Med. J. Cairo Univ., Vol. 91, No. 4, December: 1479-1486, 2023
www.medicaljournalofcairouniversity.com

Effect of Smoking in Young Adults on Angiographic Pattern of Coronary Artery Lesions

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Abstract

Background: Coronary artery disease (CAD) is the most prevalent group of cardiovascular disorders. There is a well-established link between smoking and atherosclerosis.

Aim of Study: To describe the coronary angiographic pattern of lesions among smokers compared with nonsmokers.

Patients and Methods: This descriptive observational comparative study involved 150 participants. Patients were divided into two groups, 75 participants in smoking group with history of cigarette smoking of one packet and more and 75 participants in non-smoking group including patients who had never smoked cigarettes or abandoned smoking more than 5 years. Each patient included in our study was subjected to full history taking, thorough physical examination, standard laboratory investigations, electrocardiographic examination, transthoracic echocardiography (TTE) and invasive coronary arteriography. All coronary angiograms included anatomical description of lesions site.

Results: The smokers group had 62.7% of the participants showing significant lesions, and a 37.3% with non-significant lesions. The non-smoking group had 42.7% of its participants with significant lesions compared and 57.3% with non-significant lesions. There was a significant difference between smokers and non-smokers patients regarding significance of coronary lesions. The diameter of stenosis between the two groups was compared, and was statistically different. Multivariate regression analysis determined the possible factors associated with CAD and increased affected arteries. We found that BMI, obesity, smoking, total cholesterol, LDL, EF, and FS were significant factors associated with CAD and number of affected arteries.

Conclusions: The following factors are associated with CAD and increased number of affected arteries: BMI, morbid obesity, smoking, total cholesterol, LDL, EF, and FS.

Clinical Implications: The clinical implication of our study stresses on risk factor modification with strong emphasis on quitting smoking, increasing physical activity and attempts at weight optimization in obese patients to reduce the burden of disease and early coronary intervention to manage these high-risk CAD patients Smoking cessation programs are necessary. Evidence shows risk reduction upon medical therapy and smoking cessation in atherosclerotic arterial diseases.

Key Words: *Coronary artery disease - Smoking — Cardiovascular system — Angiography — Atherosclerosis.*

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Introduction

CORONARY artery disease (CAD) is the most prevalent group of cardiovascular disorders. Over the last century; many factors have been studied and presumed of risk. There is a well-established link between smoking and atherosclerosis, a pathologic process responsible for wide variety of systemic arterial ischemic disorders including coronary artery disease. Smoking substantially increases the risk of myocardial infarction, sudden cardiac death, stroke, peripheral vascular disease, and aortic aneurysms [1].

English et al. **[2]** reported an association between cigarette smoking and coronary atherosclerosis; as it does not only increase the risk of developing coronary heart disease but also increases morbidity and mortality in patients with coronary artery lesions.

Smoking affects the vascular structure and function by various mechanisms The suggested mechanisms are endothelial dysfunction, acceleration of plaque formation, and increase of platelet aggregation **[3].** A smoker's lipid profile shows higher total and LDL cholesterol levels and lower HDL cholesterol levels than control subjects; a lipid profile that favors initiation and progression of the atherosclerotic process **[4].**

Logical assumption suggests that coronary artery lesions should progress more rapidly in smokers than in nonsmokers; however, multiple studies that used serial coronary angiography; which was

performed mainly for clinical indications, smoking was not associated with an increased rate of coronary progression **[5].**

Later studies amended these conclusions. Waters et al. **[6],** in a study that used serial quantitative assessment of the angiographic evolution of coronary atherosclerosis, concluded that smoking accelerates coronary progression and new lesion formation.

Waters et al. **[6]** disclosed that Lovastatin slows the progression of coronary atherosclerosis and prevents the development of new coronary lesions in smokers.

Scant amount of data were to found in literature describing that smoking exhibits predilection in the anatomic distribution of coronary lesions. Zwaag et al. **[7]** concluded that smoking increased the risk of coronary lesions in all coronary segments with higher predilection to the right coronary artery.

This study is designed to focus on the effect of smoking on the pattern of coronary atherosclerosis distribution.

Patients and Methods

This descriptive observational comparative study was conducted in the Department of Cardiovascular Medicine Department, Specialized Medical Hospital, Faculty of Medicine, Mansoura University, in the period from January 2022 to December 2022.

