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Relation between Smoking as a Single Cardiovascular Risk Factor and Angiographic Data in Young Patients with Acute Coronary Syndrome: A Retrospective Single-Center Study

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Abstract

Background: Smoking is widely recognized as a significant risk factor for acute coronary syndrome (ACS), not only in young patients but across the general population. The risk is further heightened by the use of other smoking products such as cannabis and water pipe smoking (hookah).

Aim of Study: The aim of this study is to evaluate the association between smoking, as an independent risk factor for cardiovascular disease, and angiographic findings in young individuals diagnosed with acute coronary syndrome.

Patients and Methods: This retrospective study involved a cohort of one hundred young patients who were smokers and presented with ACS, including both ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI), at Alexandria University Hospitals over a thirty nine-monthperiod. The smoking index was calculated to classify the smokers.

Results: This research involved 100 patients diagnosed with ACS with a history of smoking, most of whom were male (98%), with a mean age of 38.8±4.5 years. A high smoking index was found in 65% of the patients, who developed STEMI. Regarding the type of smoking, 70% of patients smoked cigarettes only, 7% smoked cigarettes with cannabis, 15% smoked cigarettes with water pipe smoking (hookah) and finally, 8% of patients smoked cigarettes with both cannabis and water pipe smoking (hookah). Patients with a high smoking index exhibited a higher prevalence of ACS. STEMI was predominant, accounting for 82% of cases, while NSTEMI constituted 12%. Angiographic data revealed that 94% of patients underwent stent placement, while 6% had normal coronary angiography. Among the diseased vessels, single vessel disease was more common compared to multivessel disease, with the left anterior descending (LAD) artery being affected in 51% of cases. A statistically significant association was observed between smoking index and the type of myocardial infarction (MI) (p=0.012).

Conclusion: Cigarette smoking is a major contributing factor in the pathogenesis of ACS. The risk of ACS is further elevated when cigarette smoking is combined with the use of water pipe smoking and cannabis. Patients with a higher smoking index exhibited greater severity of ACS than those with a lower smoking index. Therefore, calculating the smoking index is crucial for classifying patients as mild, moderate, or heavy smokers.

Key Words: *Smoking – Acute coronary syndrome (ACS) – Percutaneous coronary intervention (PCI) – Angiographic findings.*

Introduction

SMOKING plays a critical role as a cardiovascular disease (CVD) risk factor, often linked with unstable plaques that lead to ACS **[1,2].** An epidemiologi-

Abbreviations and Acronyms:

ACS : Acute coronary syndrome.
AMI : Acute myocardial infarction.
CAD : Coronary artery disease.
CPD : Cigarette per day.
CS : Cigarette smoking.
CVD : Cardiovascular disease.
ECG : Electrocardiogram.
HDL : High density lipoprotein.
IRA : Infarct related artery.
LAD : Left descending artery.
LCX : Left circumflex.
LDL : Low density lipoprotein.
LMCA : Left main coronary artery.
MI : Myocardial infarction.
MMP : Matrix metalloproteinase.
NSTEMI : Non-ST elevation myocardial infarction.
NYHA : New York Heart association.
PCI : Percutaneous coronary intervention.
PPCI : Primary percutaneous coronary intervention.
RCA : Right coronary artery.
SI : Smoking index.
STEMI : ST elevation myocardial infarction.
TC : Total cholesterol.
TG : Triglyceride.
TIMI : Thrombolysis in myocardial infarction.

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cal study of patients with ACS found that 17% were ex-smokers and 37.1% were active smokers, together accounting for nearly 54% of all ACS cases [3]. In a study conducted in Egypt, young patients with STEMI exhibited a significantly higher proportion of heavy smoking at 72.4% compared to those over 45 years of age [4]. Multiple studies have investigated the prevalence of cardiovascular risk factors across different populations, but results have been inconsistent because of the multifactorial nature of coronary artery disease (CAD). Risk factors include family history, age, hypertension, diabetes mellitus, gender, genetic factors, high blood cholesterol, and smoking [5-7]. Modifiable risk factors, including smoking, account for approximately 70% of the risk for CVD [8].

A significant number of young patients suffering from MI have a high prevalence of smoking and are predominantly male compared to older counterparts [9]. Young patients with ACS tend to smoke more cigarettes per day than older patients but have a lower pack-year history due to their younger age [10]. Studies have also revealed an association between cannabis use and MI [11], indicating a five-fold increase in MI risk within the first hour after cannabis use [11,12]. Persistent water pipe smoking (hookah) use is related to adverse cardiovascular outcomes. Numerous harmful substances found in cigarette smoke are also present in water pipe smoke, often at higher levels than in cigarette smoke [13].

Pathophysiology of acute coronary syndrome in different types of smoking:

The cardiovascular effects of cigarette smoking:

While the exact pathogenesis of smoking-induced atherosclerosis remains incompletely understood, it is thought to involve alterations in the balance between extracellular matrix production and degradation [14,15]. Nicotine, a highly addictive component of cigarette smoke, stimulates the expression of matrix metalloproteinase (MMP) MMP-2 and MMP-9, which play a role in the development of unstable plaques [16]. Furthermore, Nordskog et al. (2003) reported that exposure of human endothelial cells to cigarette smoke condensate leads to the upregulation of MMP-1, MMP-8, and MMP-9 gene expression [17]. Clinical studies further emphasize the association between smoking and MMPs. Sivaraman et al. (2014) reported elevated levels of MMP-2 and MMP-9 in patients with acute myocardial infarction (AMI), with smokers exhibiting significantly higher MMP-9 levels than non-smokers [18].

