بدون CAE و CAD انجام شد. یافته‌های دموگرافیک و اطلاعات مرتبط با مصرف تریاک و سیگار بر اساس چک‌لیست استاندارد جمع‌آوری گردید. سطح سرمی هموسیستئین، کراتینین (Creatinine یا Cr)، اوره، قند خون ناشتا (Fasting blood glucose یا FBG)، Low density lipoprotein (LDL)، تری‌گلیسرید (Triglyceride یا TG)، High density lipoprotein (HDL) و کلسترول محاسبه شد.

یافته‌ها: مصرف‌کنندگان تریاک به صورت قابل توجهی بیشتر به CAE و CAD مبتلا بودند. تریاک، سطح سرمی Cr را در افراد طبیعی افزایش داد و باعث کاهش HDL در بیماران مبتلا به CAD شد. سطح سرمی هموسیستئین تفاوت قابل توجهی بین گروه‌ها نداشت.

نتیجه‌گیری: مصرف تریاک بدون ارتباط با سطح سرمی هموسیستئین، با افزایش خطر CAE و CAD همراه می‌باشد.

واژگان کلیدی: بیماری عروق کرونر؛ تریاک؛ هموسیستئین

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