The study was conducted on 150 patients aged 18-60 years old who were admitted and underwent invasive coronary angiography. The patients were divided into two groups, 75 participants in the smoking group with history of current cigarette smoking of at least one packet and more and 75 participants in non-smoking group including patients who had never smoked cigarettes.

Exclusion criteria:

- Patients under 18 years of age or over 60.
- Patients with congenital heart defects.
- Patients with unclear or missing data of smoking.
- Patients with moderate to high-grade valvular lesions.
- Patients with primary cardiomyopathy.

Methods:

All patients were subjected to full history taking, thorough physical examination and standard laboratory investigations.

Coronary angiography was performed by a femoral approach using the modified Seldinger technique. Standardized angiographic projections were chosen for the assessment of each arterial segment. Coronary angiograms were visually assessed by two independent observers blinded to the identity and clinical characteristics of the patients.

Standard 12-lead ECG was done to all patients using a paper speed of 25mm/s and standardization of lmV/lOmm

TTE was performed with special attention to left ventricle systolic function, resting segmental wall motion abnormalities, diastolic function and assessment of valvular condition.

All patients underwent invasive coronary angiography using standard technique. Views may be modified according to each patient to ensure complete study of the coronary anatomy without foreshortening or overlapping.

All coronary angiograms reports will include anatomical description of lesions site if present. The qualitative evaluations based on the visual estimation of the lesion and each coronary segment in at least two angiographic views and it depends very much on the operator's experience.

Description with include classifications according to number, site, lesion description and significance of a lesion.

All patients undergoing coronary arteriography were tracked by the study nurses until they was either enrolled in the trial or declared ineligible. The most common reasons for exclusion were failure to meet inclusion/exclusion criteria including age, CAD as an indication for coronary aniography, being diagnosed with complex congenital heart disease, severe valvular lesion, or primary cardiomypathy.

Statistical analysis:

Data were tested for normal distribution using the Shapiro Walk test. Qualitative data were represented as frequencies and relative percentages. Chi square test (x2) and Fisher exact was used to calculate difference between qualitative variables as indicated. Quantitative data were expressed as mean ± SD (Standard deviation) and range. Independent t-test and Mann Whitney test were used to calculate difference between quantitative variables in two groups for parametric and non-parametric variables respectively.

All statistical comparisons were two tailed with significance Level of p-value 4:1.05 indicates significant, $p<0.001$ indicates highly significant difference, while $p>0.05$ indicates non-significant difference.

Appropriate charts were used to graphically present the results whenever needed.

Results

Table (1) showed that there is significant difference between smokers and non-smoker patients regarding sex.

Table (2) showed that there is a significant difference between the two studied groups regarding heart rate.

Table (3) showed that there was a significant difference regarding total cholesterol and LDL that they were significantly higher among smokers compared to non-smokers.

Table (4) that there is significant difference between smokers and non-smoker patients regarding EF, FS, LVEDd, LVESd, and E/A ratio.

Table (5) showed that there is a significant difference between smokers and non-smokers patients regarding significance of coronary lesions.

Table (6) showed that diameter stenosis and calcification frequency were significantly higher among smokers compared to non-smoker patients.

Table (7) showed that **BMI,** obesity, smoking, total cholesterol, LDL, EF, and FS were found to be significant factors associated with CAD and number of affected arteries.

Table (1): Demographic data distribution between the two studied groups.

Variable	Smoker (n=75)	Non-smoker (n=75)	t/x^2	*P*
Age (years):				
Mean ± SD	53.64±10.33	50.8±9.68	1.74	.084
Sex:				
Male	72 (96%)	47 (62.7%)	25	<0.001
Female	3 (4%)	28 (37.3%)		
BMI (kg/m²):				
Mean ± SD	26.78±2.67	26.45±3.55	.643	.521

Table (2): Vital signs between the two studied groups.

Variables	Smoker (n=75)	Non-smoker (n=75)	*t*	*p*
HR (beat/min):				
Mean ± SD	86.6±10.41	79.5±11.58	3.95	.001
SBP (mmHg):				
Mean ± SD	127.8±7.63	125.4±8.65	1.8	.074
DBP (mmHg):				
Mean ± SD	82.2±5.64	81.34±5.32	.961	.338

Table (3): Laboratory parameters between the two studied groups.