Huang et al. (2016) identified significant associations between MMP-1, MMP-9, and MMP-10 and current smoking behavior in older adults without clinically manifest CVD [19]. Páramo et al. [15] also reported elevated circulating levels of MMP-1 and MMP-10 in asymptomatic smokers relative to non-smokers, suggesting that smoking may play a role in plaque instability through MMP activity [20].

Cannabis smoking and its cardiovascular impacts:

Cannabis smoking has been linked to ACS, potentially due to an immediate increase in heart rate and blood pressure, likely mediated by sympathetic nervous system activation and parasympathetic inhibition. Additionally, platelet activation, vascular inflammation and carboxy hemoglobin formation are considered potential adverse effects of cannabis smoking [11].

Water pipe smoking (Hookah) and cardiovascular events:

Similar to cigarette smoking, water pipe smoking (hookah)contains a significant amount of nicotine [21], which can lead to cardiovascular events. The use of charcoal to heat tobacco products in water pipe smoking (hookah) results in the release of substantial amounts of combustion products, including carbon-rich nanoparticles, which are significant coronary vasoconstrictor stimuli [13,22,23].

Mechanisms and contributing factors of smoking-induced vascular dysfunction:

Cigarette smoking encompasses two phases: The gas phase and the tar phase. Both phases contain free radicals that contribute to the progression of atherosclerotic disease [24]. Over 4,000 components have been identified in cigarette smoke, and numerous individual components have been studied, including [25]:

- Carbon monoxide.
- Polycyclic aromatic hydrocarbons.
- Nicotine.
- Free radicals present in both the gas and tar phases.

All these components contribute to the progression of atherosclerosis through:

- Vasomotor dysfunction.
- Inflammatory response.
- Platelet dysfunction.
- Alteration of prothrombotic and antithrombotic factors.
- Modification of lipid profile.

Classification of smokers:

The Smoking Index (SI) formula is calculated as the number of cigarettes smoked per day (CPD) multiplied by the number of years of smoking.

Based on this index, smokers are classified as follows: **[26]**

- Mild smoker: SI ≤200.
- Moderate smoker: 200 < SI < 400.
- Heavy smoker: SI ≥400 **[27].**

Smoking pattern:

Smokers are categorized as current smokers, former smokers, or non-smokers:

- Current smokers: Individuals who have smoked more than 100 cigarettes in their lifetime and continue to smoke.
- Former smokers: Individuals who have smoked more than 100 cigarettes in their lifetime but no longer do so.
- Non-smokers: Individuals who have never smoked or have smoked fewer than 100 cigarettes and do not smoke **[28-30].**

Aim of study:

The aim of this study is to evaluate the association between smoking, as an independent risk factor for CVD, and angiographic findings in young individuals diagnosed with ACS.

Ethics approval:

The confidentiality of patient records at Alexandria University Hospital was maintained, and ethical approval was granted by the Ethics Committee of the Faculty of Medicine, Alexandria University, under the number 0107823.

Patients and Methods

Study population:

This study included one hundred young smoker patients aged 18 to 45 years who presented with ACS specifically STEMI and NSTEMI at Alexandria University Hospitals over a thirty nine-month period from April 2020 to July 2023.

The exclusion criteria were: (1) Age over 45 years, (2) Patients diagnosed with hypertension, (3) Patients diagnosed with diabetes mellitus, (4) Patients diagnosed with familial dyslipidemia and (5) Patients with family history of CAD.

Study design:

This observational retrospective study evaluates the relationship between smoking as a standalone cardiovascular risk factor and angiographic findings in young patients (aged <45 years) presenting with ACS during a thirty nine-month study durationfrom April 2020 to July 2023.

Assessment:

Data were collected from patient records as follows: (1) Demographic data and clinical parameters, including age, gender, body mass index, smoking history, blood pressure, New York Heart Association NYHA functional class, chief complaint on admission, angiographic data, and medical treatment; (2) Laboratory investigations, including complete blood count, cardiac troponin, fasting blood glucose or HbA1c, serum creatinine, and lipid profile; (3) Standard 12-lead ECG; and (4) echocardiography parameters. Types of smoking included cigarette, cannabis, and water pipe smoking (hookah), and smoking patterns included smoking history, current smoker, and former smoker. The smoking index was calculated for smoker classification: Smoking index **[26]** [smoking index = Cigarettes smoked (CPD) × years of tobacco use], categorizing individuals as mild, moderate, or heavy smokers.

Statistical analysis:

The data were input into a computer system and analyzed utilizing IBM SPSS software, version 20.0 (Armonk, NY: IBM Corp). Qualitative data were expressed as frequencies and percentages.The Kolmogorov-Smirnov testwas applied to evaluate the normality of distribution for quantitative data, which were summarized using range (minimum and maximum), mean, standard deviation, median, and interquartile range (IQR). The significance of the results was evaluated at a 5% threshold. Fisher's Exact test was employed for chi-square correction when more than 20% of the cells had an expected count of less than 5. The F-test (ANOVA)was utilized for normally distributed quantitative variables to compare multiple groups, while the Kruskal-Wallis test was employed for abnormally distributed quantitative variables to compare multiple groups.

Results

Demographic data of the study cohort:

Of the 842 patients screened, 100 fulfilled the inclusion criteria and were incorporated into the final analysis. The study was carried out at Alexandria University Hospital over a span of thirty nine months, from April 2020 to July 2023. The mean age of the patients was 38.79±4.75 years, with a predominance of male patients (98%).

Distribution of the study cohort based onclinical parameters:

The chief complaint upon admission was predominantly typical chest pain, reported by 96 patients (96%). A total of 8 patients (8%) presented with exertional dyspnea classified as NYHA functional

class II, while the remaining 92 patients (92%) were in NYHA functional class I. The mean systolic and diastolic blood pressures were 126±13.51mmHg and 77.75±9.03mmHg, respectively. Patients with hypertensionwere excluded from this study.

Distribution of the study cohort based on angiographic findings (Table 1):

Out of the total patient population, 79 (79%) had single-vessel disease, with the left anterior descending artery (LAD) affected in 51% of patients, the right coronary artery (RCA) in 15%, and the left circumflex artery (LCX) in 13%. Additionally, 15 patients (15%) had double vessel disease, which included LAD + RCA in 8% and LAD + LCX in 7% of cases. A normal coronary angiogram was noted in 6% of patients, with a mean value of 1.09±0.45%.

In terms of initial thrombolysis in myocardial infarction (TIMI) flow, 11 patients (11%) had TIMI 0-1 flow, 17 patients (17%) had TIMI 2 flow and 72 patients (72%) had TIMI 3 flow. Furthermore, 97 patients (97%) achieved final TIMI 3 flow. Only 7 patients (7%) presented with a large thrombus, while 3 patients (3%) experienced the no-reflow phenomenon. Collateral circulation was observed in just 3% of patients, with 2% at the LAD level and 1% at the RCA level.

Distribution of the study cohort based on smoking patterns (Table 2):

Of the patients, 99% were current smokers. All patients smoked cigarettes, with 70 (70%) smoking only cigarettes, 7 (7%) smoking cigarettes and cannabis, 15 (15%) smoking cigarettes and water pipe smoking (hookah), and 8 (8%) smoking cigarettes, water pipe smoking (hookah), and cannabis. The average daily cigarette consumption was 25.85±9.51, and the average duration of smoking was 18.88±4.89 years. Based on the smoking index, 65 patients (65%) were classified as heavy smokers, 30 (30%) as moderate smokers, and 5 (5%) as mild smokers.

Distribution of the studied cases according totype of MI:

Our study included 82 patients (82%) with STEMI and 12 patients (12%) with NSTEMI.

Relation between smoking index and demographic characteristics:

The average ages for mild, moderate, and heavy smokers were 40±5.34 years, 37.77±5.88 years, and 39.17±4.09 years, respectively (p=0.34). In contrast, males were predominantly represented, comprising 65% of the patients, particularly in the heavy smoker category. This association was statistically significant (p=0.002).

Table (1): Distribution of the study cohort based on angiographic findings (n=100).

Angiographic findings	No.	%
Number of diseased vessels:		
0	6	6.0
1	79	79.0
2	15	15.0
Min. – Max.	0.0 – 2.0	
Mean ± SD.	1.09 ± 0.45	
Median (IQR)	1.0 (1.0 – 1.0)	
Location of infract related artery:		
LAD	51	51.0
LCX	13	13.0
LCX + LAD	7	7.0
RCA	15	15.0
RCA + LAD	8	8.0
Normal coronary angiogram	6	6.0
Coronary angiography:		
PPCI	82	82.0
PCI	18	18.0
Initial TIMI Flow:		
TIMI 0	3	3.0
TIMI 1	8	8.0
TIMI 2	17	17.0
TIMI 3	72	72.0
Thrombus burden:		
Thrombus present	7	7.0
No Thrombus	93	93.0
Final TIMI:		
TIMI 0	0	0.0
TIMI 1	0	0.0
TIMI 2	3	3.0
TIMI 3	97	97.0
No reflow phenomenon:		
No	97	97.0
Yes	3	3.0
Collaterals:		
RCA	2	2.0
LAD	1	1.0
LCX	0	0.0
LM	0	0.0
No collaterals	97	97.0

IQR: Inter Quartile Range. SD: Standard Deviation.

Table (2): Distribution of the study cohort based on smoking patterns (n=100).

Smoking patterns	No.	%
Current smoker	99	99.0
Fomer smoker	1	1.0
Number cigarette per day:		
Min. – Max.	5.0 – 50.0	
Mean ± SD.	25.87 ± 9.51	
Median (IQR)	26.0 (20.0 – 31.5)	
Duration of smoking (years):		
Min. – Max.	7.0 – 29.0	
Mean ± SD.	18.88 ± 4.89	
Median (IQR)	20.0 (15.5 – 22.0)	
Number of smoking index:		
Min. – Max.	100.0 – 840.0	
Mean ± SD.	479.81 ± 197.51	
Median (IQR)	460.0 (311.5 – 660.0)	
Smoking index:		
Mild smoker	5	5.0
Moderate smoker	30	30.0
Heavy smoker	65	65.0
Types of smoking:		
Cigarette only	70	70.0
Cigarette + cannabis	7	7.0
Cigarette+ water pipe smoking (hookah)	15	15.0
Cigarette + water pipe smoking (hookah) + cannabis	8	8.0

IQR: Inter Quartile Range. SD: Standard Deviation.

Relation between smoking index and type of MI:

The Fisher Exact test revealed a significant relationship between the smoking index and the type of MI (p=0.012). Specifically, a heavy smoking index was noted in 73.8% of of patients with STEMI and in 26.2% of of patients with NSTEMI.