Variables	Smoker (n=75)	Non-smoker (n=75)	t	*P*
Hemoglobin (g/dl):				
Mean± SD	10.52±1.64	10.74±1.38	.889	.376
Total Cholesterol (mg/dl):				
Mean± SD	204.65±30.5	191.8± 29.42	3.48	.007
Triglycerides (mg/dl):				
Mean± SD	111.8±16.81	108.27±18.49	1.22	.223
LDL (mg/dl):				
Mean± SD	103.14±15.22	97.44±14.28	2.37	.019
HDL (mg/dl):				
Mean± SD	46.81±7.95	48.9±6.43	1.77	.079
RBS (mg/dl):				
Mean± SD	123.33±15.9	119.63±16.88	1.38	.169
Creatinine (mg/dl):				
Mean± SD	0.879±0.167	0.837±0.159	1.58	.117
ALT (UIL):				
Mean± SD	28.97±6.07	28.37±5.76	.621	.536
AST (UIL):				
Mean± SD	28.48±7.34	27.65±6.33	.742	.460
Albumin (gldl):				
Mean± SD	4.11±0.555	4.15±0.534	.450	.653

Table (4): Transthoracic echocardiographic parameters between the two studied groups.

Variable	Smoker (n=75)	Non-smoker (n=75)	*t*	*p*
EF (%):				
Mean ± SD	48.79±6.67	54.13±8.22	4.37	<0.001
FS (%):				
Mean ± SD	31.5±5.35	35.74±4.15	5.42	<0.001
LVEDD (mm):				
Mean ± SD	48.78±5.72	43.57±5.29	5.79	<0.001
LVESD (mm):				
Mean ± SD	32.6±5.01	29.49±4.62	3.95	<0.001
EIA ratio:				
Mean ± SD	1.41±0.531	1.27±0.341	2.26	.042

Table (5): Angiographic lesion characteristics distribution between the two studied groups.

	Smoker (n=75		Non-smoker (n=75)		X^2	*p*
	N	%	N	%		
Length:						
Diffuse disease	29	38.6	28	373	.962	327
Tubular lesion	16	21.3	13	173		
Discrete lesion	35	46.6	34	453		
Significance:						
Significant CAD	47	62.7	32	42.7	6.1	.014
Non-significant CAD	28	37.3	43	573		

Table (6): Angiographic characteristics between the two studied groups.

Variable	Smoker (n=75)	Non-smoker (n=75)	*t*	*P*
Lesion length (mm):				
Mean ± SD	1137±4.56	10.81±4.19	.783	.435
Diameter stenosis (%):				
Mean ± SD	69.44±12.53	64.91±11.27	23	.021
Calcification	23 (30.7%)	12 (16%)	4.51	.034

Table (7): Multivariate regression analysis to determine the possible factors associated with CAD and increased affected arteries.

	OR	Sig.	95% CI
Age	.977	.337	.932 -1.025
Male gender	12.15	.273	.139 -16.79
BMI	1 214	.027*	1.054 -1397
DM	.573	.666	.046 - 7.167
HTN	.967	.523	.870 - 1.072
Morbid obesity	1 267	.009*	.951 - 1.688
Smoking	1257	.001 *	1.019 - 1.550
Total cholesterol	.574	.012	.026 - .681
LDL	.479	.028	.143 - 1.601
EF	1.588	.033*	1.002 - 2.517
FS	1 295	.011 *	.836 -1387

Discussion

Owing to the increasing number of deaths from non-communicable diseases, especially in developing countries, the World Health Organization has declared non-communicable diseases among the health priorities for the next two decades. Among these, cardiovascular disease is recognized as the most important and leading cause of death in many countries, seen in 48% of the population in the United States (US) **[8].**

The overall percentage of deaths from cardiovascular disease during the 20th century rose from 10% to 30%. The main cause of cardiovascular disease is reported to be coronary artery disease (CAD), with 2 out of 10 deaths being associated with CAD, and CAD-related deaths are expected to rise from 17.3 million in 2012 to 23.6 million in 2030. The prevalence of cardiovascular disease is rapidly increasing in developing countries [9].

The coronary arteries, which originate in the aorta and immediately above the aortic valve, are responsible for supplying blood to the heart muscle [10].