Relation between smoking index and chief complaint on admission:

The Fisher's Exact test revealed a significant association between the smoking index and the chief complaint at the time of admission (p=0.006). A high smoking index was identified in 98.5% of patients exhibiting typical chest pain and in 1.5% of patients exhibiting epigastric pain.

Relation between smoking index and lipid profile:

The Kruskal-Wallis testwas employed to assess the relationship between smoking and lipid profile, revealing no significant differences across all lipid profile categories. The average triglyceride (TG) levels were 126.4±21.03 in mild smokers, 139.2±40.87 in moderate smokers, and 146±41.52 in heavy smokers (p=0.295). The average total cholesterol (TC) levels were 182.8±30.48 in mild smokers, 183.6±40.24 in moderate smokers, and 166.5±33.51 in heavy smokers (p=0.094). The average Low density lipoprotein (LDL) levels were 128±19.92 in mild smokers, 115.1±22.42 in moderate smokers, and 125 ± 28.10 in heavy smokers (p=0.161). The average High density lipoprotein (HDL) levels were 45.40±8.3 in mild smokers, 46.73±11.70 in moderate smokers, and 48.22±11.50 in heavy smokers (p=0.764).

Relation between types of smoking and angiographic findings (Table 3):

The relation between the type of smoking and TIMI flow, thrombus burden, no reflow phenomenon and collaterals were not significant in all categories of type of smoking in this study. A statistically significant relationship was observed between the type of smoking and the number of diseased vessels (p=0.013). Single vessel disease was strongly associated with cigarette, shisha, and cannabis use, with a prevalence of 100%. Additionally, the connection between the type of smoking and infarct-related artery (IRA) was significant (p=0.040), with the LAD being most affected and associated with cigarette, shisha, and cannabis use.However, the study showed no significant relationship between the type of smoking and TIMI flow, thrombus burden, the no-reflow phenomenon, and collateral formation across all categories of smoking (Figs. 1,2).

Relation between smoking index and angiographic findings (Table 4):

The relationship between the number of diseased vessels and the smoking index was statistically significant (p=0.001) (Fig. 3). Most patients were categorized as heavy smokers, with 80% having single-vessel disease and 20% having double-vessel disease. The mean number of diseased vessels was 1.20±0.40. In terms of the location of the IRA, most patients were heavy smokers. The distribution was as follows: LAD was highly represented at 44.6%, RCA at 21.5%, LCX at 13.8%, LCX+LAD at 7.7%, and RCA+LAD at 12.3%. This relationship was significant (p=0.001) (Fig. 4). A significant correlation was observed between the smoking index and the type of intervention, with 73.8% of patients undergoing PPCI and 26.2% undergoing PCI (p=0.012). The relation between smoking index and TIMI flow, thrombus burden, no reflow phenomenon and collateral presence was not significant across mild, moderate and heavy smokers.

Table (3): Relation between types of Smoking and angiographic findings (n = 100).

Angiographic findings	Types of smoking								Test of sig.	*p*
	Cigarette only (n = 70)		Cigarette + cannabis (n = 7)		Cigarette+water pipe smoking (hookah) (n = 15)		Cigarette + water pipe smoking (hookah) + cannabis (n = 8)			
	No.	%	No.	%	No.	%	No.	%		
Number of diseased vessels:										
0	1	1.4	2	28.6	3	20.0	0	0.0	FET=13.701*	0.013*
1	58	82.9	4	57.1	9	60.0	8	100		
2	11	15.7	1	14.3	3	20.0	0	0.0		
Min. – Max.	0.0 – 2.0		0.0 – 2.0		0.0 – 2.0		1.0 – 1.0		H=3.327	0.344
Mean ± SD.	1.14 ± 0.39		0.86 ± 0.69		1.0 ± 0.65		1.0 ± 0.0			
Median	1.0		1.0		1.00		1.0			
Location of IRA:										
LAD	40	57.1	3	42.9	3	20.0	5	62.5	FET=20.859*	0.040*
LCX	7	10.0	1	14.3	3	20.0	2	25.0		
LCX + LAD	5	7.1	1	14.3	1	6.7	0	0.0		
RCA	11	15.7	0	0.0	3	20.0	1	12.5		
RCA+LAD	6	8.6	0	0.0	2	13.3	0	0.0		
Normal coronary angiogram	1	1.4	2	28.6	3	20.0	0	0.0		
Coronary angiography:										
PPCI (82)	54	77.1	7	100.0	14	93.3	7	87.5	FET=3.120	0.355
PCI (18)	16	22.9	0	0.0	1	6.7	1	12.5		

SD: Standard deviation. *p*: *p*-value.
H: H for Kruskal Wallis test. *: Statistically significant at $p \leq 0.05$.
FET: Fisher Exact test.

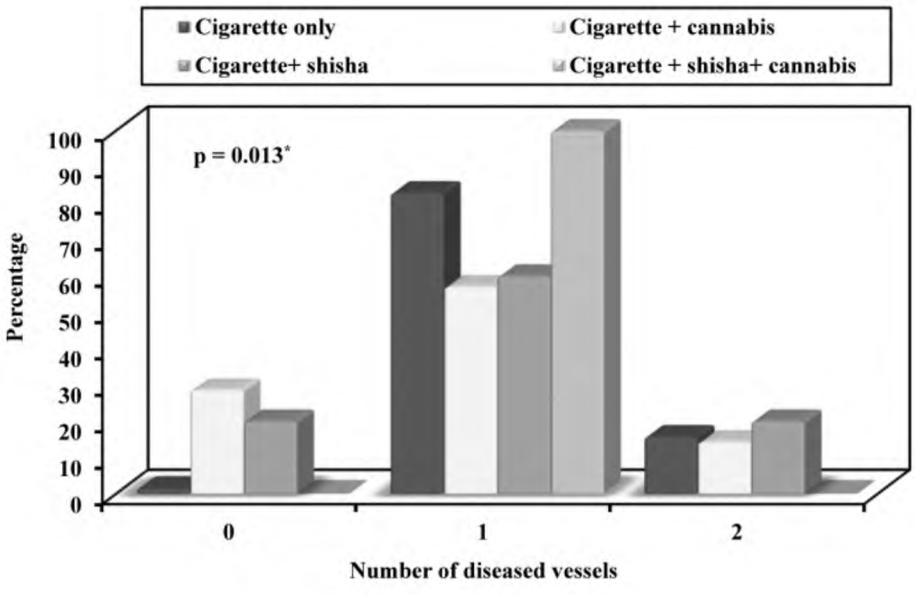

Fig. (1): Relation between types of smoking and number of diseased vessels (n = 100).

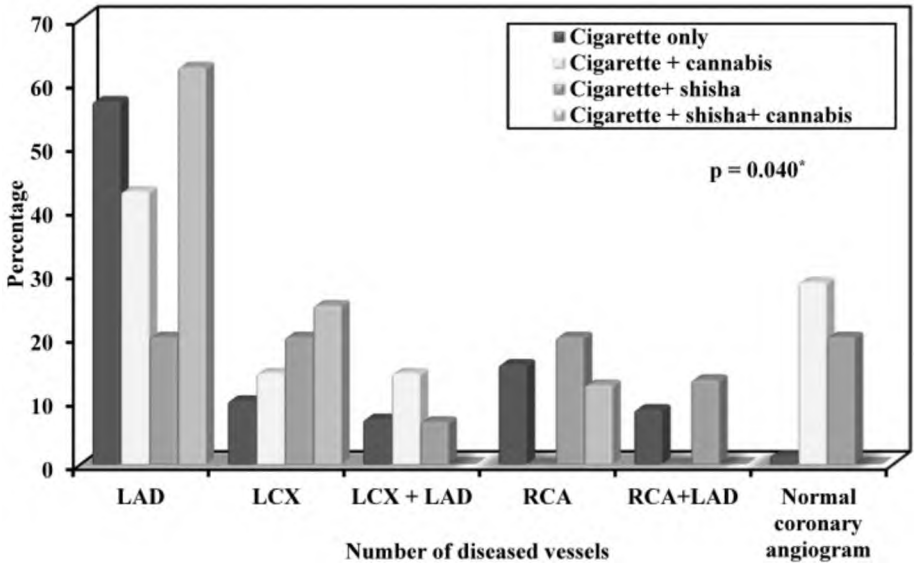

Fig. (2): Relation between types of smoking and location of infarct related artery.

Table (4): Relation between smoking index and angiographic findings (n = 100).

Angiographic findings	Smoking index						Test of sig.	*p*
	Mild smoker (n = 5)		Moderate smoker (n = 30)		Heavy smoker (n = 65)			
	No.	%	No.	%	No.	%		
Number of diseased vessels:								
0	2	40.0	4	13.3	0	0.0	FET=15.852*	0.001*
1	3	60.0	24	80.0	52	80.0		
2	0	0.0	2	6.7	13	20.0	H=12.641*	0.002*
Min. – Max.	0.0 – 1.0		0.0 – 2.0		1.0 – 2.0			
Mean ± SD.	0.60 ± 0.55		0.93 ± 0.45		1.20 ± 0.40			
Median	1.0		1.0		1.0			
Location of IRA:								
LAD	2	40.0	20	66.7	29	44.6	FET=24.756*	0.001*
LCX	1	20.0	3	10.0	9	13.8		
LCX + LAD	0	0.0	2	6.7	5	7.7		
RCA	0	0.0	1	3.3	14	21.5		
RCA+LAD	0	0.0	0	0.0	8	12.3		
Normal coronary angiogram	2	40.0	4	13.3	0	0.0		
Coronary angiography:								
PPCI (82)	5	100.0	29	96.7	48	73.8	FET=8.181*	0.012*
PCI (18)	0	0.0	1	3.3	17	26.2		

SD: Standard deviation. *p*: *p*-value.
H: H for Kruskal Wallis test. *: Statistically significant at $p \leq 0.05$.
FET: Fisher Exact test.

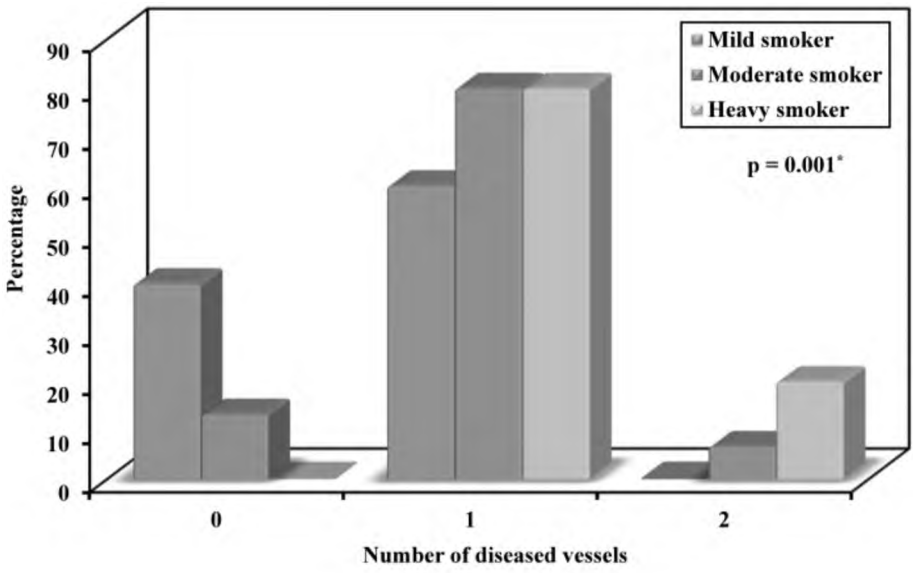

Fig. (3): Relation between smoking index and number of diseased vessels (n = 100).