Coronary angiography refers to radiographic observation of the coronary arteries after contrast injection. In addition to coronary angiography, a complete invasive examination of the patient's cardiovascular condition involves examination of the cardiac cavities and hemodynamic evaluation. It is the gold standard method for diagnosing CAD. CAD is mostly caused by smoking and physical inactivity, but high blood pressure, dyslipidemia, and some factors related to ethnicity also lead to CAD [11].

Smoking is the single most important risk factor for coronary artery disease and the smoking has a particularly large impact in the developing world and annually accounts for 1.17 million deaths worldwide. Even among nonsmokers, inhaled smoke, whether from passive exposure or from cigar or pipe consumption, increases coronary risk.

Smoking affects atherothrombosis by several mechanisms:

Unfavorable effects on blood pressure and sympathetic tone with a reduction in myocardial oxygen supply.

- Long-term smoking may enhance oxidation of low-density lipoprotein (LDL) cholesterol.
- Impair endothelium-dependent coronary artery vasodilation.
- Smoking has adverse haemostatic and inflammatory effects, including increased levels of CRP, soluble intercellular adhesion molecule-1 (ICAM-1), fibrinogen, and homocysteine.
- Smoking is associated with spontaneous platelet aggregation, increased monocyte adhesion to endothelial cells, and adverse alterations in endothelial derived fibrinolytic and antithrombotic factors, including tissue-type plasminogen activator and tissue pathway factor inhibitor **[12].**

Smoking plays a major role in premature coronary atherosclerosis and in accelerating atherosclerosis by increasing the oxidation of low-density lipoprotein (LDL) and damaging coronary endothelial vasodilation. In addition to causing CAD, premature and accelerated coronary atherosclerosis are

determinants of the severity and extent of vascular occlusion. Understanding the patient's pattern and severity of vascular occlusion is important because these factors determine the choice of appropriate treatment and the success of treatment **[13].**

Given the rapid increase in CAD and the widespread use of tobacco products, including cigarettes, understanding the importance of smoking and its effects on the pattern and severity of CAD is critical. Also, the effect of smoking on the distribution of coronary atherosclerosis as assessed by serial angiography is poorly defined.

Therefore, the objective of this descriptive observational comparative study is to describe the coronary angiographic pattern of lesions among smokers compared with nonsmokers. The study involved 150 participants. The patients were divided into two groups, 75 participants in smoking group with history of cigarette smoking of one packet and more and 75 participants in non-smoking group being patients who had never smoked cigarettes.

Each patient included in our study was subjected to full history taking, thorough physical examination, standard laboratory investigations (CBC, random blood glucose, kidney function tests and lipid profile), coronary angiography, electrocardiographic examination, transthoracic echocardiography (TTE) and invasive coronary arteriography. All coronary angiograms included anatomical description of lesions site if present. Description with include classifications according to number and site.

Our study showed that there were 72 (96%) male smokers and 47 (62.7%) males in non-smoking group ($p<0.001$). Ahmed et al. **[14]** examined the pattern of coronary atherosclerosis between smokers and non-smokers. There were 33 (17%) female smokers and 58 (26%) females in non-smoking group (p-value 0.034).

Among smokers, 41.3% had hypertension compared to 28% in non-smokers. Diabetes mellitus was present in 21.3% of smokers and 26.7% of non-smokers. 40% of smokers had dyslipidemia compared to 29.3% in non-smokers, 46.7% of smokers were obese compared to 42.7% of non-smokers, 20% of smokers had family history of CAD compared to 18.7% of non-smokers. Statistically, there is no significant difference between smokers and non-smoker patients regarding co-morbidities associated with CAD.

Ahmed et al. [14] found that smokers are more likely to have significant CAD inthe absence of major coronary risk factors. Among smokers 28.8% had dyslipidemia compared to 32.9% in non-smokers. Hypertension was present in 43.4% of smokers and 53.2% of non-smokers. There were nearly equal percentages of diabetics, 31.55% and 31.9% in smokers and non-smokers respectively. Among the risk factors hypertension was more common in both categories.