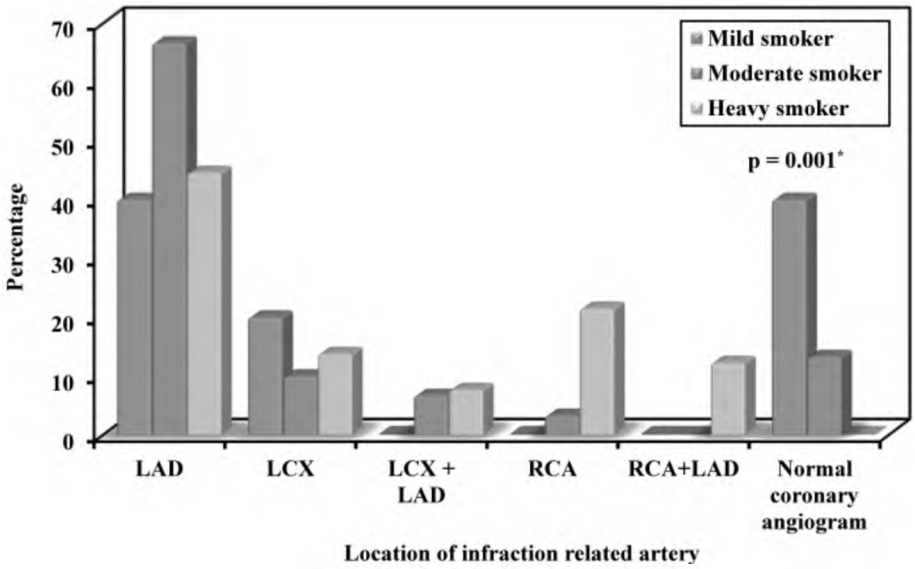

Fig. (4): Relation between smoking index and location of infract related artery (n = 100).

Discussion

Smoking, including cigarettes, cannabis, and water pipe smoking (hookah), isa well-established risk factor for ACS in both young individuals and the general population **[2,4,11,21]**. This retrospective observational study investigated the association between smoking as the sole cardiovascular risk factor and angiographic findings in patients younger than 45 years presenting with ACS. These patients presented at Alexandria University Hospitals over a thirty nine-month period. We used the calculation of the smoking index as a primary tool to classify smokers, which aided in evaluating patients according to their smoking levels **[27].**

Smoking index and demographic data:

In our study, males comprised the majority of the patient population, representing 98%. The most affected age group was between 36 and 45 years, with a mean age of 38.79±4.75 years.

This distribution aligns with the high smoking index observed. While very young patients had a higher number of cigarette consumptions, they experienced a lower pack-year or smoking index, as also noted by Barbash GI et al., in their study on acute MI in young individuals and the impact of smoking **[10].**

The association between smoking index and gender in our study was statistically significant

(p=0.002). The study included only 2 female patients, both categorized as mild smokers. On the other hand, among the male patients (98), 3 were mild smokers, 30 were moderate smokers, and 65 were heavy smokers.

Our study demonstrated a higher incidence of ACS in male patients compared to female patients, a finding consistent with the results of Samir et al. in their prospective study. It was demonstrated that males are more affected than females, potentially due to the protective role of sex hormones against vascular inflammation and atherosclerosis during a woman's childbearing years, with a subsequent increase in risk after menopause *[31]*.

Additionally, socio-behavioral factors like heavy smoking are more prevalent among males, as highlighted in our study, which showed a significantly higher proportion of young male patients under 45 years old.

Similar findings were reported by Reda et al. (2021) *[32]* in their study on the prevalence of atherosclerosis risk factors in ACS patients, showing that the majority of ACS patients were male (63% male versus 5% female).

Our study also indicated that smoking predisposes individuals to MI at a younger age, a finding consistent with the retrospective study by Bujaket al. (2023) *[33]* on the effect of smoking on outcomes in patients with STEMI receiving PPCI.

Type of smoking:

Cigarette smoking is a major modifiable risk factorfor atherosclerotic heart disease *[34]*. Cigarette smoke exposure can increase MMP activity and inflammation, which are crucial in the activation and disruption of atherosclerotic plaquesand ACS *[18,35,36]*.

The rapid elevation of heart rate and blood pressure, coupled with coronary vasospasm following cannabis use, has been linked to the onset of ACS, attributed to the activation of the sympathetic nervous system and inhibition of the parasympathetic nervous system. Vascular inflammation, platelet activation, and the production of carboxyhemoglobinhave been suggested as potential complications associated with cannabis smoking *[11]*.

In our study, all patients were cigarette smokers. Among them, 70 patients (70%) smoked only cigarettes, 15 patients (15%) smoked both cigarettes and water pipe smoking (hookah), 7 patients (7%) smoked both cigarettes and cannabis, and 8 patients (8%) smoked a combination of cigarettes, cannabis, and water pipe smoking (hookah).