Our study showed that there is no significant difference between smokers and non-smokers patients regarding affected arteries. We reported that LAD was involved in 25.3%, LCX in 6.7% and RCA in 8% in smokers. LAD artery was involved in 21.3%, LCX in 6.7% and RCA in 9.3% in non-smokers. The LAD was the more affected artery in both groups. Ahmed et al. **[14]** found that among smokers LAD was involved in 71.7%, LCx in 56% and RCA in 59.2%.

The discrepancy between our results and that of others could be explained by the small sample size of our study and the varied demographic traits, including risk factors of CAD.

In our study, single vessel disease was seen in 40%, two-vessel disease was seen in 33.3% and triple vessel disease in 26.7% of smokers. On the other hand, non-smokers had single vessel disease in 37.3%, two-vessel disease in 37.3% and triple vessel disease in 25.3%. The p-value is statistically not significant.

Fakhir Nafakhi **[15]** studied the effect of smoking on angiographic results in Iraqi patients with history of coronary artery disease submitted to routine coronary angiography. They illustrated the distribution of occlusive lesion in the 4 major coronary arteries between smoker and non-smoker groups and reported that the RCA and left main stem (58% and 57%) were more likely to be diseased in smoking group rather than non-smoker.

We demonstrated that the prevalence of double vessels was less common in smoking group compared to non-smoking group (33.3% versus 37.3%), but the prevalence of triple vessels was more common in smoking group compared to non-smoking group (26.7% versus 25.3%). Fakhir Nafakhi **[15]** found that the prevalence of two vessel disease (63% vs. 37%) ($p< 0.02$) and to lesser extent the three vessel disease pattern (53% vs. 47%) was more common in smoking group compared to non-smoking group with normal coronary arteries more in non-smokers group (35% vs. 65%).

Apart from coronary distribution, our description of the lesions found in the two studied groups was done mainly according to their length, significance and calcification.

38.6% were found to have diffuse disease, 21.3% were found to have tubular lesions, and 46.6% were found to have discrete lesions in the smoking group. The non-smoking group had 37.3% of the patients with diffuse disease, 17.3% with tubular lesions, and 45.3% with discrete lesions. The mean length

of the lesions was 11.37±4.56mm in the smoking group, compared to 10.81±4 19mm in non-smoking group, showing no statistical difference between the two groups regarding lesion length.

As for lesions significance, the smokers group had 62.7% of its patients' with meeting the definition of a significant lesion, and a 37.3% had non-significant lesions. In contrast, 42.7% of the non-smokers group had significant lesions compared and 57.3% with non-significant lesions. There was a significant difference (p-value=.014) between smokers and non-smokers patients regarding significance of coronary lesions. When comparing the diameter of stenosis between the two groups, the mean diameter stenosis in the smoking group was 69.44±12.53% compared to 64.91±11.27% in the non-smoking group, showing statistical difference (p-value= .021).

30.7% of the smoking patients had calcific lesions, compared to only 16% of the non-smoking patients. This demonstrates a significantly higher rate of calcification among smokers (p-value=.034).

Linking smoking to arterial calcification have been established in multiple studies. The association of smoking with vascular calcification have been demonstrated by radiological calcium scoring with a conclusion that smoking for a high number of pack-years contributes to coronary artery and aortic calcification **[16].**

Hirooka et al. **[17]** concluded that smokers have higher tendency to develop coronary artery calcification much earlier than never-smokers.

The suggested mechanism is that nicotine promotes vascular calcification via intracellular Ca2+-mediated, Nox5-induced oxidative stress, and extracellular vesicle release in vascular smooth muscle cells **[18].**

Multivariate regression analysis determined the possible factors associated with CAD and increased affected arteries. We found that BMI, obesity, smoking, total cholesterol, LDL, EF, and FS were significant factors associated with CAD and number of affected arteries.

In a series of 313 patients who had two coronary arteriograms mainly for clinical indications, current smoking was independently associated with more new coronary occlusion not with other types of progression. Smoking causes endothelial dysfunction, increased platelet aggregability, and increases the risk of myocardial infarction **[3].**

The results of our study clarified the evidence that smoking is associated with the severity of CAD and showed that smoking was associated with occlusion of the LAD artery and that smokers may be more likely to have non-proximal coronary artery occlusion. The reason for this association may be owing to the effect of cigarettes and nicotine on the vascular epithelium; under the influence of nicotine, the coronary vascular epithelium can become damaged. Smoking also increases sympathetic tone and causes vasospasm.