Bhatnagar et al. *[13]* found that both water pipe smoking (hookah) and cigarette smoking are potent factors for MI, with water pipe smoking (hookah) causing more severe decrements in endothelium-dependent and flow-mediated functions compared to cigarette smoking. In our study, water pipe smoking (hookah) and cigarette smoking were associated with MI, a finding also supported by Abdulmajeed et al. *[37]*. In their study assessing water pipes and the risk of MI, which indicated a significant association between current water pipe smoking (hookah) smoking and MI.

The increased use of cannabis has a potential link with MI, as stated by Justin Lee et al. *[38]* in their study on cannabis and MI. In the study "Cannabis Use Linked to Elevated Myocardial Infarction and Stroke Risk" conducted by Samanta Anderer et al. *[39]*, daily cannabis users were found to have a 25% higher odds of MI. This finding is in agreement with our results, as cannabis consumers in our study developed MI at a rate of 15%.

Smoking index and type of MI:

Our study indicated that a high smoking index is likely to promote the development of ACS. This was observed as 65% of our ACS patients had a high smoking index, 30% were moderate smokers, and 5% were mild smokers, with this association being statistically significant (p=0.012).

Similar results were demonstrated by Notar et al., who observed that patients with a high smoking index had an increased risk of ACS, with a prevalence of 57.8% *[40]*. Comparatively, Sia et al., in their retrospective study assessing the relationship between smoking status and outcomes in MI patients who underwent PCI stated that smoking increases the risk of AMI, making smokers more prone to developing STEMI and NSTEMI *[41]*.

Our study found that cigarette smokers are more prone to developing STEMI than NSTEMI due to the prothrombotic effects of smoking. This is supported by our data showing that 82% of our patients developed STEMI, and 18% had NSTEMI, which was statistically significant (p=0.012). The same findings were also observed by Dominique Himbert et al., in their study on cigarette smoking and ACS, *[42]* as well as by Robertson et al., in their study *[43]*.

Smoking index and typical chest pain:

In our study, a significant association was observed between the smoking index and the chief complaint at the time of admission (p=0.006).

Among heavy smokers, 98.5% presented with typical chest pain. This observation aligns with findings from Friedman et al., who identified typical chest pain as a principal symptom in smoker patients with ACS [44].

Smoking index and lipid profile:

Cigarette smoking contributes to the advancement of atherosclerosis by directly impacting the lipid profile.

In their study on cigarette smoking and serum lipid and lipoprotein levels, Craig et al., found that smoking raises TC, TG, and LDL, while lowering HDL [45]. However, our study did not show a significant association between the smoking index and lipid profile, in line with the findings of Toluey et al. who reported a reduced prevalence of hyperlipidemia among smokers [46].

Types of smoking and angiographic data:

A significant correlation was observed between the type of smoking and the number of diseased vessels, as well as the location of the infarct-related artery (p=0.013 and p=0.040, respectively).

Several studies have explored the impact of smoking on coronary lesions, producing conflicting results. In line with our findings, Koliaki C et al., observed a positive correlation between smoking and the location of coronary lesions in LAD, RCA and LCX arteries in their study on the relationship between cardiovascular risk factors and angiographic data [47]. Additionally, Salehi et al., in their study on the effects of smoking on coronary arteries and the severity of CAD, cigarette smoking was found to be associated with obstruction of the LAD artery [48]. In a similar study, Toluey et al., reported that smoking elevates the risk of lesions in the RCA to a greater extent than lesions in other coronary vessels [46]. In contrast, Koz et al., study on coronary risk factors and lesion distribution involving the left main coronary artery (LMCA), proximal or mid LAD artery, and other coronary lesions found no association between smoking status and location of coronary lesions [49].

Smoking index and angiographic data:

In our study, the relationship between smoking index and the number of diseased vessels, as well as the site of the IRA, was significant (p=0.001). We observed that moderate and heavy smokers are more likely to develop single-vessel disease (80% of patients) compared to double-vessel disease (20% of patients). In the moderate smoker group, the LAD artery was affected in 66.7% of the 30 patients, whereas in the heavy smoker group, it was affected in 44.6% of the 65 patients. In the heavy smoker group, the RCA was involved in 21.5% of the 65 patients, while the LCX was affected in 13.8% of the same cohort. Among patients with double-vessel disease in the heavy smoker group, 7.7% exhibited involvement of the LCX and LAD, while 12.3% had involvement of the RCA and LAD. These findings highlight the impact of heavy smoking on the development of coronary atherosclerotic disease. In our study, only three patients had three-vessel disease, and they were excluded due to diabetes mellitus, which was an exclusion criterion. A significant association was identified between the smoking index and the type of PCI, with 73.8% of patients undergoing PPCI and 26.2% undergoing PCI with a p-value of 0.012.

Our findings are in agreement with the study conducted by Robertson Jo et al., which investigated the influence of cigarette smoking on the severity of coronary artery disease and the prognosis of patients with NSTEMI ACSs [43]. Comparable findings were reported by Barbash GI et al., who investigated the paradoxical beneficial effects of smoking in patients receiving thrombolytic therapy for AMI. They reported that smokers with NSTEMI had a similar experience to those with STEMI, with less multivessel involvement [50].

In our study, the initial TIMI flow was predominantly classified as TIMI 3 in 72% of patients, whereas TIMI 0–1 was noted in 11% and TIMI 2 in 17% of patients.