Salehi et al. **[19]** summarized evidence regarding the effect of smoking on the number of occluded coronary arteries and the severity and pattern of CAD. They found smoking to be related to CAD severity. However, there was no significant association of smoking with the number of damaged arteries and location of arterial occlusion.

The strength of the study:

The strength of the study lies in the efficiency of the data acquisition and the use of different tools and scales in order to achieve the aims of the study. Our study is directed at studying the pattern of coronary atherosclerosis in smokers; and to establish whether it shows special pattern of artery and lesion predilection compared to non-smokers. Furthermore, our study included various predictors of significant and insignificant coronary artery tortuosity. Our study supports the evidence of smoking as major risk factor for significant CAD, and supports prior evidence in literature of no major difference in lesion distribution compared to non-smokers.

Limitations of the study:

Despite the promising outcomes in the current study, the following limitations must be taken into-consideration:

First, Our study's small sample size (150 cases) limits its generalizability even though our results were significant with excellent discriminatory power. Hence a definite conclusion cannot be made based on this study alone. A large number of cases has to be studied to confirm our findings.

Second, The fact that we did not do extensive assessment scores to show further comparative features.

The discrepancy between our results and that of others could be explained by the small sample size of our study and the varied demographic traits, including risk factors of CAD.

Conclusion:

In conclusion, the current study showed:

1- LAD was the artery most involved in the smoking group.
2- Significant coronary lesions were more prevalent in smoking group.
3- The following factors are associated with CAD and increased number of affected arteries: BMI, morbid obesity, smoking, total cholesterol, LDL, EF, and FS.

Declarations:

Ethics approval and consent to participate: The study protocol was approved by Faculty of Medicine, Mansoura University and an informed consent was obtained from every participant in the study.

Consent for publication: I am the single author for this research article, and I give my consent to publish it.

Availability of data and material: All data and materials are available when requested.

Competing interests: No competing interests.

Funding: Self-funded.

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تأثير التدخين على أنماط آفات الشرايين التاجية في التصوير الوعائى عند شباب البالغين

تمهيد: أمراض الشريان التاجى هى المجموعة الأكثر انتشارًا ضمن أمراض القلب والأوعية الدموية. هناك علاقة راسخة بين التدخين وتصلب الشرايين التاجية. التدخين يسبب خلل فى بطانة الأوعية الدموية عن طريق آليات مختلفة.

المرضى وطرق البحث: الدراسة عبارة عن دراسة وصفية مقارنة أجريت فى قسم طب القلب والأوعية الدموية بالمستشفى الطبى التخصصى بكلية الطب جامعة المنصورة فى الفترة من يناير ٢٠٢٢ إلى ديسمبر ٢٠٢٢. وقد أجريت الدراسة على ١٥٠ مريضًا تم إدخالهم إلى المستشفى وخضع لتصوير الأوعية التاجية. تم تقسيم المرضى إلى مجموعتين، ٧٥ مشاركاً فى مجموعة المدخنين و٧٥ مشاركاً فى مجموعة غير المدخنين.

النتائج: أظهرت نتائج الدراسة أنه لا يوجد فرق احصائيا بين المرضى المدخنين وغير المدخنين فيما يتعلق بتوزيع الشرايين المصابة. كان الشريان التاجى الأمامى النازل هو الشريان الأكثر إصابة عند المدخنين. وكان انتشار إصابة الأوعية الثلاثية أكثر شيوعا في مجموعة المدخنين مقارنة بمجموعة غير المدخنين (٢٦,٧٪ مقابل ٢٥,٣٪). وكان مقدار تضيق آفات الشريان التاجى أعلى فى آفات المدخنين. هناك معدل أعلى إحصائيا لتكلس الشريان التاجي بين المدخنين. ترتبط العوامل التالية بمرض الشريان التاجى وزيادة عدد الشرايين المصابة: مؤشر كتلة الجسم، والسمنة المرضية، والتدخين، والكوليسترول الكلى، والكوليسترول منخفض الكثافة والكفاءة الانقباضية لعضلة القلب.

الاستنتاج: لا يختلف توزيع الشرايين التاجية بشكل كبير لدى المدخنين عن غيرهم؛ إلا أن نسبة حدوث تضيق الآفات وتكلسها أعلى.