The final TIMI flow was classified as TIMI 3 in 97% of patients. Thrombus burden was identified in 7% of patients, and collaterals were found in 3% of patients 2% in the LAD position and 1% in the RCA position.

In comparison to other studies, Jason O. Robertson et al., reported similar initial TIMI 3 flow and final TIMI 3 flow following PCI with rates of 71% and 96.5%, respectively, versus 72% and 97% in our study [43].

However, no statistically significant correlation was found between the smoking index and both initial and final TIMI flow (p=0.141). Alaarg AF et al., also found no significant association between smoking status and initial and final TIMI flow in their study [51].

Contrary to our findings, which showed no significant relationship between smoking index and collaterals due to our small sample size (100 patients), Jeroen Koerselman et al., reported a significant relationship between smoking and coronary collaterals in their study [52].

Kloner et al., defined the no-reflow phenomenon as suboptimal myocardial reperfusion after complete coronary revascularization [53]. The association between the smoking index and the no-reflow phenomenon was not significant in our study (p=1.000).

This result differs from the study by Fajar J et al., which identified smoking as a predictor of the no-reflow phenomenon following PCI in patients with STEMI [54].

Consistent with our findings, Ndrepepa G et al., found no correlation between smoking and the no-reflow phenomenon in their study on predictors and outcomes of no-reflow after PPCI in patients with AMI. They found that a large infarct size, reduced myocardial salvage, and impaired left ventricular ejection fraction were linked to the no-reflow phenomenon [55].

Conclusion:

Cigarette smoking is a significant contributor to the pathogenesis of ACS. The concurrent use of cigarettes, water pipe smoking (hookah), and cannabis further elevates the risk of ACS. Patients with a higher smoking index exhibit a more pronounced severity of ACS than those with a lower smoking index. Calculating the smoking index is essential for classifying patients as mild, moderate, or heavy smokers.

Recommendation:

Emphasizing smoking cessation campaigns for ACS prevention in both youth and the general population is crucial. Future researchers are encouraged to conduct multicenter studies with large sample sizes. Further research on myocardial infarction and various types of smoking is also highly recommended.

Study limitation:

We conducted the study on a small population due to the lack of certain information and some patients' refusal to disclose their smoking status over the phone, which affected our sample size. The data were collected from a single center, and there are limited studies on myocardial infarction with smoking as a single risk factor.

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Conflict of interest:

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

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العلاقة بين التدخين كعامل خطر واحد للقلب والأوعية الدموية وبيانات تصوير الأوعية الدموية لدى مرضى متلازمة الشريان التاجى الحادة من الشباب: دراسة مركزية واحدة بأثر رجعى

يعد تدخين السجائر من أهم عوامل الخطورة التى يمكن الوقاية منها لمرض تصلب الشرايين التاجية، كما أن الارتباط بأنواع أخرى من التدخين مثل الشيشة والحشيش يزيد من خطر الإصابة بالاحتشاء القلبى لدى المرضى صغار السن وكذلك لدى عامة السكان.

تم الكشف عن أن النيكوتين الموجود فى منتجات التبغ يعزز تكوين اللويحات وتراكم الصفائح الدموية.

إن التعبيرعن المصفوفة ميتالوبروتيناز-٢ والمصفوفة ميتالوبروتيناز-٩ الناجم عن النيكوتين الموجود فى السجائر يساهم فى تكوين لوحة شريانية غير مستقرة.

وبالتالى كان الهدف من دراستنا هو تقييم أنماط التدخين كعامل خطر وحيد لمتلازمة الشريان التاجى الحادة لدى المرضى الذين تقل أعمارهم عن ٤٥ عامًا.

شمل هذا البحث على ١٠٠ مريض أصيبوا بمتلازمة الشريان التاجى الحادة مع وجود تاريخ مرضى للتدخين، وكان غالبية المرض من الذكور بنسبة ٩٨٪، وكان متوسط العمر ٣٨.٨ ± ٤.٥، وقد وجد أن مؤشرالتدخين المرتفع فى دراستنا وصل إلى٦٥٪ وتطور هؤلاء المرضى لمتلازمة احتشاء القلب المصاحب بإرتفاع المقطع إس-تى.

وفقا لنوع التدخين، ٧٠٪ من مرضانا يدخنون السجائر فقط، ٧٪ يدخنون السجائرمع الحشيش، ١٥٪ يدخنون السجائر مع الشيشة وأخيراً ٨٪ من المرضى يدخنون السجائرمع الحشيش والشيشة.

أصيب المرضى الذين لديهم مؤشر تدخين مرتفع بمتلازمة الشريان التاجى الحادة مقارنة مع المرضى الذين لديهم مؤشر تدخين منخفض وكان احتشاء عضلة القلب النصفى (مصاحب بإرتفاع المقطع الكهربائى اس-تى) ممثلاً بشكل كبير بنسبة ٨٢٪ مقارنة مع الغير مصاحب لإرتفاع المقطع الكهربائى اس-تى بنسبة ١٢٪.

وفقًا لبيانات تصوير الشرايين التاجية، تم علاج ٩٤٪ من المرضى عن طريق قسطرة الأوعية الشرايين التاجية باستخدام الدعامات و ٦٪ من المرضى لديهم تصوير الأوعية التاجية الطبيعى، وفقًا للأوعية المصابة، كان الشريان التاجى المفرد ممثلاً بشكل كبير مقارنة بإصابة الشرايين المتعددة وتأثر الشريان التاجى الأمامى بنسبة ٥١٪.